Aspects of Postpartum Depression - A Literature Review

Deane L. Burns
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For information regarding this report, please contact:
Bonnie Wooten
Manager
Young Families Team
Family Health Services Division
Middlesex-London Health Unit
Phone: 519-663-5317 Ext. 2248
E-mail: Bonnie.Wooten@mlhu.on.ca

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EXECUTIVE SUMMARY

Postpartum depression has received increasing attention from researchers, clinicians, and public health professionals. This document offers a review of the literature concerning postpartum depression with respect to the following categories: descriptive statistics, risk factors, biological model, treatment, and screening. As much as possible, postpartum depression is presented against the background of major depressive disorder as the two conditions have marked similarities. This is especially so with respect to their nosology, symptomatology, and treatment. However, there are differences between postpartum depression and major depressive disorder and these are addressed in this document.

Despite the centuries old history and awareness of depression as a psychological illness – including historical references that describe the depression associated with childbirth – and the sophistication of our research efforts – there are many questions that remain unanswered today.

What causes postpartum depression? It is not clear. But, several factors are probably involved. Some relate to biology others to psychosocial characteristics. For example, women with a personal history of postpartum depression may be at high risk for recurrence. Having a history of another mood disorder, such as major depression or bipolar disorder, may also place women at higher risk. Psychosocial risk factors that have been implicated include: poor spousal/social support, no spouse/partner, and a host of socio-demographic characteristics that appear as risk factors in some study populations but not in others. The involvement of various labour and delivery events/conditions also reveals inconsistent results.

The role of biology may contribute to the onset of postnatal depression. Genes, hormones, neurotransmitters and other physiologic elements thought to influence mood, comprise a profoundly complex system. Women who develop postpartum depression may be particularly vulnerable to the hormonal changes that occur with child delivery.

Treatments for postpartum depression borrow heavily from the treatment approaches already in place for major depression. However, women with postpartum depression may receive shorter courses and lower dosages of antidepressant medications than non-puerperal patients. Psychological treatments such as cognitive behavioural therapy and interpersonal therapy show some promise but this work needs to be replicated with well-designed trials. Other work that suggests a lasting beneficial effect should be followed by studies that confirm this benefit including its duration.

Researchers and clinicians agree that early detection can lead to early treatment and reduced deleterious effects for the patient, her infant, other children, and her spouse. The Edinburgh Postnatal Depression Scale and Beck's Postpartum

Depression Screening Scale are two instruments used during the postpartum. Although it may be beneficial to use either, the implementation of a screening program raises some concerns. Who should screen, when to screen, how many times, and what supports are in place should the demand for professional and community resources increase.

While several medical science and community health professionals may wonder if they should be ‘doing something different’ – the process and method to address this question is another complexity for the mix. Should new roles or new tasks be defined using a comprehensive needs-impact based planning process?

What elements of the research should be included and how should disparate and conflicting findings be managed within the decision-making framework? These questions lay in the wake of our attempts to re-examine current priorities in order to address the challenges posed by postpartum depression.

DEPRESSION: A MOOD DISORDER

For most people, moods are temporary emotional states in response to ordinary living. However, a mood state that becomes an involuntary way of "being" may represent a psychological illness.

A depressive mood disorder is an illness involving the body, mind, mood, and thoughts. In fact, disturbed mood of sufficient duration, severity, and persistence can be a disabling illness that interferes with daily life.[1]

Mood disorders with depressive symptoms are classified in the Diagnostic and Statistical Manual IV-TR into three main categories. These include major depressive disorder, dysthymic disorder, and depressive disorder not otherwise specified. [2]

Postpartum Depression

Postpartum depression is classified as a sub-type of major depressive disorder. The criterion for this sub-type - onset of depression within four weeks postpartum – is a special descriptor or specifier.[3]

Postpartum psychological illness was initially conceptualized as a group of disorders specifically linked to pregnancy and childbirth. They were considered diagnostically distinct from other types of depressive disorders. However, current evidence suggests that the characteristics of mood disturbance that emerge during the puerperium do not differ significantly from non-puerperal mood disorders. In addition, population based studies have revealed similar rates of minor and major depression in puerperal and non-puerperal cohorts.[4]

Depression: Symptoms and Diagnosis

Depression is not a new illness. Hippocrates made one of the earliest references to depression – melancholia – during the 5th Century BC.[5]

An episode of major depressive disorder of sufficient intensity and duration can adversely affect many core aspects of one's life – thoughts, feelings, self-esteem, relationships, work life, physical health, and behaviour.

In order to distinguish major depression from feeling low or a situational period of grief, a major depression must meet a threshold of severity, last for at least two weeks, and involve the symptoms listed in Table 1.

Table 1
Criteria for Major Depression

Depressed mood; feeling sad; feeling empty or
Marked loss of interest or pleasure in activities enjoyed in the past

Plus at least 4 of:

Changes in appetite: weight loss or weight gain
Trouble falling asleep; sleeping too much; early waking
Psychomotor agitation or retardation
No energy; lethargy; fatigue
Feelings of worthlessness; guilt; hopelessness
Difficulty concentrating; difficulty making decisions
Recurrent thoughts of death; suicidal ideation

The symptoms do not meet the criteria for other psychiatric conditions.
The symptoms impair functioning at work, school, and social activities.
The symptoms are not caused by substance or a medical condition.
The symptoms are not accounted for by bereavement.

Source: http://www.currentpsychiatry.com/images/pdf/cp0502/postpartum/0502_postpartum_tb2.pdf
Adapted from: *Diagnostic and Statistical Manual of Mental Disorders*. 4th Ed
Text revision. Washington: American Psychiatric Association, 2002

The cardinal symptom(s) of major depression is depressed mood and/or loss of interest or pleasure. Other symptoms can vary within these criteria.[6] Researchers believe that depression may have a number of complex and interacting causes.[7]

Postpartum Depression

The term postpartum depression usually refers to three postpartum psychological conditions: postpartum blues, postpartum depression, and postpartum psychosis. These range in severity, duration, and treatment. Some describe these disorders as a "continuum". Although, severe postpartum blues may be followed by postpartum major depression[8] a woman is not destined to experience one illness then the other along the continuum.

Postpartum Blues

Postpartum blues is a period of heightened emotion occurring in up to 85% of women. This condition peaks at 3 to 7 days following delivery and can last for up to 10 days to 2-3 weeks. Symptoms include: always being "on the verge of tears", crying spells, sleep disturbance, fatigue, and headache. Feeling sad or irritable is also present. [9]

However, a woman with the "baby blues" is not disproportionately stressed, anxious, or adversely overwhelmed. Also, severe depressive feelings are not

associated with postpartum blues. Treatment is not indicated as the condition is said to be self-limiting and recovery appears to be complete.

Postpartum Depression (Postpartum Major Depression)

Postpartum depression occurs in 10 -15% of women.[10] Onset can range from 24 hours following delivery to 4 - 6 weeks or 6 -12 months postpartum.[11] However, symptoms are generally seen within the first month.[12] Onset can be abrupt or gradual. Untreated postpartum depression may last for 3 to 14 months.[13]

Symptoms of postpartum depression include: depressed mood, tearfulness, mood swings, inability to enjoy activities that used to be of interest, sleep disturbance, fatigue, difficulty concentrating, and altered appetite.

