TOWARDS A NORMATIVE MODEL OF POSTNATAL MOOD; FEATURES OF DEPRESSION AND ANXIETY AMONG WOMEN AFTER CHILDBIRTH

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TOWARDS A NORMATIVE MODEL OF POSTNATAL MOOD; FEATURES OF DEPRESSION AND ANXIETY AMONG WOMEN AFTER CHILDBIRTH

Abstract

Background: Presently, there is lack of a *normative* model of postpartum mood. In contrast, *postnatal depression* represents the principal model of postnatal mental health founded upon the presence of transient *symptoms* of depression and anxiety in the early postnatal period. However, anxiety is shown to be central to *normative* maternal attachment whilst evolutionary hypotheses propose that the primary purpose of depression and anxiety is to promote survival. In this study postpartum symptoms of depression and anxiety were explored in efforts to demonstrate how these might be organised to suggest a characteristic pattern of postpartum mood.

Methods: Postnatal subjects (n = 767), recruited from the caseloads of generic health visitors (n =124), completed the Edinburgh Postnatal Depression Scale (EPDS), Beck Depression Inventory (BDI) and Beck Anxiety Inventory (BAI) to investigate rates of postpartum *symptoms* of depression and anxiety on two occasions. Principal component analysis was used to assess scale performance; operational score categories were generated to assess point and period rates of depression and anxiety symptoms, their co-existence and change in rates overtime.

Results: Factorial validity demonstrated the single construct status of the BDI (depression) and BAI (anxiety) with the EPDS confirmed as a measure of depression *and* anxiety. Specific EPDS items for depression *and* anxiety comprised a sub-scale

that contributed almost 50% of the total EPDS score. The majority of subjects rated co-existing symptoms of depression and anxiety at a level proposed to be *without impairment or distress*, with a statistically significant reduction in rates over time. Subjects who met the criteria for *risk* of disorder on two occasions were two to three times less than the reported 10% -15% prevalence of postnatal depression.

Conclusion: With consideration for the limits of the study, findings suggest that non-pathological postpartum mood is characterised by (1) specific symptoms of anxiety and depression and, more broadly (2) co-existing symptoms that exist most frequently below the threshold for disorder, the rate and severity of which spontaneously diminish overtime.

Acknowledgments

In 1999 I began work as a new health visitor which brought me into daily, close contact with postpartum women and their babies. I entered a culture where *postnatal depression* dominated practice and shaped how health care professionals understood the mental domain of postpartum women. Around that time I also became a volunteer facilitator with *Walk Free* a therapeutic, self help group for those with anxiety disorders. It is through contact with these groups that the idea for this thesis evolved. My thanks therefore are first extended to these individuals for teaching me so much about the range of human experience.

In the early days of working with essentially healthy postpartum women and a separate group of individuals suffering a mental health disorder (*Walk Free*), I was struck by the similarities in the accounts of their emotional experience. I became conscious that no one had considered the boundary between *normative* and pathological mood states in new mothers. As a result I came to consider what might be *normal* for postpartum women.

In 2001 I approached Professor Steve Rollnick with my ideas as I knew him as the founder of *Walk Free*. Without his vision and committed support it is unlikely that this project would ever have got off the ground. My gratitude to Professor Rollnick is therefore owed for more than his supervisory expertise. His advice and guidance particularly around the writing and structure of the thesis served to contain periods of uncertainty and helped me to navigate and express complex ideas.

With the help of Professor Rollnick I was awarded a grant for novice researchers from the Research and Development Committee Cardiff and Vale NHS Trust. This allowed me to arrange for one day a week study leave and to fund the research materials. I am grateful to this committee and to my employer Cardiff and Vale University Health Board Trust for providing subsequent study leave whilst I continued my full time work as a health visitor.

I have been very privileged that my second supervisor is Professor Kerry Hood whose statistical expertise has taught me so much. Her enthusiasm has left me with a desire to learn more.

I am grateful to the health visitors and mothers who participated in the study and to my immediate work colleagues who have been generous in their ongoing support. It has been my good fortune to share an office with Dr. Myfanwy Davies. I have benefitted from her academic wisdom and friendship.

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CHAPTER 1

INTRODUCTION

1.1 Background

There is clear evidence that maternal psychiatric disorder (for example, Hemphill 1952; Kumar and Robson1984; Stein et al 2009), personality disorder (Crandell et al 2003; Hobson et al 2005; Newman and Stevenson 2005) and insecure patterns of attachment (Fonagy 1991; Bifulco 2004) impose serious, adverse consequences for the emotional and cognitive development of offspring. Moreover, the effects of maternal psychiatric disturbance have been shown to persist beyond the period of exposure, after remission of the parental illness (Stein et al 2009). Appropriately, the status of maternal mental health receives considerable attention and has been studied mainly in relation to the occurrence of depression in postnatal women (Feldman 2007; Stein 2009). Depression in postnatal mothers has been shown to compromise the social, emotional and cognitive regulatory skills of infants (Goodman and Gotlib 1999). Maternal depression is associated with impaired s*ynchrony* in the mother-infant relationship (Feldman 2007), which puts at risk the physiological systems that support social engagement in offspring, making them less adaptive (Moshe and Feldman 2006).

