DISCLAIMER

The information contained within this document does not constitute medical advice or diagnosis and is intended for education and information purposes only. It was current at the time of publication and every effort is made to keep the document up to date.

The information contained herein includes both psychological and non psychological interventions. The delivery of psychological services requires a medical referral whilst non psychological services do not.

Each person is an individual and has a unique psychological profile, biochemistry, developmental and social history. As such, advice will not be given over the internet and recommendations and interventions within this website cannot be taken as a substitute for a thorough medical or allied health professional assessment or diagnosis.

Depression

Article QUICK LINKS :

INTRODUCTION

Depression affects most people at some stage of their lives. Clinical depression may be defined as a mood disorder which impairs normal function.

Depression is a 'whole-body' disorder affecting many levels of being including the body, nervous system, moods, thoughts, and behaviour. Observable symptoms of depression may include chronic fatigue, sleep disturbances, changes in appetite, headaches, backaches, digestive disorders, restlessness, irritability, loss of interest or pleasure in hobbie, and feelings of worthlessness and inadequacy.

DSM-IV (The Diagnostic and Statistical Manual published by the American Psychiatric Association) lists the essential feature of these disorders also referred to as **affective disorders** as persistent or episodic exaggeration of mood state.

The DSM-IV further delineates and differentiates the following mood disorders:

- **Mood Episodes:** Major Depressive Episode | Hypomanic Episode | Manic Episode | Mixed Episode
- **Depressive Disorders:** Dysthymic Disorder | Major Depressive Disorder: Single Episode | Recurrent
- **Bipolar Disorders:** Bipolar I Disorder | Bipolar II Disorder | Cyclothymic Disorder
- **Mood Disorder due to a General Medical Condition with:** Depressive Features | Manic Features | Mixed Features
- **Substance-Induced Mood Disorder**

Persons with major depression feel down, discouraged and often hopeless, it may seem to them that nothing is right with their lives. Typical cognitive symptoms of depression are low self-esteem, loss of motivation, and pessimism. A single event which they perceive as failure may seem insurmountable or a foreshadowing of worse things to come, and may lead to anxiety. The depressed person will often experience a very low level of energy and may accordingly slow down their movements and rate of speech. Difficulty in sleeping or waking up may also be experienced.

In noting the symptoms of depressed individuals, clinicians often look to the causes and distinguish them between external, environmental variables, and internal physiological variables as depicted in the following table:

Depression Type	**Description**	**Example Cause**
Exogenous Depression	A reaction to external, environmental factors	Conflict with a spouse or lover, stress on the job, failure to achieve a goal, or similar types of events.
Endogenous Depression	Reaction to internal, physiological factors such as an imbalance of particular neurotransmitters	Chronic depression without correlation to what is going on about the person may stem from a family history of depression (genetic). May be dietary related.
Primary Depression	Depression is the main medical problem	Being depressed over a relationship breakup and feeling unable to get out of bed as a result.
Secondary Depression	Another disorder has caused the depression	Being injured, or bedridden and becoming depressed about the physical limitations imposed - May also be caused by medications.
Involutional Depression	Associated with advanced age	Consequences associated with age such as the realisation that it is too late in life to achieve goals that were set at a prior life stage
Postpartum Depression	Occurs after childbirth and can last from weeks to a year	Stress is usually the primary cause; hormonal changes may be implicated; changes in neurotransmitters, and fatigue; external locus of control, anxiety, hostility; and lack of spousal or other support may also be factors. Inadequate intake of essential fatty acids has also been cited as a cause.

The aspects of self-concept, activity level and body maintenance (or vegetative) functions are altered in these disorders as much if not more than mood.

Clinical experience has shown that understanding of the affective disorders needs to take place at three levels of enquiry:

1. The experience of the mood disorder, which refers to the conscious feeling of depressive affect or sadness at one end of the continuum or of the elation and euphoria at the other.

2. The outward expression of the affective disorder including changes in the level of activity and neurovegetative functioning

3. The cognitive components of these disorders characterised by the either unduly negative or unduly positive appraisal of internal or external events.