Anxiety, obsessive worry about the baby's health, and/or a profound sense of guilt may also be present. These symptoms are not typical of major depression *per se* but can be part of postpartum depression [See Table 2]. Feeling inadequate and overwhelmed are other symptoms.[14]

A mother may also be afraid to be alone with the baby, or she may think about harming her infant. However, postpartum depression is a non-psychotic depression and only very rarely will a woman carry out harmful thoughts.[15]

It is important for a clinician to distinguish between the expected upsets reported by a new mother and the onset of clinical depression. For instance, detailed questioning may reveal the difference between a patient who cannot sleep because of night-time feedings versus a mother who cannot sleep even when she has the chance i.e., when baby is sleeping.[16]

Postpartum Psychosis (Puerperal Psychosis)

Approximately 1-2 per 1,000 women develop postpartum psychosis. In terms of disabling symptoms, need for treatment, and irreparable consequences, it is considered the most serious form of postpartum psychological disturbance.[17]

The onset of postpartum psychosis can occur 2-3 days or up to 4 weeks following childbirth. In the majority of cases symptoms appear within 2 weeks.[18]

Early onset symptoms include: restlessness, irritability, and difficulty sleeping. Subsequent symptoms evolve rapidly presenting as depressed or profoundly elevated mood and disorganized/confused behaviour. [19] Some women may appear as manic despite the depressive classification of this illness [20.]

Table 2
Major Depression and Postpartum Depression

Symptoms of Major Depression may include:	In addition, PPD may include:
• Sluggishness, fatigue, exhaustion • Sadness, depressed mood, hopelessness • Poor concentration, indecisiveness, or confusion • Memory loss • Uncontrollable crying, irritability • Agitation or slowed movements • Recurrent thoughts of death or suicide • Significant weight loss when not dieting, or weight gain, or decrease or increase in appetite • Markedly diminished interest or pleasure in all or almost all activities • Exaggerated highs and/or lows • Lack of interest in sex • Insomnia or hypersomnia • Feelings of worthlessness or excessive or inappropriate guilt	• Excessive concern for the baby or excessive anxiety over the baby's health • Guilt, inadequacy, worthlessness, feeling like a failure at motherhood • Fear of losing control or "going crazy" • Irritability • Lack of interest in the baby • Fear of harming the baby • Diminished libido • Obsessive thoughts

From: Kennedy, R.S., & Suttenfield, K. (2001). Postpartum Depression. *MedGenMed*, 3(4), 2001

http://www.medscape.com/viewarticle/408688

The psychotic element includes having false beliefs (delusions) about the baby and/or seeing or hearing things that are not there (hallucinations). For example, a woman may believe her child is evil. Or, she may see recurrent and vivid images of blood and weapons.[21]

Other symptoms can appear as: severe/profound sleep disturbance, i.e., no sleep for several days, agitation, appetite changes, low libido, no energy, and mood swings.

In contrast with postpartum major depression, women may carry out the harmful actions their recurrent thoughts or visuals suggest. A woman may hurt or kill her child and herself. Other children in the family may also be harmed or killed.

Less severe cases of postpartum psychosis are often dismissed as part of the normal maternal adjustment associated with childbirth.[22]

However, postpartum psychosis is a psychiatric emergency that requires immediate medical attention - hospitalization and treatment. It can lead to some of the most severe disturbances seen in psychiatry.[23]

POSTPARTUM DEPRESSION: RISK FACTORS

Research suggests that women with various *risk factors* – characteristics related to health and environment - may have a higher likelihood of developing a postpartum depressive disorder. A risk factor may involve biology, lifestyle, or some aspect of the social and family environment, i.e., psychosocial.

Although some contradictory results are present among various studies, it is generally believed that risk factors can help identify women who may develop postpartum depression.[24, 25] Consequently, these women may receive appropriate care during pregnancy or follow-up after delivery.

Previous Postpartum Depression

Citing the work of Llewellyn, Stowe, & Nemeroff (1997), and O'Hara, Neunaber, & Zekoski (1984), Stocky and Lynch reiterate that women who report a previous episode of postpartum depression may be at increased risk to experience a recurrence of postpartum depression.[26]

Various researchers have estimated the re-occurrence rate of postpartum depression. Women with a history of postnatal depression may be at 50% risk to experience a subsequent depressive episode related to childbirth.[27, 28] Epperson (1999) concluded that approximately one third of women may experience another depression associated with a future delivery.[29]

Although researchers agree that a history of postpartum depression a risk factor others cautiously maintain that the magnitude of this risk is unclear.[30]

Previous Psychiatric Illness

Women with previous psychiatric illness are also at greater risk to develop postpartum depression. Women with a history of mood disorder are especially at increased risk.[31]

Recurrence rates for women with bipolar disorder range from 20 - 50%.[32] Referring to work by Cohen et al. (1995), Epperson cites a recurrence rate of 60% for bipolar women.[33]

Women with a history of postpartum psychosis appear to be at greatest risk for recurrence of postpartum depression. Nonacs and Cohen (1998) cite previous work that estimates a 70% relapse rate for this subgroup of women.[34]

Steiner (2002) found that 78% of women diagnosed with postpartum depression had either a personal history of psychiatric illness or a close family member had a history of psychiatric illness.[35]

Fossey et al. (1997) showed that severe maternity blues present at 5 days postpartum was also present at 8 months postpartum. They hypothesized that having a severe case of the 'maternity blues' may be a predictor, i.e., risk factor, for postpartum depression.[36]

Psychosocial Risk Factors

Researchers have examined the influence of various psychosocial characteristics that may be associated with postpartum depression.

Low Social/Spousal Support

In recent work, Epperson (1999) reiterates findings from O'Hara (1986) suggesting that dissatisfaction with the marital relationship and poor social support increases a woman's risk to develop postpartum major depression.[37]

Citing five previous studies (including two from O'Hara), Nonacs (1999) agrees that women with inadequate social supports and poor marital relationships appear to be particularly vulnerable to develop postpartum depression.[38]

Single Parent/Low Income

Susman (1996) maintains that postpartum affective illness can be more pronounced in single parents or mothers with low economic status.[39]

Additional research lists the suspected involvement of other psychosocial risk factors. These include: poor communication with the spouse[40], loss of a mother or other close family member[41], having other children in the home, low educational level, being an adolescent mother[42], having low life-satisfaction in general[43], being stressed by the new role[44], feeling that this pregnancy was unintended or unwanted[45], and delivery of a low birth weight infant.[46]

Others have found that mothers who had used a high number of sick leave days during pregnancy[47], or made excessive use of the hospital emergency room, or several paediatric/antenatal visits[48] were at risk to develop postnatal depression.

C. T. Beck offers this list as risk factors: previous prenatal depression, previous depression, lack of spousal/social support, life stress, child care stress, severe maternity blues, marital dissatisfaction, and prenatal anxiety. [48a]

Labour/Obstetric Risk Factors

Contradictory results have been found with respect to labour and delivery events and their possible role in postpartum depression. Some research suggests that complications of labour have not been shown to predict the occurrence of postpartum depression. Other research suggests that obstetric factors may increase the risk of postpartum depression.

For example, emergency caesarean section appeared as a risk factor in work completed by Boyce & Todd (1992) but not in work completed by Josefsson et al. (2000). [49]

Risk Factors: Interpret with Caution

Apart from their descriptive value, the clinical utility of some psychosocial risk factors seems limited in light of the contradictory results obtained across several studies. Some work supports the contention that socio-economic and demographic variables may influence the development of postpartum affective illness and other work does not - or finds no association.

Also, the magnitude or strength of the predictive value of some risk factors is unknown. The role of multiple risk factors (having multiplicative or additive effects) is also not clear. Subsequently, the role of protective (as opposed to risk) factors is largely undetermined.

Study design, small sample size, how the sample of women was selected (convenience or random), and the characteristics of the study sample can also limit the ability of research findings to be extended to the general population of women.