Clinical depression in postnatal mothers is a major cause for concern because similar to depression in general it is prevalent and therefore contributes the greatest mother-related risk to the wellbeing of infants. However, it is possible that the prevalence of maternal depression has become synonymous with perceptions of an innate and heightened risk of maternal psychiatric disorder in relation to childbearing.

Consequently, *postnatal depression* represents the prevailing model of postnatal mental health. It is described as one of the '*more common complications'* of childbearing (Gavin 2005) and has been influential in shaping lay and scientific perceptions of maternal mental health. From the lay perspective the problem of postnatal depression is seen to be one where mothers' and families are '*robbed'* of a singularly joyous experience (Beck 1996), the birth of a new baby. From a scientific perspective postnatal depression can be said to have retained its *atypical* status (Pitt 1968), generally viewed as distinct from depression occurring at any other time of life.

In contrast to the emphasis upon maternal psychopathology and postnatal depression, understanding of the *normative,* mental domain of postpartum experience is limited. This *normative,* mental domain is understood here to represent a characteristic, non-pathological mood state in postnatal women that facilitates the *norm* of optimum, secure infant attachment by promoting maternal bonding behaviours.

1.2 Synopsis

The aims of this chapter are to define the subject under investigation, set the context of the thesis and provide an overview of the study themes, aim and objectives. These are achieved through;

(a) a definition of *postpartum mood* within the context of the thesis

(b) a rationale for the need to explore a non-pathological model of the postpartum mood,

(c) an overview of the themes which underpin the literature review and, finally,

(d) an outline of the main study aim and objectives.

1.3 Postpartum mood; the presence of symptoms of depression and anxiety

The subject under investigation is *postpartum mood,* defined in this study as the presence of symptoms of depression and anxiety as non-pathological features in women after childbirth.

Anecdotally, in health visiting practice the recurring narratives and experience of healthy postnatal women have been interpreted to include frequent expressions of depression and anxiety, which do not reflect the scientific evidence for *postnatal depression.* Similarly, evidence from the extensive use of the Edinburgh Postnatal Depression Scale (EPDS) (Cox et al 1987) in practice suggests that specific items for depression and anxiety are more commonly selected by postnatal women, compared with other EPDS items. Consequently, it is considered here that postpartum symptoms of depression and anxiety can be expressed in the absence of pathology, existing below as well as above the threshold for risk of disorder and, potentially, represent characteristic and *normative* features of maternal mood in the early postnatal period.

1.4 In pursuit of a *normative* model of postpartum mood

Postnatal depression is diagnosed according to medical thresholds for disorder in the general population despite failing to meet recognised diagnostic criteria as a discrete disorder (DSM-1V, APA 1994; ICD-10, WHO 1994). Conversely, evaluation of the wider evidence provides alternative view points to the occurrence of postpartum symptoms of depression and anxiety and their potential, non-pathological role in *healthy* maternal-infant attachment.

Why is it necessary to investigate the potential for a *normative* model?

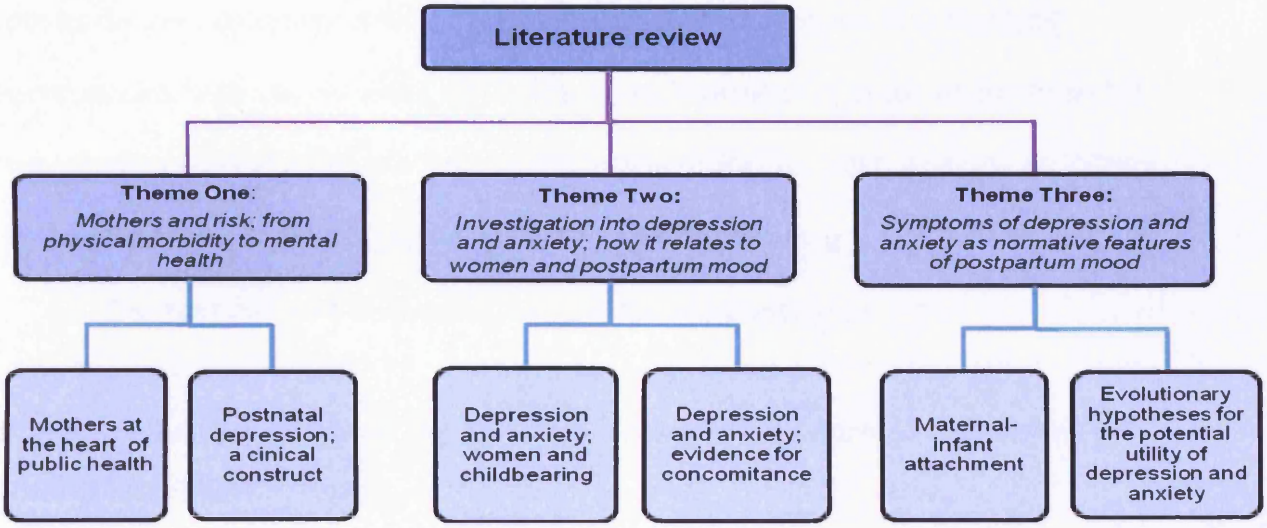
Firstly, it is essential that health care professionals interact with healthy postnatal women in a manner that promotes the well being of the maternal/ infant dyad. This capacity must be founded upon a working knowledge of *normative* postpartum *mood.* Secondly, it is a matter of governance, where service provision is evidence based and appropriately responds to the needs of postpartum women and infants (NICE 2007). Thirdly, defining what is *normal* can help to identify the boundary between health and pathology (Nesse 1999) and provides the basis for developing effective psychotherapeutic interventions for perinatal psychiatric disorders. Fourthly, and finally, *primum non nocere;* 'first, do no harm'. Ethical questions are raised regarding the principle of non-maleficence (Amarakone and Panesar 2009, p 8-9), where the continued emphasis upon the heightened risk of maternal psychopathology in relation to childbearing in the light of opposing evidence cannot be justified and might in itself prove harmful to postnatal women and infants.