The affective disorders including depression can be characterised by their interference with the emotional tone of the processing of information.
The deficits so produced are more by way of a loss of effortful processing rather than intrinsic problems with the actual information processing.

As we can see, depression has multiple possible explanations, and accordingly, there are various schools of thought on the roots of depression, and as is usual, since we are considering a human being's complexity on many levels, all have their merits and require consideration.

PSYCHODYNAMIC EXPLANATIONS

Psychodynamic theory begins with Freud's (1917-1957) observational analogy between depression and mourning, whereby he noticed that in both cases, there is a strong sense of overwhelming sorrow, and that people in mourning often become depressed. He felt that when we lose an object of our love, we incorporate aspects of that person in a fruitless effort to regain at least parts of the person - yet often there are ambivalent feelings - we miss the person, but are angry at our loss. He suggested that this anger directed at a person whose 'parts' we have incorporated is anger directed at ourselves and is the source of depression at least symbolically.

LEARNING THEORY EXPLANATIONS

The learning theory of depression is basically that depressed people receive fewer rewards and more punishment than people who do not feel depressed. Thus, we may conclude on that statement that fewer things make a depressed person happy, and more things make a depressed person unhappy. This implies that depression may be a self-sustaining state.

COGNITIVE EXPLANATIONS

Cognitive theory suggests that a form of learned helplessness leading to frustration is the cause of depression. Aaron Beck says that inappropriate attributions and inferences directly contribute to depression and that depressed persons are particularly susceptible to errors in thinking - in particular - logical errors that lead them to see things in an unfavourable manner by:

1. Drawing a conclusion even though there is little or no evidence to support it.
2. Focussing on an insignificant detail of a situation while ignoring the more important features.
3. Drawing global conclusions about ability on the basis of a single fact or episode.
4. Committing gross errors of evaluation by magnifying small, unfavourable events, yet minimising important large and favourable events.
5. Taking personal responsibility for events that are situational.

It should be noted that a given thought pattern may involve several of these distortions simultaneously.

HUMANISTIC-EXISTENTIAL EXPLANATIONS

Viktor Frankel (1959) observed through his own experiences of the World War in a nazi concentration camp, that depression results from a lack of purposeful living. This suggests that finding meaning or spirituality in our lives is important in ameliorating depression, as well as aiding us in confronting other forms of challenge in our lives.

NEUROPSYCHOPHYSIOLOGICAL EXPLANATIONS

The consistent finding of neuropsycholphysiological studies of depression is that abnormally low levels of neurotransmitters - mainly norepinepherine, dopamine and serotonin - appear to be linked to depression. Other findings correlate with an imbalance of the thalamocortical circuiting and feedback and gating circuits. In the depressed state the overactive circuits represent autonomous and exaggerated activity of prefrontal or basal ganglia circuits that code for negative imagery of self and the larger world.[3]

Work as early as 1937 (Papez, 1937) suggested that reverberations through the limbic system were responsible for generating emotional activity. And subsequent studies as well as PET investigations in 1992 by Drevets et.al., found that increased blood flow through the amygdala may be a trait marker for depressive disorders whether depression is manifest or not. Drevets, in comparing his findings to other neurophysiological data available, suggests that the functioning of a prefrontal-amygdala-medial dorsal thalmic circuitry is overactive in a depressed individual's brain[3]. CT studies by Schlegal and Ketzschmar in 1987 cited ventricular enlargement in unipolar as well as bipolar depression.[4]

LeDoux, Romanski and Xagoraris (1989) found that there are correlations between the Automatic Nervous System (ANS) balance and depression, citing that different patterns of ANS arousal and different emotions correspond.[10] This also corresponds to endocrine system dysfunction. (See the article: "The Relationship between Vital Energy, The Brain and the Human Nervous System" for a more information of the implications of ANS dysfunctions)

A study by Starkstein and Robinson in 1997[5], suggests that dysfunction of the frontal lobes (the executive and decision making area of the brain) may produce overinhibition of dorsal brain areas, thus abnormally reducing motor, instinctive, intellectual, and emotional output, and further studies in this area by Mayberg[6], suggest that recovery from depression will involve inhibition of the overactive ventral regions (amygdal/limbic system) and normalisation of the frontodorsal hypofunction.