POSTPARTUM DEPRESSION AND BIOLOGY

Researchers acknowledge that it is unlikely that one cause can account for the complexity that depression presents as an illness. Instead, a combination of genetic, psychological, and environmental factors is most likely involved in the onset of a depressive disorder.[50]

Some research explores the biological etiology of depression. Evidence from neuroscience, genetics, and clinical investigation demonstrate that depression is a disorder of the brain. Modern brain imaging technologies are revealing, that in depression, neural circuits responsible for the regulation of moods, thinking,

sleep, appetite, and behaviour fail to function properly, and that critical neurotransmitters – chemicals used by nerve cells to communicate – are out of balance.[51]

Genetic Link and Depression

Having a family history of depression is considered to be a risk factor for depression. However, no specific gene has been identified as "the gene for depression". In addition, studies that examine depression in families and twins do not conclusively demonstrate that depression is expressed in subsequent generations through a genetic link. Yet, researchers suspect that a gene(s) may be involved and work is continuing in this area.[52] For example, some genetics research suggests that a vulnerability to depression may result from the influence of multiple genes acting together with environmental factors.[53]

Depression and Hormones

Hormonal factors may contribute to the increased rate of depression in women - particularly such factors as menstrual cycle changes, pregnancy, miscarriage, the postpartum period, pre-menopause, and menopause.[54]

Women appear to be at increased risk to develop depression, as the rate of depression in women is approximately twice the rate seen in men.[55] One possible hypothesis that may account, even in part, involves the different roles that various sex hormones in male and female biology. Specifically, hormones related to the female reproductive system.[56]

Unlike males, women's biology is marked by hormonal fluctuations that occur during various events along the female lifespan. For example, before menstruation levels of estrogen and progesterone are lowest. It is hypothesized that the onset of premenstrual dysphoric disorder (premenstrual syndrome) may be related to the drop in estrogen. Similarly, depression at menopause may be related to the decreased production of estrogen. Finally, postpartum depression may be related to the substantial decrease in estrogen that occurs after childbirth. [57] (Levels of estrogen may fall 1000-fold within 48 hours after birth.[58])

Hormones and Mood Regulation

Sex or gonadal hormones and mood regulation are related. They are aspects of the neuroendocrine system. This system regulates multiple functions of the mind, the brain, and the body, and can influence the way nerves function. In particular, gonadal hormones have a pronounced effect on the central nervous system – including the areas responsible for mood and cognition.[59]

Estrogen and its involvement with serotonin regulation has been studied extensively. Fluctuating levels of estrogen may interfere with serotonin regulation and this may influence the onset of depression.[60]

The behaviour of other hormones (e.g., progesterone, cortisol) and other neurotransmitters (e.g., norepinephrine, dopamine) is also part of the biological aspects of mood regulation and the onset of depression.[61]

Hormones and Postpartum Depression

Some researchers suspect that postpartum depression is associated with the rapidly changing hormonal environment during the postpartum.[62]
Since depression may be related to falling levels of estrogen, then the onset of postpartum depression may be related to the rapid decline of estrogen that occurs at childbirth.[63]

Ultimately, the occurrence of postpartum depression may be associated with three conditions. These include the abrupt hormonal shifts that occur after delivery, the intense psychosocial stresses related to childbirth and motherhood, plus an unknown underlying vulnerability that contributes to postpartum depression in some women and not others.[64]

POSTPARTUM DEPRESSION: TREATMENT

Treatment options for postpartum depression include antidepressant medications, various forms of psychological counselling, and electroconvulsive therapy. These three treatment modalities are also used to treat non-puerperal depression.[65]

Medications for Postpartum Depression

There are three main classifications of antidepressant medications. They are: tricyclic antidepressants, serotonin reuptake inhibitors, and the novel (or atypical) agents.

Serotonin Reuptake Inhibitors (SSRI)

During the early 1980s, European scientists developed a class of drug – serotonin reuptake inhibitors (SSRIs) - that affect the mood-modifying neurotransmitter serotonin. In North America, during the late 1980s, the Eli Lilly Company developed the SSRI called fluoxetine (Prozac®). [66]

The selective serotonin reuptake inhibitors (SSRIs) have become the first-line agents for the treatment of depression because of their favourable side-effect profile, ease of use, and proven efficacy.[67]

However, despite the similarity between major depressive disorder and postpartum depression, postpartum depression is not always treated for the same duration and at similar dosages as non-puerperal major depression. As the

data do not suggest that an episode of postpartum major depression should be managed differently, women with non-psychotic postpartum depression should be treated for similar periods of time and with comparable doses as prescribed for patients with non-puerperal depressive illness.[68]

The effectiveness of antidepressant medications to treat postpartum mood disturbance has been demonstrated in both open trials and double-blinded studies. Several studies demonstrate the efficacy of fluoxetine and sertraline. In all of this research, standard doses of the antidepressant medication were effective and well tolerated.[69]

The use of SSRIs should be seriously considered to treat women who have moderate to severe postpartum depression, especially if they have suicidal thoughts, difficulty functioning, or have not responded to nonpharmacologic treatment.[70]

Breastfeeding and SSRI Medications

All psychotropic medications (including SSRIs) are excreted in breast milk and their use during breastfeeding is a significant concern.[71] Whether or not a mother should breastfeed while taking SSRIs is ultimately a risk-benefit decision. The risks of untreated depression (inhibited mother-infant attachment, alcohol/smoking use during untreated depression etc.) must be compared to the benefits, i.e., the safety of not exposing the infant to any medication.[72]

Although the use of SSRIs is not expressly contraindicated for mothers who wish to breastfeed, it is important to acknowledge that no research has demonstrated that infants are not at risk from breastmilk that contains undetectable concentrations of SSRIs and their metabolites. A child will be exposed but the short-term and long-term effects are unknown.[73]

However, mothers who take a SSRI and also wish to breastfeed can minimize infant exposure by waiting until after the medication peaks in her system (breastmilk concentration). In the case of sertraline, waiting 1 to 9 hours after taking a dose may limit the infant's exposure to the medication.[74]

Tricyclic Antidepressants

During the late 1950s, a tricyclic compound was used to treat psychiatric patients. It appeared to work by blocking the reuptake of two neurotransmitters - norepinephrine and serotonin. As a result, imipramine became the first of many tricyclic antidepressants.[75] During the 1960s and 1970s, they were the first-line class of antidepressants.[76] Today, tricyclics such as amitriptyline and clomipramine are second-line agents used to treat major depressive disorder.[76]

Replaced by first-line antidepressants, tricyclics and their role in the management of postpartum depression is not a current area of research.

Pregnancy, Breastfeeding, and Side Effects

Although tricyclic antidepressants have been available for almost fifty years, research that examines their use during pregnancy is still plagued with methodological difficulties. Some fetal abnormalities have been reported, but no clear associations have been demonstrated.[77]
Breastfed infants have undetectable serum levels. However, short-term and long-term effects of exposure are unknown and it is recommended that a mother express breastmilk immediately before she takes this medication or wait 4 hours after a dose before breastfeeding her infant.[78]

Side effects such as dry mouth, sedation, and postural hypotension are more pronounced in pregnant women and can cause withdrawal symptoms of tachypnea, tachycardia, cyanosis, irritability, and diaphoresis in the neonate.[79]

Novel (or Atypical) Agents

The novel agents include Wellbutrin (bupropion) and venlafaxine. Wellbutrin (bupropion) is a norepinephrine dopamine reuptake inhibitor used to treat depression. It has no clinically significant impact on serotonin.[80, 81] The increased risk of seizures associated with bupropion deters its use during pregnancy, especially in women subject to eclampsia.[82]

Cohen et al. found improvement in screening scores after an 8-week course of venlafaxine. [83]

Hormone Treatments for Postpartum Depression

Since the onset of postpartum depression may be related to the drop in estrogen and progesterone the use of hormonal therapy has been considered as a treatment.