1.5 Study themes

The following review is comprised of three themes which emerge from relatively disparate areas of scientific literature to examine the relationship between women, depression, anxiety and postpartum mood. These offer both opposing and complimentary viewpoints to the reporting of *postnatal depression*, which combined are proposed to offer a cohesive argument for this investigation into *postpartum mood.*

Diagram 1.1 provides an outline of the literature review themes.

Diagram 1.1 outline of literature review



Theme One: *Mothers and risk; from physical morbidity to mental health*

Theme one (discussed in Chapter 2) considers the historical milieu in which the concept of *postnatal depression* emerged and how its progression might have been influence by the introduction of the Edinburgh Postnatal Depression Scale (EPDS) (Cox et al1987). Theme one provides the basis for critically evaluating arguments for postnatal depression as a discrete diagnostic construct and supports wider investigation into *non-pathological* postpartum mood.

Theme one is divided into two sections as follows;

- **Mothers at the heart of public health**
- **Postnatal depression; a clinical construct**

Theme Two: ***Investigation into depression and anxiety and how it relates to women and postpartum mood***

Theme two (discussed in Chapter 3) examines findings for depression and anxiety both as discrete disorders and their concomitance. The intention is to highlight discrepancies between the findings for these two investigative areas as the basis for re-evaluating postnatal depression. Theme two is divided into two sections as follows;

- **Depression and anxiety; women and childbearing**
- **Depression and anxiety; evidence for concomitance**

Theme Three: ***Symptoms of depression and anxiety as normative features of postpartum mood***

The third theme of this thesis (discussed in Chapter 4) considers evidence and hypotheses for the non-pathological role of depression and anxiety in relation to postpartum mood. Theme three also has two sections;

- **Maternal-infant attachment**
- **Evolutionary hypotheses for the utility of depression and anxiety**

1.6 Outline of the main study aim and objectives

The main aim of this thesis is to investigate how features of depression and anxiety might be organised to suggest a characteristic pattern in postpartum mood, in a population sample of postnatal women using the EPDS, Beck Depression Inventory (Beck 1961) and Beck Anxiety Inventory (Beck 1988).

The study objectives are,

(1) To measure postpartum symptoms of depression and anxiety at two periods of time in a population sample of postnatal women using the EPDS, BDI and BAI.

(2) To examine the relationship between rates of anxiety and guilt vis-à-vis other EPDS symptoms.

In Chapter 2 to follow, *Theme One* explores the risk of harm attributed to mothers regarding infant morbidity and the emergence of postnatal depression as a clinical construct. The review traces how perceptions of risk progressed from public health concern over a failure of mothers in general to provide physical care for infants to the innate vulnerability of maternal mental health and the deleterious impact upon infant development.

CHAPTER 2

THEME ONE: *MOTHERS AND RISK; FROM PHYSICAL MORBIDITY TO MENTAL HEALTH*

2.1 Synopsis

Chapter 2 presents the first theme of the literature review. The aim is to track the progression of postnatal depression as a clinical construct. Theme one is considered in two sections:

- **Mothers at the heart of public health**

This section explores the relationship between women, children and families and early public health endeavours. The aim is to introduce some of the historical factors that are proposed to have influenced perceptions of mothers as a source of risk for infants and the population in general.

- **Postnatal Depression; a clinical construct**

This section considers the medico-social milieu in which early scientific interest into atypical, postnatal depression occurred and the influence of the EPDS, which was developed for the widespread screening of mothers. The intention is to illustrate how both theory and practice may have led to the reporting of postnatal depression as a discrete disorder.

Mothers at the heart of public health

2.2 Historical background

The industrial revolution which spanned the late 18th and early 19th century fuelled the urbanisation of large sections of the population in the pursuit of employment. The extreme poverty and squalor of the poor working class contributed to the unabated spread of disease and escalating rates of mortality and morbidity. With scientific and political recognition of the link between the environment and health, housing and environmental hygiene assumed a primary focus for the first Public Health Acts (1848, 1872 and 1875) and rapidly progressed to embrace the social welfare of the working class.