EEG findings in studies of genetic unipolar depressives show that depressed persons display a disorganised atypical sleep pattern which skips a 'level' of deep sleep and prominent delta and theta waves (which are sleep waves) in the waking state.[7] So we may conclude that the depressed person's brainwave activity in sleep is invaded by 'waking' waves and the reverse in the waking state.

The Brain-Thyroid axis has also been implicated in the biology of depression and the blunting of the thyroid stimulating hormone (TSH) response to thyrotropin releasing hormone and the elevation of serum T4 are the most consistent findings.[8]

Recent studies suggest that closed head injury of the coup-contra coup type involving axonal shear contribute significantly to depression via reduction of serotonin receptors and disruption of the connections between dorsal and ventral areas of the brain.[9]

Many studies have found relationships between the type of food we ingest and depression. One of the most common causes of depression is in fact food allergies, and another is hypoglycaemia (low blood / sugar).[11]

Heredity (genetics) is a significant factor which can not be overlooked. In up to 50% of people suffering depression, one or both parents also experienced depression.

STATISTICS

SUICIDE

Of major concern is the increase in suicide rates worldwide, and the connection to depression. It should be noted though that whilst most people who have committed suicide felt depressed; not everyone who is depressed attempts suicide.

- Depression is one of the most common mental disorders affecting 340 million people in the world today, accounting for a full 10% of productive years lost throughout the world.
- No one is immune from depression - it occurs in people of all social classes, all countries and all cultural settings.
- One in four women and one in ten men can expect to develop depression during their lifetime, but it's not just adults who suffer, depression affects at least one in 50 children under 12 and one in 20 teenagers.
- About half of all cases of depression is unrecognised and untreated.
- About 10-15 per cent of depressed people take their own lives.
- Depression costs for example the United States an estimated $53 billion each year.
- The World Health Organisation predicts that by the year 2020 depression will be the greatest burden of ill-health to people in the developing world, and that by then severe depression will be the second largest cause of death and disability.

DEPRESSION IS ONE OF THE MOST TREATABLE MENTAL ILLNESSES.

STRATEGIES FOR DEPRESSION

There are, broadly speaking, three therapeutic approaches: the use of psychological or talk therapies and biofeedback; the use of antidepressant drugs; and a combination of both. Many people believe that psychological therapies make more sense than the use of antidepressants.

In reality, the choice of the correct protocols for an individual depends upon a variety of factors, such as severity and type of depression, presenting symptoms, history including familial, age, diet and lifestyle etc., thus a holistic approach in both assessment and program strategies would seem to be best.

MODERN MEDICINE

Treats depression with anti-depressants (medication).

Antidepressant therapies began in 1956 when a drug used in the treatment of tuberculosis was found to elevate mood. Two years later, the antidepressant imipramine was marketed.

Today, there is a wide choice of antidepressants available. They are the medically recognised treatment for all forms of moderate and severe depression *regardless of cause*. About 70 per cent of patients with depression respond to treatment with antidepressants, yet the side effects of these drugs can be a major contributor to depression.

Anti-depressants can be distinguished most commonly by their safety profile or tolerability, although there are indications that efficacy, especially in severe depression, varies between classes of antidepressants

Antidepressants do not act immediately. The lifting of depressed moods typically takes up to two weeks and can take longer. There are two other unique features about antidepressants, which distinguish them from other chemicals which act on the brain.

Many drugs which act on the brain have much the same action on people who are unaffected by mental illness or those who have a problem.

Dr D Healy in his book 'Psychiatric Drugs Explained' (Mosby, 1997) explains effects of antidepressants using comparison with coffee or alcohol intake. One cup of coffee may be alerting, two cups more so and three cups makes you really wired up. Similarly the more you drink the greater the effect that alcohol has on the brain. But with antidepressants taking a dose above a certain threshold will not increase the antidepressant effect. The only thing which happens is that you will get more than the usual side effects. And more worrying, some antidepressants can be fatal if taken in relatively small amounts above the recommended maximum dose because of their effects on the heart.