Estrogen Hormonal Therapy

The use of estrogen to treat a variety of psychiatric disorders including postpartum depression has received renewed interest over the past few years.[84]

A double-blinded study compared the use of 17-β estradiol with a placebo in 61 women with major postpartum depression. Within three months, 80% of the women receiving estrogen (via an estrogen patch) scored below the threshold for depression using the Edinburgh Postnatal Depression Scale. The placebo group did not fare as well – 31% scored below the scale's depression cut point.

Although this may suggest that the estrogen treatment was better than no treatment - the placebo - nearly half of the estrogen-treated patients were taking antidepressant medication. This confounding aspect makes it difficult to assess whether estrogen alone was effective.[85]

An 8-week study by Ahokas et al. involved 23 women with major depression (with documented estrogen deficiency) who were treated with sublingual 17-β estradiol. A significant reduction in depression was seen during the first week, and by the end of the second week 19 of 23 patients were considered clinically recovered.[86]

In addition, findings from a controlled study by Gregoire et al. and an open-label study by Sichel et al. suggest that high doses of estrogen may be effective to treat or prevent postnatal depression, However, serious methodological shortcomings limit these findings to be generalized. The need for an anticoagulant medication is also a concern with high-dose estrogen therapies.[87]

Progesterone Hormonal Therapy

A study conducted by Dalton used progesterone treatments to prevent the reoccurrence of postnatal depression. Treated women had a reoccurrence rate of 7% compared to 67% of untreated women. This study had serious methodological limitations and its findings have not been duplicated since its completion in 1989.[88]

Recently, Lawrie et al. maintained that there is no place for synthetic progestogens in the prevention or treatment of postnatal depression. They found that long-acting norethisterone enanthate is associated with an increased risk of postnatal depression. The role of progesterone has yet to be evaluated in a randomized placebo-controlled trial.[89]

Psychotherapy for Postpartum Depression

These non-pharmacologic treatments for depression and postnatal depression include interpersonal psychotherapy and cognitive behavioural therapy. Aspects of psychodynamic and experiential psychotherapies are widely practiced but have not been adequately evaluated. As such, they do not represent a current area of research to include here.

Cognitive Behavioural Therapy

Cognitive Behavioural Therapy (CBT) is a brief psychotherapy that borrows theory and technique from both cognitive therapy and behavioural therapy.

The *cognitive* aspect of CBT is based on the supposition that a person's thoughts are directly connected to how one feels. Therefore a patient/client is assisted to identify and correct distorted thinking that leads to emotional discomfort.

The *behavioural* aspect of CBT is based on the premise that primary learning comes from repeated experience. Further, maladaptive behaviours can be unlearned by taking on new behaviours that provide rewarding or positive outcomes.

Compared to older more traditional theoretical orientations CBT tends to focus on current life concerns troubling the patient/client rather than historical aspects of conflict and early life experience.[90]

Cognitive Behavioural Therapy and Postpartum Depression

CBT can address a woman's unrealistic expectations regarding her new role and alleviate feelings of guilt when she needs to save time for herself or significant other. In addition, a woman can be encouraged to avoid self-blame when she is unable to accomplish all of her expanded responsibilities. [91]

Appleby and colleagues demonstrated that short-term CBT is as effective as fluoxetine in the treatment of postpartum depression. [92] In other work, health visitors provided CBT-based counselling. At the end of 12 weeks the women randomized to the six-session CBT group showed more improvement than the one-session group.

However, there are few, if any, well-designed studies that demonstrate the successful treatment of postpartum depression using CBT. However, its success with non-puerperal depression may mean success with postpartum depression.[93]

Furthermore, as CBT interventions may have an enduring effect beyond the treatment of (non-puerperal) depression, and considering that depression may reoccur, teaching people to deal with or prevent their own affective distress could be a real boon to public health. [94]

Interpersonal Therapy

Interpersonal Psychotherapy (IPT) is a short-term therapy proven to be effective for treating acute episodes of depression.[95] It helps a person deal with changing roles and relationship stressors by learning how to communicate more effectively.

O'Hara (2000) et al. found that a 12-week course of interpersonal therapy was an efficacious treatment for postpartum major depression. One hundred and twenty women were randomized to either a waiting list or the treatment group. The resulting scores using the Hamilton Rating Scale for Depression were significantly lower for the women receiving interpersonal therapy. Scores were also significantly lower using the Beck Depression Inventory.[96]

In a controlled trial, Spinelli and Endicott randomized 50 depressed pregnant women to a treatment group or a control group. Those who received interpersonal psychotherapy showed significant improvement compared to a control program.[97] However, the authors caution that this work was preliminary and that its small sample size and large attrition rate inhibits the findings to be generalized to the greater population.

Electroconvulsive Therapy

The ancient Greeks and Romans induced seizures in patients to treat illness. For example, headache was treated by the convulsions produced by holding an electric torpedo fish against a patient's temple. Since then, the generation of seizures to bring about a therapeutic effect has appeared throughout the centuries.[98]

During the 1930s, the use of induced seizures to treat *mentally* ill patients became common. Insulin shock therapy and the use of metrazol were used to *chemically* induce seizures. These treatments were eventually replaced by *electrically* induced seizures by way of electroconvulsive therapy. The use of electroconvulsive therapy became widespread in North America during the 1940s.[99]

Today, over fifty years after its introduction, electroconvulsive therapy remains a controversial method of psychiatric treatment.[100]

What is Electroconvulsive Therapy?

Electroconvulsive therapy is one of the most effective yet most stigmatized treatments for severe depression. Electroconvulsive therapy produces a seizure in the brain of a patient under general anaesthesia by applying electrical stimulation through electrodes placed on the scalp. Repeated treatments are necessary to achieve the most complete antidepressant response.[101]

The technique and the associated anesthesiology interventions are so highly refined that electroconvulsive therapy is considered a safe and effective treatment for patients with major depressive disorder, manic episodes, schizophrenia, and other serious mental disorders. Up to 90% of patients suffering from major depressive disorder respond favorably to electroconvulsive therapy. Further, there are no studies showing any treatment to be superior to electroconvulsive therapy in the short-term treatment of severe depression.[102]

Although its use was largely replaced during the 1950s and 1960s by psychotropic medications, interest in electroconvulsive therapy was renewed so that medication-resistant cases (of depression) could be treated. [103]

As with most biological treatments in psychiatry, however, we still do not know precisely how electroconvulsive therapy works.[104]

Electroconvulsive Therapy and Postpartum Depression

The American Psychiatric Association practice guidelines (1993) suggest electroconvulsive therapy as a primary treatment for major depression (and bipolar disorder) during pregnancy. It has a high efficacy and low risk in the management of these disorders during all three trimesters of pregnancy, as well as postpartum. Potential risks include: spontaneous abortion, preterm labour, uteroplacental insufficiency, or placental abruption.[105]

Electroconvulsive therapy should be considered early for those suffering from severe postpartum illness. Three treatments over four days is a recommended in-hospital course. In the case of puerperal psychosis, electroconvulsive therapy is very useful and time-efficient. Failure to treat aggressively places mother and baby at increased risk of harm. [106]