Early public health activities were local, philanthropic and bottom-up although a shared focus emerged in the health and welfare of women and children. Without any formal entitlement emphasis was placed upon the ability of educated middle class women to gain access to the homes of the impoverished working class through friendship and support. The purpose of the lay *female home visitor* was to impart advice on matters such as domestic arrangements, hygiene and infant feeding and childcare. By the early 1900's the discourse of medicine influenced the development of public policy aimed at addressing the welfare of the nation; health became a matter for the state rather than charity with responsibility for health and illness borne by the individual (Ross 1993, pg.'s 198-199).

Political and scientific attention increasingly focused on motherhood and childcare as areas central to public health; lay efforts gave way to state organised activities which advanced under wide ranging legislation that reflected a national concern for the high

rate of infant mortality. *The Intradepartmental Committee on Physical Deterioration* report (1904) addressed the poor health of the nation and the persistently high rates of infant mortality. Despite the fact that the impact of the environment on health was acknowledged, the causal role of mothers was emphasised. As Newman (1906) records, the occupation of women in mills and factories was understood to generate circumstances in which infants failed. Mothers frequently worked late into their confinement as a matter of economic necessity, returning to employment early after giving birth. These circumstances not only compromised maternal health and the chance of delivering healthy offspring but it committed very young infants to the often neglectful care of others including the hazards of artificial feeding (Newman 1906, pg's 93-97, 133).

Even so, mothers were bestowed a more ominous role regarding infant mortality with the impact of the environment relegated to a secondary position. The problem as chronicled by Newman (1906) was seen to be '*mainly a question of motherhood'* (Newman 1906, pg. 151), due to the *'ignorance and carelessness of mothers in respect of infant management'* (Newman 1906, pg. 262) with an emphasis upon maternal '*negligence and ignorance'* (Newman 1906, pg. 219). *'Ignorant and feckless mothers'* were charged not only for the poor health of their immediate families but ultimately the poor health of the nation (Ross 1993, pg. 199).

The Intradepartmental Committee on Physical Deterioration Report (1904) caused a decisive shift in public health regulations with the family unit, women and children formally targeted. Legislation such as the Notifications of Birth Act (1907) mandated local authorities to provide a home visit for every birth although this mandate did not extend to the family. Health visitors held professional responsibility to *'...carry out the*

system of home visitation, including that under the Notification of Births Act...' and bring *'...the homes of the poorer section of the working classes into direct contact with every remedial agency' and '...try to induce mothers to take advantage of the help provided and follow-up the advice given at the centres'* (Edward James Smith 1918).

Innovations such as the Bradford Maternity Child Welfare Scheme, established in 1912, arguably served to shape public health provision for infants on a national scale. Under the scheme child health clinics were established for the purpose of weighing infants, screening for illness and developmental delay and to instruct mothers in childcare practices such as the making up of formula milk. Work undertaken at the clinic was followed up by health visitors in the home. In addition, the National Maternity and Child Welfare Scheme sought to improve maternal and child care provision to the poor. It called for the routine care of women in the antenatal, intra partum and postnatal periods as well as the provision of services to infants and the pre-school child, which included systematic home visiting. Around a century later these child and family focused initiatives remain bedrock features of contemporary health visiting practice in Britain.

2.3 Section summary: Mothers at the heart of public health

The public health movement at the turn of the twentieth century targeted *failing mothers and* imposed systematic programs of care for mothers and offspring. State legislation responded to the unacceptably high rates of infant morbidity and mortality, contributing to the medicalisation of childrearing and the postnatal experience.

Postnatal depression as a clinical construct

2.4 Public health and the medicalisation of infant care

It is important to appreciate that the industrial revolution imposed radical change on the socio-economic structure in Britain with adverse consequences for women and children (see Newman 1906). Despite awareness of the relationship between the environment and health, public policy perpetuated notions of the *'careless and ignorant'* working class mother. As a consequence mothers and infants remained the subjects of uncompromising political and medical scrutiny. The remedy came in the medicalisation of childcare (see Newman 1906) which assured that, providing the mother complied with the instructions of health visitors and doctors then she could have full power over the life and death of her infant. In other words the death of a child was the fault of the mother *...'making the loss of a child a social stigma'* (Ross1993, pg. 202). Ross (1993, pg. 202) proposed that a *'combined legislative and propaganda assault'* during the early 20th century *'helped to create the cultural conditions of the guilty mother'*, who was otherwise perceived as inappropriately *'worried'* (Ross 1993, pg. 202).

2.4.i Early research into postnatal depression

By the mid 20th century improvement in rates of infant morbidity and mortality alongside an upsurge in interest in mental health saw a shift in attention from physical wellbeing to concerns over the deleterious impact of maternal mental health upon infant development. Psychiatric disturbance detected in postnatal women became the focus of intense scientific investigation. The prevalence of postnatal depression within the first 3 months following childbirth was reported to vary between 4% and 15% (Pitt

1968; Kendell et al 1984; Paykell 1980; Cox et al 1982; Kumar and Robson 1984; O'Hara et al 1984) with serious consequences for the health and development of infants and young children (e.g. Hemphill 1952; Kumar and Robson1984).