Antidepressants work within the brain to either increase the levels of noradrenaline, serotonin or both. For example, tricyclic antidepressants like amitriptyline increase the levels of both noradrenaline and serotonin in the brain - they consequently have a dual action effect.

However the selective serotonin reuptake inhibitors , SSRIs, work only on the serotonin system. There is a belief among experts that they are not quite as effective as 'dual action' drugs in treating more severe forms of depression.

WHAT HAPPENS IF YOU DON'T RESPOND TO TREATMENT WITH ANTIDEPRESSANTS?

First you have to be taking an 'adequate' dose of antidepressant regularly for up to six weeks before your doctor will conclude that you have not responded to treatment. If you still haven't improved by this stage, your doctor has a number of alternatives to consider, including raising the dose, switching to another antidepressant or even trying any combination of the available antidepressants.

If you still have not responded your doctor may then consider electroconvulsive therapy (ECT), which is reserved for very severe depression that has not responded to other treatments or for people who cannot take antidepressants for other reasons. Failing this, there is always surgery. Cingulotomy, limbic leucotomy, capsulotomy and general psychosurgery have all been performed to treat depression with varying 'results'.[9]

Antidepressants differ widely in the side effects they produce.
All classes of psychotherapeutic drugs are capable of depleting vital nutrients necessary for the maintenance of good health in the body.[15]

In Australia, the following drugs are listed in the MIMS (an Australian publication of 'approved' drugs prescribed doctors) as being suitable for depression:- Allegron, Anafranil, Arima, Aropax, Auroix, Auscap, DBL Clomipramine, DBL Fluoxetine, DBL Moclobemide, Deptran, Dothep, Efexor, Efexor XR, Endep, Erocap, Fluohexal, Lovan, Lumin, Luvox, Melipramine, Nardil, Parnate, Perofan, Placil, Prothiaden, Prozac, SBPA Fluoxetine, Surmontil, Tofranil, Tolvon, Tryptanol, Zactin, Zoloft. Other drugs not specifically designed for depression may also be prescribed by your doctor depending upon presentation.

Following is a *brief* overview of the main characteristics of the major classes of antidepressants:

Tricyclic antidepressants e.g. amitriptyline and clomipramine
Tricyclic antidepressants (TCAs) were introduced over 30 years ago. They are widely regarded as among the most effective antidepressants available. But they have a number of major down sides because their effects in the brain are not only restricted to alleviating depression. They also interact with a number of other brain receptors, thus causing side effects. This means patients are often unable to tolerate them.

TCAs interfere with other receptors in the brain and this means that they tend to be sedative when they are first taken. It's a good idea to take these drugs just before you go to sleep. Curiously in some people, about one in ten, these antidepressants can actually be arousing , making them more alert.

TCAs also interfere with cholinergic receptors in the brain and they are commonly associated with anticholinergic side effects like, dry mouth, blurred vision, constipation, urinary difficulties and tremor. In addition, they tend to lower blood pressure which means that if someone stands up suddenly, they can produce a feeling of faintness and the elderly in particular can fall over, possibly even leading to bone fractures. Other problems include impotence.

Most importantly, TCAs are not safe when taken in overdose. In addition to what is mentioned above, the TCAs also have other effects on the heart inducing tachycardia (an increase in heart rate) and arrhythmia (abnormal rhythms of heart beat). Cardiotoxicity is the most common cause of fatality following TCA overdose.

The tricyclic antidepressants are usually used in more severe cases of depression, but are also prescribed in general practice.

Monoamine oxidase inhibitors (MAOIs) e.g. phenelzine
MAOIs work by inhibiting the action of an enzyme which is responsible for the breakdown of noradrenaline and serotonin in the brain. They are free of the anticholinergic effects described for the TCAs but side effects include insomnia, agitation and headache.