SCREENING for POSTPARTUM DEPRESSION

Two common screening tools designed to detect symptoms related to postnatal depression are available today. A score indicative of depression would alert a health care professional that a woman is experiencing a high level of distressing symptoms that *may* be diagnosed as a major depression. [107]

The Edinburgh Postnatal Depression Scale

Developed by J.L. Cox et al. (1987) the Edinburgh Postnatal Depression Scale (EPDS) is a well-validated screening tool used during the postnatal period to identify women who may be depressed. It is a 10-question self-report questionnaire that rates the intensity of various depressive symptoms such as mood, anxiety, and suicidal ideation. Each question has four choices that are scored from 0 to 3 with a possible total score of 30. It can be completed in 5 minutes. Generally, a score of ≥12 may indicate depression. The sensitivity of the EPDS at this score is 87% with a positive predictive value of 73%. [108]

C.T. Beck's Postpartum Depression Screening Scale

The Postpartum Depression Screening Scale (PDSS) is a recently developed self-report screening tool from C. T. Beck and R. K. Gable. It is a Likert-type scale containing 35-items. There are seven symptom areas that relate to postpartum depression: sleeping/eating disturbances, anxiety/insecurity, emotional lability, mental confusion, loss of self, guilt/shame, and suicidal thoughts. A score of ≥80 may indicate depression. The PDSS has a sensitivity (can detect depression) of 94% and a specificity (can detect depression-free women) of 98% and a positive predictive value (percentage of those who score ≥80 and later diagnosed with depression) of 90%. [109, 110] Women who score as

'not depressed' should be re-tested every three months during the first year postpartum.

Many researchers support the contention that screening would help to identify women at risk, thus leading to appropriate diagnosis and treatment. In addition, the American College of Obstetrics and Gynaecology states that all women should be considered at risk for postpartum depression, and that all postpartum women should be screened.[111]However, this position raises critical questions.
Screening Issues: Who and When to Screen

Among the eligible clinicians who could administer either the EPDS or the PDSS are: nurse midwives, family practice/obstetrical and gynaecological physicians, nurse practitioners, hospital nurses, public health nurses, prenatal care coordinators, clinic nurses at well baby clinics, lactation educators, and home visitors. Is there one in particular who should take the lead?

Or, given that postpartum depression could appear up to 12 months after delivery, should *everyone* screen at every available opportunity?

Some clinicians advise to screen at 6 weeks and again at 3 months. To identify depression in the most women possible, others suggest that four screens be done: the first prenatal visit, during the third trimester, at 6 weeks postpartum, plus one more screen during the first year postpartum.

If only *one* screening is done postpartum, the 6-week postpartum visit is the optimal time. However, women also see their own physician and their child's paediatrician. These are also opportunities to screen. [112]

Screening Issues: Availability of Mental Health Resources

If screening detects a substantial number of women who may have major depression or minor depression, a response plan should be in place. For some communities this may be challenging. Are there enough physicians, psychiatrists, public health professionals, and community resources to deliver care? Are waiting times "too long". If so, perhaps women should at least be provided with local information regarding support groups or other programs and services in their community that may be helpful.[113]

DISCUSSION

The body of literature concerning postpartum depression contains several references that describe it as a "hidden" illness with women needlessly suffering in silence. Other statements claim that postpartum depression is substantially undetected, under diagnosed, and under treated. With the consistently

demonstrated prevalence of postpartum mood disturbance - even across different cultures - it is striking (that) it is so commonly missed.[114]

However, postpartum depression is a complex and variable illness. Despite the centuries old history of non-puerperal depression, postpartum depression has only recently – within the last ten years – been assigned a designation under the DSM-IV. Yet, debate is still present regarding its merit as a distinct depression apart from other depressive episodes experienced by women during their lifespan.

Despite their sophistication, efforts to explore a biologic etiology of depression represent a developing area of research that attempts to examine brain and body influences upon mood. The profoundly complex interaction among hormones, neurotransmitters, and a host of other physiologic aspects of mental health, has yet to be elucidated - not only in the case of postpartum depression, but also for depression and mental illness in general.

The exploration of psychosocial factors offers a powerful hypothesis that characteristics external to our biology can impart a substantial and deleterious effect upon mental health. Establishing major and contributing risk factors has, on the whole, contributed to our knowledge of postpartum depression, but additional work is required. Well-constructed research offering consistent results that can be generalized to the greater population of women must be completed.

However, limitations in study design such as small sample size, non-random selection, no randomized assignment, appropriate comparison groups, and attrition, are common weaknesses in postpartum depression research.

Core aspects of inquiry – the presentation, onset, course, duration, treatment, and prognosis for postpartum depression are mired in unknowns that obstruct our understanding. As a result, these unresolved aspects confound diagnosis, delay treatment, and prolong the debilitation and misery associated with major depression.

An additional facet often complicates this scenario. C.T. Beck maintains that depression is *the thief that steals motherhood*. The effects of major depression can impair a mother's ability to provide vital necessities – both emotional and task-related duties – to achieve optimal infant health. Other children in the family may also be adversely affected and the spousal relationship can be disrupted.

But, researchers and clinicians concur that the use of a screening tool to detect depression is a quick and simple unobtrusive procedure. Consensus is apparent: it is worthwhile to identify women who may be at risk for postpartum depression - even *during* their pregnancy. Agreement is also evident regarding the screening tool to use - both the EPDS and Beck's PDSS are available and offer acceptable validity.

Despite the numerous health care professionals who *could* offer a screening test it seems that most do not. Some might raise the topic during patient visits even though research has demonstrated that the systematic use of a screening tool can detect more depression in a patient population compared with clinical interviews alone.

With the recent emphasis on women's health issues, and child and maternal health services, perhaps, both clinical and public health practice could offer a co-ordinated and systematic screening effort.
However, if screening detects more women who need care, then additional clinical and public health resources may be needed to provide it.

Since undetected and untreated postpartum depressive illness can have adverse consequences for mother, her infant, and other family relationships, it is becoming increasingly important to offer appropriate and co-ordinated resources to address the needs of those affected.

Works Cited

1 The Mood Disorders Association of Ontario. (2003). What is a Mood Disorder? Source: *Kaplan & Sadock. (1998). Synopsis of Psychiatry.* http://www.mooddisorders.on.ca/whatis.htm Last accessed: November 12, 2003.

2 Parikh, S., Lam, R.W., & CANMAT Depression Work Group. Clinical Guidelines for the Treatment of Depressive Disorders. (2001). *Canadian Journal of Psychiatry*, 46, S1, 13S-S37.

3 Fenna, C. (2002). Postpartum Depression. *Behavioral Medicine Briefs.* University of Minnesota. http://www.med.umn.edu/fp/bmb/bmb23.pdf Last accessed: November 12, 2003.

4 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S 2, 34-40. http://www.emedicine.com/med/topic3408.htm#section-postpartum_psychosis Last accessed November 12, 2003.

5 David Satcher. (1999). Mental Health: A Report of the Surgeon General. http://www.surgeongeneral.gov/library/mentalhealth/chapter4/sec3.html Last accessed November 12, 2003.

6 David Satcher. (1999). Mental Health: A Report of the Surgeon General. http://www.surgeongeneral.gov/library/mentalhealth/chapter4/sec3.html Last accessed November 12, 2003.

7 National Institute of Mental Health. Depression Research. (2002). http://www.nimh.nih.gov/publicat/depresfact.cfm No. 00-4501. Last accessed November 12, 2003.

8 Fossey, L., Papiernik, E., & Bydlowski, M. (1997). Postpartum blues: a clinical syndrome and predictor of postnatal depression? *Journal of Psychosomatic Gynecology*, 18, 17-21.