It is evident from the early literature that despite opinions being mixed regarding the aetiology of postnatal mental illness, scientific thinking and discourse assumed a particular trajectory. Some authors suggested that puerperal mental illness was indistinguishable from other *non-specific mental reactions* occurring as a result of the interaction between external factors and individual predisposition; pregnancy was of psychological importance only (Brew and Seidenberg 1950). Others were more strident in pursuing a physiological or endocrine cause. Hemphill (1952) proposed that there was no evidence to justify that psychogenic factors, such as psychological stress and parity, were of importance in postpartum mental illness. On the contrary, childbearing women were considered resistant to those stressors usually associated with neurotic or psychotic illness. Hemphill (1952) further proposed that postnatal psychiatric disturbance was one of the most serious complications of pregnancy occurring after an '*endocrine event and prolonged'*, *which '...disturbs the mother-infant relationship, disrupts the home and makes a terrible start to family life'* (Hemphill 1952).

Scientific effort to show that depression in postpartum women was a discrete disorder and different to depression in the general population continued. In his seminal paper '"*Atypical" Depression Following Childbirth' (*Pitt 1968), which is described by Kumar and Robson (1984) as a 'classic study', *Pitt (1968)* states '*It is common knowledge that women get depressed after childbirth'* with study outcomes interpreted to suggest that '*a sizeable proportion of the cases of atypical depression in the community must*

arise after childbirth' (Pitt 1968). Notably, Pitt (1968) attributes his research to the suggestion of a health visitor who had commented upon the frequency of depression in newly delivered women with the request for help to understand why this occurred and for guidance in responding.

Prior to Pitt's influential study (Pitt 1968) a number of authors had reported upon the apparent higher rates of postpartum psychiatric disturbance (Osmond 1953; Pugh 1963; Jacobson 1965; Association for Improvement in the Maternity Services 1965) with maternal concerns such as the "vomiting baby" interpreted as a presenting symptom of puerperal depression (Carne 1966). A range of other symptoms were suggested as discrete indicators of puerperal depression including maternal anaemia, tearfulness, labile mood, despondency, anxiety, irritability, fatigue, exhaustion, impaired concentration, sleep disturbance, and a lack of *normal* sexual interest (Osmond 1953; Pugh 1963; Pitt 1968; Jacobson 1965; Association for Improvement in the Maternity Services 1965).

In a survey of postnatal women Pitt (1968) reported the prevalence of postnatal depression within the first 3 months at 10.8 per cent, where symptoms persisted or developed beyond the 2 week *'readjustment period'* or lying-in period (Nuche 2002). Significantly, Pitt (1968) identified that in *atypical* postpartum depression the diurnal fluctuations in mood showed some worsening later in the day, in contrast to the early morning worsening found in classic depression; *atypical depressives* were also noted to exhibit more neurotic symptoms that overshadowed depression, such as anxiety, irritability and phobias. In addition to those symptoms of puerperal depression previously cited, Pitt (1968) identified a number of postpartum features specifically related to the baby. These were determined as guilt and self reproach over not caring

for or loving the baby enough; feelings of inadequacy and an inability to cope, particularly in relation to the baby; babies who would not sleep and kept crying were seen to be *hard to love* leaving mothers feeling guilty whilst others were found to be concerned with spoiling their infant. Undue fatigue and exhaustion was also viewed as symptomatic of *atypical* postpartum depression and meant that new mothers could '*barely cope with their babies let alone address the needs of the family such as shopping and housework' (Pitt 1968).*

Scientific opinions were conflicted regarding what potential, causal variables were at play in postnatal depression. A number of factors were subject to investigation such as the impact of pregnancy and delivery complications (Tod 1964; Jacobson 1965; Pitt 1968; Martin 1977; Playfair and Gowers 1981; Cox et al 1982); the relevance of antenatal anxiety and depression (Tod 1964; Dalton 1971; Mears 1976; Cox et al 1982); parity (Tod 1964; Hayworth et al 1980; Cox et al 1982); miscarriage (Playfair and Gowers 1981) and the role of progesterone, as expounded by Dalton (1971). Many early studies revealed a number of psychosocial variables with the potential to influence postpartum mood but the picture was unclear. The impact of external stressors such as housing and marital difficulties received reasonable consensus (Martin 1977; Paykell et al 1980; Playfair and Gower 1981; Brown and Harris 1978) although was not supported by the work of, for example, Cox et al (1982); findings for an association between postpartum depression and social class also conflicted (e.g. Brown and Harris 1978; Watson et al 1984). The work of Frommer and O'shea (1973) and Brown and Harris (1978) did, however, support that a women's disrupted attachment with her own mother in childhood, including maternal death before the age of 11 years, was a predisposing factor to depression at any time.