However the most worrying problem associated with these drugs is something called the cheese effect. Cheese and certain other foods contain a substance called tyramine which is normally broken down in the gut. However these antidepressants also inhibit this enzyme so the tyramine enters the body where it can cause a dangerous rise in blood pressure. Tyramine is also found in avocado, bananas, caviar, canned fig, liver and, wines and beers. This effect has severely limited the use of this antidepressant although moclobemide, a newer version of this class of drugs, is not associated with this effect and appears to be safe in this respect.

Selective serotonin reuptake inhibitors e.g. fluoxetine, paroxetine, sertraline, citalopram, fluvoxamine.
SSRI antidepressants were introduced in the late Eighties. These drugs are better tolerated and more acceptable to patients than the older TCAs. They have few of the anticholinergic effects, they lack the cardiotoxic effects and are probably safe in overdose.

However they are characterised by a group of side effects which have become known as the serotonergic side effects. One of these that has been the subject of much media coverage in recent years is the fact they can interfere with sexual functioning, usually by causing delayed ejaculation or orgasm. This is a particularly problematic side effect if these antidepressants are used for long term treatment.

Another major group of side effects is gastrointestinal side effects which vary from mild discomfort, nausea, weight loss and in extreme cases vomiting and diarrhoea.

Sleep disturbances are another important problem with the SSRIs particularly because it is a symptom of depression from which people commonly seek relief. In addition varying amounts of nervousness, anxiety and agitation occur in some patients early in treatment although they often subside relatively quickly as the body becomes accustomed to this assault.

There are many more side effects listed in the MIMS - so it is important to ask your prescribing doctor of the possible side effects and interactions with other drugs/foods. If he can't tell you - perhaps he should find out before writing the prescription for you.

PSYCHOLOGICAL INTERVENTIONS

The most common forms of psychotherapy are cognitive behavioural therapy and interpersonal psychotherapy.

Cognitive behavioural therapy is a short term (usually about 12-20 weeks) structured psychotherapy which aims to help the depressed person replace negative thoughts and attitudes with a more realistic view of themselves and the world about them.

Interpersonal psychotherapy focuses more on past and present relationships and examines how they affect the individual's current functioning.

LIFESTYLE FACTORS

A balanced lifestyle is of course a major consideration in overcoming any disorder. Setting realistic goals, adequate rest, diet and nutrition, and of course adequate exercise, all contribute greatly to a healthy mind and body.

NUTRITION

Foods profoundly influence the brain's behaviour. A poor diet - especially one full of junk foods, is a common cause of depression. The neurotransmitters which direct brain function, and regulate our behaviour are closely linked to mood, and are controlled by what we eat.

Recent research into the effects of different foods on the human system indicates that depression can be ameliorated by eating more of certain types of foods, and avoidance of others. Food sensitivities (allergies) are in fact the most common cause of depression - which suggests that susceptibility may be genetically based. Further, it has been demonstrated that genetic expression can be modified through diet and nutrition.[12]

- Of particular concern to the person who is depressed are gluten (wheat) and dairy products - these products should be avoided as they have been linked with the depressive disorders.

- Avoid diet soft-drinks. These contain aspartame which blocks the formation of serotonin causing headaches, insomnia and depression (not mentioning the link to cancer).

- Similarly, avoid products which contain phenylalinine which is one of the major components of aspartame.

- Avoid saturated fats ie fried foods etc. As consuming these leads to sluggishness, slow thinking and fatigue as saturated fats cause slow circulation of blood (nutrients) to the brain.

- Avoidance of all forms of sugar is recommended as sugar gives an instant high energy to the body which is immediately followed by fatigue and depression. Stevia - a concentrated natural sweetener derived from a South American shrub seems to be ok and without detrimental side effects.

- Avoid alcohol, caffeine and processed foods.

- Most medications prescribed for depression will deplete the body of essential vitamins and nutrients - if you must take medication, ask your doctor for specifics in this regard - if he can't tell you, then perhaps he should find out before prescribing it to you. Remember, ultimately your health is your responsibility.

BIOFEEDBACK

Biofeedback has a long history in mood affect disorders.