9 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment Guidelines. *Journal of Clinical Psychiatry*, 59, S 2, 34-40.

10 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S 2, 34-40.

11 Epperson, C.N. (1999). Postpartum major depression: Detection and treatment. *American Family Physician.* 59, 2247-2254.

12 Georgiopoulos, A., Bryan T., Yawn, B., Houston, M., Rummans, T., & Therneau, T. (1999). Population-based screening for postpartum depression. *Obstetrics and Gynecology*, 93, 653-657.

13 Seidman, D. (1998). Postpartum psychiatric illness: The Role of the pediatrician. *Pediatrics in Review*, 19, 128-131.

14 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S. 2, 34-40.

15 Jennings, K., Ross, S., Popper, S., & Elmore, M. (1999). Thoughts of harming infants in depressed and nondepressed mothers. *Journal of Affective Disorders,* 54, 21-28.

16 Seidman, D. (1998). Postpartum psychiatric illness: The role of the pediatrician. *Pediatrics in Review*, 19, 128-131

17 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S 2, 34-40.

18 Susman, J.L. (1996). Postpartum depressive disorders. *Journal of Family Practice,* 43, S17-S24.

19 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S. 2, 34-40.

20 Stocky, A., & Lynch, J. (2000). Acute psychiatric disturbance in the pregnancy and the puerperium. *Baillière's Clinical Obstetrics and Gynaecology*, 14, 73-87.

21 Epperson, C.N. (1999). Postpartum major depression: Detection and treatment. *American Family Physician,* 59, 2247-2254.

22 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S 2, 34-40.

23 Gold, L.H. (2001). Clinical and forensic aspects of postpartum disorders. *Journal of the American Academy of Psychiatry and the Law*, 29, 344-7.

24 Johnstone, S.J., Boyce, P.M., Hickey, A.R., Morris-Yates, A.D., & Harris, M.G. (2001). Obstetric risk factors for postnatal depression in urban and rural community samples. *Australian and New Zealand Journal of Psychiatry*, 35, 69-74.

25 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S 2, 34-40.

26 Stocky, A., & Lynch, J. (2000). Acute psychiatric disturbance in the pregnancy and the puerperium. *Baillière's Clinical Obstetrics and Gynaecology*, 14, 73-87.

27 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S. 2, 34-40.

28 Lumley, J., & Austin, M-P. (2001). What interventions may reduce postpartum depression? *Current Opinion in Obstetrics and Gynecology*, 13, 605-611.

29 Epperson, C.N. (1999). Postpartum major depression: Detection and treatment. *American Family Physician.* 59, 2247-2254.

30 Susman, J.L. (1996). Postpartum depressive disorders. *Journal of Family Practice,* 43, 6, S17-S24.

31 Epperson, C.N. (1999). Postpartum major depression: Detection and treatment. *American Family Physician.* 59, 2247-2254.

32 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S 2, 34-40.

33 Epperson, C.N. (1999). Postpartum major depression: Detection and treatment. *American Family Physician.* 59, 2247-2254.

34 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S 2, 34-40.

35 Steiner, M. (2002). Postnatal depression: A few simple questions. *Family Practice*, 19, 469-470.

36 Fossey, L., Papiernik, E., & Bydlowski, M. (1997). Postpartum blues: a clinical syndrome and predictor of postnatal depression. *Journal of Psychosomatic Obstetrics and Gynaecology*, 18, 17-21.

37 Epperson, C.N. (1999). Postpartum major depression: Detection and treatment. *American Family Physician.* 59, 2247-2254.

38 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: Diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S 2, 34-40.

39 Susman, J.L. (1996). Postpartum depressive disorders. *Journal of Family Practice,* 43, 6, S17-S24.

40 Epperson, C.N. (1999). Postpartum major depression: Detection and treatment. *American Family Physician.* 59, 2247-2254.

41 Susman, J.L. (1996). Postpartum depressive disorders. *Journal of Family Practice,* 43, 6, S17-S24.

42 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S.2, 34-40.

43 Bergant, A. Heim, K., Ulmer, H., & Illmensee, K. (1999). Early postnatal depressive mood: Associations with obstetric and psychosocial factors. *Journal of Psychosomatic Research,* 46, 391-394.

44 Susman, J.L. (1996). Postpartum depressive disorders. *Journal of Family Practice,* 43, 6, S17-S24.

45 Tatano Beck, C. (2001). Predictors of postpartum depression: An update. *Nursing Research,* 50, 275-285.

46 Bergant, A. Heim, K., Ulmer, H., & Illmensee, K. (1999). Early postnatal depressive mood: Associations with obstetric and psychosocial factors. *Journal of Psychosomatic Research,* 46, 391-394.

47 Josefsson, A., Angelsiöö. L., Berg, G., & Ekström, C. (2002). Obstetric, somatic, and demographic risk factors for postpartum depressive symptoms. *Obstetrics and Gynecology*, 99, 223-228.

48 Mandl, K., Tronick, E., & Brennan, T. (1999). Infant health care use and maternal depression. *Archives of Pediatric and Adolescent Medicine,* 153, 808-813.

48a Tatano Beck, C. (2001). Predictors of postpartum depression: An update. *Nursing Research,* 50, 275-285.

49 Josefsson, A., Angelsiöö. L., Berg, G., & Ekström, C. (2002). Obstetric, somatic, and demographic risk factors for postpartum depressive symptoms. *Obstetrics and Gynecology*, 99, 223-228.

50 National Institute of Mental Health. (2002). Depression. http://www.nimh.nih.gov/publicat/ptdep4. Last accessed on November 25, 2003.

51 National Institute of Mental Health. (2002). Research at the National Institute of Mental Health. Fact Sheet: Depression. Last accessed on November 25, 2003. http://www.nimh.nih.gov/publicat/depresfact.cfm

52 Mazure, C., Keita, G., & Blehar, M. (2002). Summit on women and depression: Proceedings and recommendations. Washington, DC: American Psychological Association. Online at: http://www.apa.org/pi/wpo/women&depression.pdf

53 National Institute of Mental Health. (2002). Research at the National Institute of Mental Health. Fact Sheet: Depression. Last accessed on November 25, 2003. http://www.nimh.nih.gov/publicat/depresfact.cfm

54 National Institute of Mental Health. (2002). Depression. http://www.nimh.nih.gov/publicat/ptdep4. Last accessed on November 25, 2003.

55 Epperson, C.N. (1999). Postpartum Major Depression: Detection and Treatment. *American Family Physician.* 59, 2247-2254.

56 National Institute of Mental Health. (2002). Depression. http://www.nimh.nih.gov/publicat/ptdep4. Last accessed on November 25, 2003.

57 Mazure, C., Keita, G., & Blehar, M. (2002). Summit on women and depression: Proceedings and recommendations. Washington, DC: American Psychological Association. Online at: http://www.apa.org/pi/wpo/women&depression.pdf

58 Sharma, V. (2003). Role of sleep loss in the causation of puerperal psychosis. *Medical Hypotheses*, 61,477-481.

59 Epperson, C.N. (1999). Postpartum Major Depression: Detection and Treatment. *American Family Physician.* 59, 2247-2254.

60 Women's Mental Health Center. Women and Depressive Episodes: Female Biology and Depression. http://www2.womens-health.com/health_center/mental/depress_backgrd.html

61 Hollon, S.D., Thase, M.E., & Markowitz, J.C. (2002). The treatment and prevention of depression. *Psychological Science in the Public Interest*, 3,39-77.

62 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S.2, 34-40.