Interestingly, Watson et al (1984) reported findings from a prospective study surveying women in pregnancy and the first postnatal year, suggesting there was little to distinguish women with depression in the puerperium from those suffering at other times of life. In contrast to the earlier findings of Pitt (1968), a vulnerability to postpartum relapse, conferred by previous postnatal depression, a psychiatric history and/ or severe blues (Playfair and Gower 1981; Watson et al 1984) was also becoming evident. However, Kumar and Robson (1984), whilst highlighting the impact of the methodological limitations of many of the studies into postnatal depression, ultimately confirmed that '*Childbirth can affect the mental health of women*'.
This conflict regarding the validity of postnatal depression as a clinical disorder persists at the highest level: NICE (2007) comments on the misuse of the term postnatal depression. Widespread '*common false beliefs...*' are noted to endorse postnatal depression as different to depression at other times of life and wrongly associate the cause of depression postpartum with factors such as hormones and breastfeeding (NICE 2007). In contrast, the WHO (2008) describes postnatal depression as '*the most common complication of child bearing*'

2.4.ii Development of the Edinburgh Postnatal Depression Scale

Scientific interest in postpartum maternal mental health continued into the 1980's. Cox et al (1982) reported that the illness of postnatal depression caused much personal and family distress, which prevented women from carrying out household tasks and made coping with the baby difficult. Despite earlier public health emphasis upon the risk mothers posed to the physical health of their offspring, sufferers were identified as being excessively concerned about the health of their babies, doubted

their ability to look after them and to live up to their own high expectations of motherhood (Cox 1982).

In a prospective study of postnatal psychiatric disorder, using a representative sample of 101 postnatal women, Cox et al (1982) estimated a 13% prevalence rate of postnatal depression within the first 3 months following childbirth. Two findings emerged from the study that proved influential in the development of the Edinburgh Postnatal Depression Scale (EPDS). Firstly, Cox et al (1982) identified that most of the women with postnatal depression had not received any sustained treatment or referral to mental health services. It was noted that, in the main, despite fairly intense contact between mothers and general practitioners, midwives and health visitors, professionals failed to identify or properly respond to maternal depression. Secondly, Cox et al (1982) considered that the screening tools used to assess mood had limitations when applied to childbearing women. Measures for depression tended to over emphasise the somatic symptoms of depression, which in the puerperium could be associated with the physiological changes of childbirth. Cox et al (1982) also proposed that primary care workers such as health visitors might be reluctant to use time-consuming questionnaires with poor validity. At the same time there was a consensus amongst researchers for the need to modify existing screening tools or develop new ones for use in community samples with specific clinical conditions (Williams et al 1981; Snaith 1983; Kumar 1983; Cox et al 1982).

Using a sample of 84 postnatal women, the 10-item self-report EPDS was validated as a specialised tool to confirm postnatal depression where it was already suspected by the health visitor (Cox 1987). The majority of mothers were recruited from an existing study into therapeutic interventions for postnatal depression with a control

group of 12 *normal women* included. *Subjects* completed *the 10-item EPDS* at 6 and 11 postnatal weeks, prior to assessment using Goldberg's Standardised Psychiatric Interview (Goldberg et al 1970) with EPDS total scores (0 – 30 points) compared with a diagnosis for depression using the Research Diagnostic Criteria (Spitzer et al 1975).

Based on a validated cut-off score of ≥13 to identify *risk* of depression (Cox (1987), which may be achieved by several low scoring variables or a few high ones, the EPDS was found to demonstrate good sensitivity (86%) and specificity (78%) with a positive predictive value of 73%: Lowering the EPDS cut off to 9/10 was found to improve sensitivity to over 90% with the reduction in specificity to around 51% considered less important in a community setting. Cox (1987) concluded that the EPDS offered simplicity and validity in assessing postnatal depression, was *useful in routine work* and valid when administered in a domiciliary setting (Cox 1987). Even so, it was emphasised that the EPDS was not a substitute for clinical assessment, with the recommendation that those identified as *at risk* should be '*assessed by the primary care worker to confirm whether or not clinical depression is present'* (Cox 1987).

More recently the status of the EPDS as a single construct measure of postnatal depression has been challenged. Studies using factor analysis confirm that the EPDS is comprised of two sub-scales (Brouwers et al 2001; Ross et al 2003) which have been interpreted as measures of discrete depression and pathological anxiety (Brouwers et al 2001; Ross et al 2003).

2.4.iii Tensions and challenges to the use of the EPDS

In general, there was little resistance to the routine screening of postnatal women for depression using the EPDS. That which did occur arose from concerns that health visitors might be deskilled, with experience and intuition in detecting emotional problems postpartum devalued in favour of a screening tool (Barker 1998). Barker (1998) also considered that routine screening could be counterproductive and damaging to some mothers. However, this concern was firmly rebutted from a research perspective by the fact that the EPDS had been more successful in identifying women with postnatal depression at 6 weeks postpartum than the clinical judgement of health visitors (Holden 1994). Barker's views (Barker 1998) were seen as *sentimental* and to contribute to the deskilling of health visitors who were required to develop a sound knowledge base in order for the profession to survive (Taylor 1998).