EEG BIOFEEDBACK

By training a person to regulate their own brainwaves and remain "in state" for longer periods of time, stability in brain structure communication is introduced, enabling the person to control their brain wave patterns at will. Several protocols have been used successfully worldwide for depression including the now famous Penniston Protocol.

Training is similar to stimulation, and constitutes a push that invokes the brain's innate capacity for restoring homeostasis. Over the longer term, this results in a long-term increase in stability. Training at a specific frequency is then a push in a very specific direction, which can be chosen in light of specific arousal disregulation or attentional deficits found in each case.

This brain exercise moves the individual into regions where he or she may not heretofore have been able to reside comfortably or stably. This is made possible not only by increased flexibility of state, but by an increased ability to maintain overall nervous system stability.

It is noteworthy that depression is among the easiest conditions to overcome with EEG biofeedback. These findings cover not only the mild depression that is frequently seen in connection with ADD, such as the dysthymia observed in childhood or the kind of low-grade pervasive depression for which Prozac has become the palliative of choice. They also cover episodes of deep depression, including some which are accompanied by episodes of suicidality, and even reactive depression.[13] (**CLICK HERE** to see a list of references to peer reviewed literature on the efficacy of EEG biofeedback for Depression).

HEG BIOFEEDBACK

Developed in the United States in 1977, hemoencephlagraphy is a relative newcomer to the field of biofeedback and works by training the person to increase blood flow to a targeted area of the brain, effectively substituting blood oxygenation for the same purpose as brainwaves in EEG biofeedback.
A spectrophotometer device is worn on the head during training. The technique provides a means of training selected brain areas merely by intensively willing it. Sessions are usually administered in 10 minute segments and 3-4 such segments constitute the session.[14]

Given that hypofusion of the executive areas of the brain (frontal lobes, temporal lobes) is a common trait amongst sufferers of depression it is easy to see the relevance of HEG biofeedback in the amelioration of this disorder.

For more information or to make an appointment please contact us on (02) 9637 9998 during business hours.

FURTHER READING SUGGESTIONS

- The Relationship between Vital Energy, The Brain and the Human Nervous System
- Counselling
- Dietary Supplements
- Biofeedback
- Neurofeedback - EEG Biofeedback - a Drug-Free Strategy for ADHD, Learning Disorders and Other Conditions

LINKS

PLEASE NOTE :
Learning Discoveries offers the links below as a convenience to our clients and the users of this website. However, we do not control third party websites and we are not responsible for the websites content.

- **DSM-IV, By the American Psychiatric Association**

  To view the DSM-IV criteria and revisions online please go to:

  http://en.wikipedia.org/wiki/DSM-IV_Codes

  DSM-IV is a coded reference manual published by the American Psychiatric Association to provide clear descriptions of diagnostic categories in order to enable clinicians and investigators to diagnose, communicate about, study, and treat people with various mental disorders.

- **ICD-10, By the Word Health Organisation**

  To view the ICD-10 criteria and revisions online please go to:

  http://apps.who.int/classifications/apps/icd/icd10online/

  The ICD - 10 is a coding of diseases and signs, symptoms, abnormal findings, complaints, social circumstances and external causes of injury or diseases, as classified by the World Health Organization.

- **Prozac Truth, by Jim Harper**

  http://www.prozactruth.com/

  http://www.prozactruth.com/antidepressants.htm

  Explains what the side effects are of antidepressants and why antidepressants are being prescribed by the millions. It explains why antidepressants are prescribed and how to taper off antidepressants.

REFERENCES

1. Sternberg, R.J., In Search of the Human Mind, 1995, Harcourt Brace and Co, Orlando, Florida

2. Crowe, S.F., 1998, Neuropsychological Effects of the Psychological Disorders, Harwood Academic Press, Melbourne.

3. Evans, J.R., & Arbarbanel, A., 1999, Quantitative QEEG and Neurofeedback, Academic Press, New York.

4. Schlegal, S. & Kretzschmar, K., 1987, Computed tomography in affective disorder, I. Biological Psychiatry, 22, 4-14.

5. Starkstein, S.E., & Robinson, R.G., 1997, Mechanism of disinhibition after brain lesions, Journal of Nervous and Mental Disease, 185, 108-114.
6. Mayberg, H.S., 1997, Limbic-cortical disregulation: A proposed model of depression, Journal of Neuropsychiatry and Clinical Neurosciences, 9, 471-481.