63 Joffe, H., & Cohen, L.S. (1998). Estrogen, serotonin, and mood disturbance: Where is the therapeutic bridge? *Biological Psychiatry*, 44, 798-811.

64 National Institute of Mental Health. (2002). Research at the National Institute of Mental Health. Fact Sheet: Depression. Last accessed on November 25, 2003. http://www.nimh.nih.gov/publicat/depresfact.cfm

65 Hollon, S.D., Thase, M.E., & Markowitz, J.C. (2002). The treatment and prevention of depression. *Psychological Science in the Public Interest*, 3,39-77.

66 A Brief History of Depression. (*June 1999*). *Johns Hopkins Medical Letter: Health After 50*. Last accessed November 26, 2003. http://www.hopkinsafter50.com/html/silos/depression/haARTICLE_history.php

67 Kennedy, S.H., Lam, R.W., Cohen, N. L., Ravindran, A.V. & the CANMAT Depression Work Group. (2001). Clinical Guidelines for the Treatment of Depressive Disorders: IV. Medications and other biological treatments. *Canadian Journal of Psychiatry*, 46, S1, 39S-57S.

68 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S.2, 34-40.

69 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S.2, 34-40.

70 Epperson, C.N. (1999). Postpartum Major Depression: Detection and Treatment. *American Family Physician.* 59, 2247-2254.

71 Newport, D.J., Hostetter, A., Arnold, A., &Stowe, Z.N. (2002). The treatment of postpartum depression: Minimizing infant exposures. *Journal of Clinical Psychiatry*, 64, S7, 31-44.

72 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S.2, 34-40.

73 Epperson, N. Czarkowski, K.A., Ward-O'Brien, D., & et al. (2001). Maternal sertraline treatment and serotonin transport in breastfeeding mother-infant pairs. *American Journal of Psychiatry*, 158, 1631-1637.

74 Susman, J.L. (1996). Postpartum depressive disorders. *Journal of Family Practice,* 43, 6, S17-S24.

75 A Brief History of Depression. (*June 1999*). *Johns Hopkins Medical Letter: Health After 50*. Last accessed November 26, 2003. http://www.hopkinsafter50.com/html/silos/depression/haARTICLE_history.php

76 Hollon, S.D., Thase, M.E., & Markowitz, J.C. (2002). The treatment and prevention of depression. *Psychological Science in the Public Interest*, 3,39-77.

77-79 Susman, J.L. (1996). Postpartum depressive disorders. *Journal of Family Practice,* 43, 6, S17-S24.

80 Biovail Corporation. (2003). The Food and Drug Administration Approves Wellbutrin XL. http://www.biovail.com/english/Investor%20Relations/Latest%20News/default.asp?s=1&state=showrelease&releaseid=444290

81 Kennedy, S.H., Lam, R.W., Cohen, N. L., Ravindran, A.V. & the CANMAT Depression Work Group. (2001). Clinical Guidelines for the Treatment of Depressive Disorders: IV. Medications and other biological treatments. *Canadian Journal of Psychiatry*, 46, S1, 39S-57S.

82 Susman, J.L. (1996). Postpartum depressive disorders. *Journal of Family Practice,* 43, 6, S17-S24.

83 Cohen, L.S., Viguera. A.C. & Bouffard S.M. et al. (2001). Venlafaxine in the treatment of postpartum depression. *Journal of Clinical Psychiatry*, 62, 592-596.

84 Sharma, V. (2003) Pharmacotherapy of postpartum depression. *Expert Opinion Pharmacotherapy*, 3, 1421-1431.

85 (Hendrick, V., Altshuler, L., & Suri, R.) (1998). Hormonal changes in the postpartum and implications for postpartum depression. *Psychosomatics,* 39, 93-101).

86 Sharma, V. (2003). Pharmacotherapy of postpartum depression. *Expert Opinion Pharamacotherapy*, 3, 1421-1431.

87 Epperson, C.N., Wisner, K.L., & Yamamoto, B. (1999). Gonadal Steroids in the Treatment of Mood Disorders. *Psychosomatic Medicine*, 61, (5), 1-38.

88 Epperson, C.N., Wisner, K.L., & Yamamoto, B. (1999). Gonadal Steroids in the Treatment of Mood Disorders. *Psychosomatic Medicine*, 61, (5), 1-38.

89 Lawrie, T.A., Herxheimer A., & Dalton K. Oestrogens and progestogens for preventing and treating postnatal depression (Cochrane Review). In: *The Cochrane Library,* Issue 4, 2003. Chichester, UK: John Wiley & Sons, Ltd.

90 The Depression Center at the University of Michigan. Last accessed November 29, 2003. http://www.med.umich.edu/depression/cbt.htm

91 Susman, J.L. (1996). Postpartum depressive disorders. *Journal of Family Practice,* 43, 6, S17-S24.

92 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S.2, 34-40.

93 Hollon, S.D., Thase, M.E., & Markowitz, J.C. (2002). The treatment and prevention of depression. *Psychological Science in the Public Interest*, 3, 39-77.

94 Hollon, S.D., Thase, M.E., & Markowitz, J.C. (2002). The treatment and prevention of depression. *Psychological Science in the Public Interest*, 3,39-77.

95 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S.2, 34-40.

96 O'Hara, M.W., Stuart, S., Gorman, L.L., & Wenzel, A. (2000). Efficacy of interpersonal psychotherapy for postpartum depression. *Archives of General Psychiatry*, 57, 1039-1045.

97 Spinelli, M.G., & Endicott, J. (2003).Controlled clinical trial of interpersonal psychotherapy versus parenting educatioin program for depressed pregnant women. *American Journal of Psychiatry*, 160, 555-562.

98 Milner, G. (1999). The present status of electroconvulsive therapy. *Medical Journal of Australia*, 171, 687. Letter. http://www.mja.com.au/public/issues/171_11_061299/milner/milner.html#refbody2 http://members.fortunecity.com/anemaw/ray.htm

99 Milner, G. (1999). The present status of electroconvulsive therapy. *Medical Journal of Australia*, 171, 687. Letter. http://www.mja.com.au/public/issues/171_11_061299/milner/milner.html#refbody2

100 Endler, N.S. (1988). The origins of electroconvulsive therapy (ECT). *Convulsive Therapy*, 4, 5-23. Toronto, Canada.

101 National Institute of Mental Health. (2003). Depression Research at the National Institute of Mental Health: Depression Fact Sheet. Last accessed on November 29, 2003. http://www.nimh.nih.gov/publicat/depresfact.cfm

102 Regan, J., & Alderson, A. (200x). Electroconvulsive therapy: Brief review and present utilization. Department of Mental Health and Developmental Disabilities. Tennessee, USA. http://www.state.tn.us/mental/omd/ect.pdf

103 Fink, M. (2001). *Journal of Affective Disorders*. Mar;63(1-3):1-15.

104 Enns, M.W., & Reiss, J.P. (2003). Electroconvulsive Therapy. Canadian Psychiatric Association. http://www.cpa-apc.org/Publications/Position_Papers/Therapy.asp

105 Rabheru, K. (2001). The use of electroconvulsive therapy in special patient populations. *Canadian Journal of Psychiatry*, 46, 710-719.

106 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S.2, 34-40.

107 Wisconsin Perinatal Care Association. Screening for Perinatal and postpartum depression. Position Statement. http://www.perinatalweb.org/association/pub_pStateMoreBlues.html

108 Morris-Rush, J.K., & Bernstein, P.S. (2002). Postpartum depression. *Medscape Ob/Gn\yn & Women's Health*, 7(1). On-line at http://www.medscape.com/viewarticle/433013

109 Morris-Rush, J.K., & Bernstein, P.S. (2002). Postpartum depression. *Medscape Ob/Gn\yn & Women's Health*, 7(1). On-line at http://www.medscape.com/viewarticle/433013

110 Beck, C.T., & Gable, R.K. (2001). Comparative analysis of the performance of the postpartum depression screening scale with two other depression instruments. *Nursing Research*, 50, 242-250.