As far as validity was concerned the level of replication by the numerous studies investigating postnatal depression using the EPDS served to substantiate that, from a research perspective, no further evidence was required to support its routine application (Holden 1994). However, tensions did surface around the routine use of the EPDS but they were mainly in relation to practice issues and training. Whilst the EPDS was intended to identify depression at the lower diagnostic threshold in order to facilitate early intervention, it was acknowledged that this imposed additional demands upon health visitor time (Lee 1998). In addition, Leverton and Elliot (2000) highlighted a number of implications arising from routine screening. In particular, the availability of therapeutic services in response to detection of postnatal depression would depend upon local resources with little consideration given to the quality and

quantity of training and supervision necessary for health visitors to undertake therapeutic interventions. Furthermore, Leverton and Elliot (2000) identified the need for specialist care that involved mental health workers, where listening visits (Holden 1989) by health visitors were considered inappropriate. Also, it was recognised that primary and secondary care providers would require education and training regarding the EPDS and the repercussions of routine screening (Aitken and Jacobson 1997). Importantly, Cox (1987) recognised that health professionals required more training than the information provided by research reports in journals. Others advocated that researchers should take responsibility for training purchasers and providers to interpret both the findings and limitations of research (Beutler et al 1993) and how to integrate outcomes with existing knowledge and the views of users (Geddes et al 1998; Roth and Fonagy1996). Barker et al (1998, 1999) criticised that such recommendations had been ignored by purchasers and managers who were responsible for determining health care systems with the routine use of EPDS '*driven by policies to tick boxes and checklists'*.

Ultimately, the concerns highlighted by Barker (1998) were fully aired at the Society for Reproductive and Infant Psychology Annual Conference in 1999: Leverton and Elliot (2000) reported on a number of inappropriate perceptions and beliefs that were suggested to be held at the interface of the health visitor/ client relationship, regarding the EPDS and *postnatal depression*. Those inappropriate perceptions and beliefs included;

- 'The EPDS identifies depression, meaning that it is *diagnostic*'.
- 'The EPDS can be used as a 'backup in cases of uncertainty', again implying confirming a diagnosis'.

- 'A score below cut-off on the EPDS confirms a diagnosis of no disorder'
- 'Women can pass or fail the EPDS implying both a clear dichotomy and a value judgement'.
- 'The EPDS administered on 1 occasion can determine mental health, that is, mental health over the first postnatal year'.
- 'The EPDS system was designed to assess the wellbeing of (health visitor's) clients'.
- 'Adoption of the EPDS screening system requires "imposing its use on all new parents"'.
- 'Imposed EPDS's are as valid as EPDS's administered with the person centred approach'.
- 'The EPDS indicates the replacements of health visitors' who "called to inspect bathrooms" by those using "pop psychology".
- 'The EPDS should replace the judgement of a trained professional'.

Despite these tensions and challenges around the routine use of the EPDS (Leverton and Elliot 2000) and its failure to meet the criterion for a valid screening tool given the wide variation in estimates of EPDS postnatal depression (UK NSC 2010) the scale remains the most frequently used measure of *postnatal depression* in research and practice (Gibson et al 2009). Based upon Wilson and Jungner's (1968) criteria the EPDS has arguably asserted considerable influence upon the progression of postnatal depression from a clinical concept to its status as a clinical disorder that, by definition, is viewed as an '*important health problem for the individual and community*'...'*with an acceptable screening test' (*Wilson and Jungner *1968)*.

2.5 Section summary: Postnatal depression as a clinical construct

Following improvement in the physical health of the nation scientific attention turned to concern over the deleterious impact of maternal atypical postnatal depression upon offspring and the family unit. Maternal worry over infant health and well being was considered symptomatic of postnatal depression. The failure of community health care professionals to respond appropriately to depression in mothers motivated the development of the EPDS as a specific measure of postnatal depression, prompting the widespread screening of postnatal women. Despite concerns over the EPDS the scale remains the most frequently used measure of *postnatal depression* both in research and practice.

2.6 Chapter summary

By the late 19th century political and scientific opinion converged in public health legislation that focused upon mothers and medicalised childcare. The loss of a child became regarded a social stigma which could potentially be avoided if mothers followed the advice of healthcare professionals (Ross1993, pg. 202). *Worry* was noted to define mothers (Ross 1993, pg. 202). By the mid 20th century professional concern over physical health shifted to concern over the impact of maternal *atypical* postnatal depression (Pitt 1968) on the health and development of infants. The endocrine event of parturition was considered to be causal and maternal worry expressed over the wellbeing of objectively well infants was seen to be symptomatic of *postnatal depression*. Despite concern over the screening childbearing women for postnatal depression using the EPDS, the scale remains the most frequently used measure both in research and practice.