7. Robbins, J. 2000, A Symphony in The Brain, Atlantic Monthly Press, New York.

8. Bauer, M. S., & Whybrow, P.C., 1988, Biology of depression and mania. Current Opinion in Psychiatry, 6, 75-85.

9. Miller, B.L. & Cummings, J.L., 1999, The Human Frontal Lobes, The Guilford Press, New York.

10. Le Doux, J.E., Romanski, L., Xagoraris, A., 1989, Indelibility of subcortical emotional memories, Journal of Cognitive Neuroscience, 1, 238-243.

11. Balch, P.A., & Balch, J.F., Prescription for Nutritional Healing, 2000, Avery Publisher, New York.

12. Bland, J.S., et al, 1999, Clinical Nutrition: A Functional Approach. Institute For Functional Medicine, Gig Harbour, Washington.

13. Othmer, S., Kaiser, D.A., & Othmer, S.F., 2000, EEG Biofeedback: A Generalised Approach to Neuroregulation, EEG Spectrum July 2000 Training Manual, pp5.1 - 5.57

14. Toomin, H., 2001, Hemoencephlography -(HEG) The Study of Regional Cerebral Blood Flow rCBF & rCBO2, HEG Workshop, Society For Neuronal Regulation Conference, Monterey, October, 2001.

15. Pelton, R., & LaValle, J.B., 2000, The Nutritional Cost of Prescription Drugs. Morton Publishing Co, Englewood, CO.

ADDITIONAL REFERENCES : SPECIFIC TO THE EFFICACY OF BIOFEEDBACK AS A TREATMENT FOR DEPRESSION

1. Baehr, E., & Baehr, R. (1997). The use of brainwave biofeedback as an adjunctive therapeutic treatment for depression: Three case studies. Biofeedback, 25 (1), 10-11.

2. Baehr, E., Rosenfeld, J. P., & Baehr, R. (1997). The clinical use of an alpha asymmetry protocol in the neurofeedback treatment of depression: Two case studies. Journal of Neurotherapy, 2 (3), 10-23.

3. Hammond, D. C. (2001). Neurofeedback treatment of depression with the Roshi. Journal of Neurotherapy, 4 (2), 45-56.

4. Hardman, E., Gruzelier, L, Chessman, K., Jones, C., Liddiard, D., Schleichert, H., & Birbaumer, N. (1997). Frontal interhemispheric asymmetry: Self-regulation and individual differences in humans. Neuroscience Letters, 221, 117-120.

5. Jenkins, P., & Moore, W. H. (1985). The effects of visual feedback on hemispheric alpha asymmetries and reported processing strategies: A single-subject experimental design. Brain & Cognition, 4 (1),47-58.

6. Kotchoubey, B., Schleichert, H., Lutzenberger, W., Anokhin, A. P., & Birbaumer, N. (1996). Self-regulation of interhemispheric asymmetry in humans. Neuroscience Letters, 215, 91-94.

7. Kumano, H., Horie, H., Shidara, T., Kuboki, T. et al. (1996). Treatment of a depresive disorder patient with EEG-driven photic stimulation. Biofeedback & Self-Regulation, 21 (4), 323-334. 1

8. Rockstroh, B., Elbert, T., Birbaumer, N. L, & Lutzenberger, W. (1990). Biofeedback-produced hemispheric asymmetry of slow cortical potentials and its behavioral effects. International Journal of Psychophysiology, 9, 151-165.

9. Rosenfeld, J. P. (1997). EEG biofeedback of frontal alpha asymmetry in affective disorders. Biofeedback, 25 (1), 8-25.

10. Rosenfeld, J. P., Baehr, E., Baehr, R., Gotlib, 1. H., & Ranganath, C. (1996). Preliminary evidence that daily changes in frontal alpha asymmetry correlate with changes in affect in therapy sessions. International Journal of Psychophysiology, 23,137-141.