111 Indiana Perinatal Network. (2002). Postpartum depression consensus statement. Executive Summary. Last accessed November 30, 2003. http://www.indianaperinatal.org/Images/ppdexsum.pdf

112 Wisconsin Perinatal Care Association. Screening for Perinatal and postpartum depression. Position Statement. http://www.perinatalweb.org/association/pub_pStateMoreBlues.html

113 Wisconsin Perinatal Care Association. Screening for Perinatal and postpartum depression. Position Statement. http://www.perinatalweb.org/association/pub_pStateMoreBlues.html

114 Nonacs, R., & Cohen. (1998). Postpartum mood disorders: diagnosis and treatment guidelines. *Journal of Clinical Psychiatry*, 59, S.2, 34-40.

Glossary

Antenatal: The period of time between conception and birth.

Amitriptyline (Elavil): An antidepressant medication that elevates mood by raising the level of neurotransmitters in the brain.

Bupropion (Wellbutrin; Zyban): An antidepressant medication that that works primarily by inhibiting the reuptake of the neurotransmitter dopamine.

Clozapine (Clozaril): An anti-psychotic medication that works by blocking receptors for several neurotransmitters in the brain.

Convenience Sample: Not randomly selected, entered into a research study using no particular order.

Dysphoric: Low level depression with duration of at least two years.

DSM-IV: The short form used to refer to the fourth edition of the Diagnostic and Statistical Manual for Mental Disorders. Published by the American Psychiatric Association it sets out diagnostic criteria, descriptions, and other information used to classify and diagnose mental disorders. The DSM-IV was published in 1994, replacing DSM-IIIR. In turn, the DSM-IV was replaced in the year 2000 by the DSM-IV-TR. It is expected that DSM-V will replace DSM-IV-TR sometime in the future.

Major Depressive Episode: When an individual experiences a discrete episode of persistent and pervasive emotional depression, this term may be applied. Recurrence may mean that the individual may be diagnosed with one of the Mood Disorders, either Major Depressive Disorder or a Bipolar Disorder.

Estradiol : An estrogen produced by the ovary.

Estrogen: The primary female hormone secreted by the ovaries.

Gonadotropins: The hormones produced by the pituitary gland that control reproductive function follicle stimulating hormone (FSH) and luteinizing hormone (LH)

Gonads: Organs that produce the sex cells and sex hormones. In men: testicles; in women: the ovaries.

Hormone: A substance produced by an endocrine gland. The hormone is secreted into the bloodstream where it travels to a specific organ where it exerts its hormonal effect.

Hypothalamus: A gland in the brain that releases GnRH (see GnRH), which in turn stimulates the production of LH and FSH by the pituitary gland.

Hypothyroidism: The under activity of the thyroid gland. Low levels of thyroid hormone in the blood can lead to hyperprolactinemia.

Imipramine: A medication that belongs to the tricyclic class of antidepressants or TCAs. Imipramine was first synthesized in the late 1940s, and then approved by the FDA in 1959 to treat depression.

Lithium (Eskalith, Lithobid): Lithium is used to treat bipolar disorder(s). It interferes with the synthesis and reuptake of neurotransmitters. Lithium also affects the concentrations of tryptophan and serotonin (a neurotransmitter) in the brain. Lithium has been in use since the 1950s. However, the Food and Drug Administration approved the current preparation -lithium carbonate – to treat bipolar disorder(s) in 1970.

Luteinizing Hormone (LH): A hormone secreted by the pituitary gland. Along with FSH, it is one of the two most important hormones that regulate ovarian function. The role of LH is to trigger ovulation and help prepare the endometrial lining for implantation.

Multiparous: Having had two or more pregnancies resulting in viable fetuses.

Ovary: Ovaries produce eggs and female hormones.

Ovulation: release of an egg from the ovary

Parturition: Childbirth, the process of delivering a baby and placenta to the outside world.

Pituitary Gland: A small gland at the base of the brain that receives instructions from the hypothalamus. The pituitary gland secretes many important hormones. These include: FSH, LH, TSH, and prolactin. FSH and LH control the ovaries, TSH controls the thyroid gland, and prolactin controls milk production.

Population-based Study: Research study that obtains its subject participants from the general population.

Primary Prevention: Efforts to prevent disease in individuals or populations through promotion of health and healthy behaviours.

Progesterone: A hormone whose role is to prepare the uterine lining to support the implantation of a fertilized egg. It can also be administered as a medication in injectable, oral, and intravaginal formulations.

Prolactin: A hormone secreted by the pituitary gland to control milk production. However, it can interfere with normal ovulation if present in high amounts.

Prostaglandins: a group of hormone-like substances that exert various effects on the reproductive organs; so named because they were first discovered in the prostate gland

Sertraline (Zoloft): One of the selective serotonin reuptake inhibitors (SSRIs). Other drugs of this class include: Prozac (fluoxetine), Paxil (paroxetine), Celexa (citalopram), and Luvox (fluvoxamine).

Serotonin: A neurotransmitter (a chemical messenger) used by nerves in order to communicate with one another. A nerve releases its serotonin into the space surrounding it. The serotonin either travels across the space where it attaches to surface receptors on nearby nerves or it returns to its source nerve – this called re-uptake.

Selective serotonin reuptake inhibitors (SSRIs): This is a class of antidepressant medications. SSRIs block the reuptake of serotonin and therefore allow serotonin to remain in the brain. The Food and Drug Administration approved Sertraline in December 1991.

Sign and Symptoms: subtle distinction From: http://www.behavenet.com
In behavioral health care as in general medicine, when an individual complains of a subjectively experienced disturbance or unpleasant perception such as pain or anxiety, we call this a symptom. We distinguish this from a sign which is an observable phenomenon, such as slurred speech.

Sleep: Sleep is important to avoid mental illness. What is sleep? Normal sleep progressively passes through five stages defined by their associated electrical patterns produced using an electroencephalogram (EEG).
During the first four stages of sleep, the muscles of the eyes are relaxing. These stages are collectively referred to as nonrapid eye movement (nonREM) sleep. The last stage of sleep is associated with increased contraction of the eye muscles. As a result, the fifth stage of sleep (when eye movement is active) is called rapid eye movement sleep or REM sleep.

Mood: Mood may refer to the feeling tone of a subject observed during a psychiatric examination. Or it may refer to the emotional state experienced by an individual for a limited period of time. Examples of mood include depression, mania, hypomania, euphoria, dysphoria, elation, anger, irritability, happiness, sadness, and many others. Disturbances of mood are the primary manifestation of Mood Disorders. Mood from: http://www.behavenet.com

Mood Disorders (Affective Disorders): A defining feature of a mood disorder is an unhelpful and persistent (can also be episodic) exaggeration of a mood state.

Major Depressive Disorder: A mood disorder where mood depression, i.e., depressed mood, is the essential feature.

Testosterone: the primary male sex hormone.

Thyroid gland: the endocrine gland in the front of the neck that produces thyroid hormones that regulate the body's metabolism. For example, Thyroxin (T4) and T3 are important thyroid hormones.

Thyroid Stimulating Hormone (TSH): A hormone secreted by the pituitary gland that controls the thyroid gland. Elevated levels imply an abnormally low thyroid function.