2.7 Conclusion

The literature for *postnatal depression* confirms that its history is *recent*, *Northern European* and mainly *English speaking* in origin, with some of the earliest references occurring as late as the 1950's (Hemphill 1952; Osmond 1953). Mothers remained the focus of ongoing scrutiny at public health level with the widespread belief that childbearing posed an innate risk for maternal depression. The prominence of postnatal depression gathered momentum without the counter challenge of a normative model of postnatal mental health. The practice of screening women for postnatal depression using the EPDS was assigned to health visitors by psychologists, not because of any relevant expertise but because of their access to postpartum women. By default screening for postnatal depression with the EPDS may have been instrumental in promoting the concept of postnatal depression to its implied status as a clinical disorder with both the scale and the concept failing to meet the criteria for screening. More recently, the methodological limitations of the EPDS are illustrated in findings for sub-scales that are proposed to measure depression *and* anxiety, challenging its validity as a single construct measure of depression.

Chapter 3 to follow considers the second theme of this thesis which reviews literature for depression and anxiety, women and childbearing.

CHAPTER 3

THEME TWO: *INVESTIGATION INTO DEPRESSION AND ANXIETY AND HOW IT RELATES TO WOMEN AND POSTPARTUM MOOD*

3.1 Synopsis

The second theme of this review addresses the first objective of this thesis and examines the literature for depression and anxiety and their concomitance, with an emphasis upon women and childbearing. The aim is to explore the evidence for single disorders and co-existence and to illustrate the discrepancies which emerge between these two areas of investigation that are relevant to the question of postpartum mood. *Theme two* is considered in two sections;

- **Depression and anxiety; women and childbearing**

This section addresses the evidence for depression and anxiety as discrete disorders. It presents an overview of their classification status (DSM, APA 1994; ICD, WHO 1994) and reports on epidemiological findings (general, sex difference and postpartum). Broad explanations for the aetiology of depression and anxiety are also provided.

- **Depression and anxiety; evidence for concomitance**

This section examines the evidence for concomitant depression and anxiety, both in terms of a temporal sequence and co-existence. Similar to the preceding section concomitant depression and anxiety is considered in terms of classification constructs, epidemiology, sex-linked difference, temporal sequence and the clinical significance of threshold and non-threshold symptoms.

3.2 Depression: classification and definition

In practice and in research the diagnostic threshold for depression is informed by the categorical model of disorder, based upon qualitative and quantitative rates of symptoms. According to the DSM -1V (APA 1994) the differential diagnostic threshold for minor or major sub-types of depression is determined by the number and severity of symptoms experienced. Either depressed mood (e.g. sadness or tearfulness) or loss of interest (apathy or anhedonia) must be present. Similar criteria are applied by the ICD-10 (WHO 1994) with mood disorder classified as *Depressive Episode (mild, moderate or severe).* For the purpose of this review all reference to *depression*, unless otherwise stated, includes both major and minor disorders and excludes bi-polar disorder.

3.2.i Characteristics of depression

Broadly, a diagnosis of depression represents a level of emotional disturbance that is dysfunctional and disabling (DSM-IV, APA 1994). Characteristics of depressive disorder (not bi-polar disorder) include wide-ranging symptoms arising from four discrete aspects of functioning.

Box 1 provides an overview of the symptom domains and characteristic features of clinical depression (Gilbert 1997, pg 1)).

Characteristics of clinical depression
1. Motivational – Apathy, loss of energy and interest: things seem pointless, hopeless. 2. Emotional – Depressed mood, plus emptiness, anger or resentment, anxiety, shame, guilt. 3. Cognitive – Poor concentration, negative ideas about self, the world and future. 4. Biological – Sleep disturbance, loss of appetite, early morning waking, changes in hormones and brain chemicals.

Box 1 (Gilbert 1997, pg 1)

3.3 Depression: incidence and prevalence

Incidence is defined as '*the number of new cases in a particular period. Incidence is often expressed as a ratio, in which the number of cases is the numerator and the population at risk is the denominator*' (Mosby's Medical Dictionary 2009).

Epidemiological investigation into the *incidence* of depression has been limited. Murphy et al (1988) cite two reasons for this. The first reason was methodological with the proposed need for a prospective, longitudinal follow-up design in order to identify first onset and recurrence (Lapouse 1969). Although such an approach is recognised to be ideal, more recently, it is suggested that a retrospective approach using a life history method with a genetic design is promising and more pragmatic

(Kalidini and McGuffin 2002). The second reason offered by Murphy (1988) is that the onset of psychiatric disorder is typically insidious and therefore difficult to identify. Findings from the Stirling County Study (Murphy et al 2000) suggest a stable incidence of depression between 3-5 per 1000 annually with lifetime prevalence rates estimated to range between 6% and 17% (Blazer et al 1994; Angst 1997); peak onset typically occurs during the 3rd decade APA 1994; Murphy 1988). Comparison between primary care and general population samples, investigated by Maire et al (1997), found rates of Major Depressive Disorder (MDD) of 9.5% and 4.5%, respectively.

Findings from a range of epidemiological studies, reviewed by the Cross-National Collaborative Group (CNCG 1992; Weisman 1993), suggest that a significant increase in rates of depressive disorder has occurred in those born more recently. Whilst these findings have been considered to potentially result from the *cohort effect* (Kessler 2003) a number of long-term, prospective epidemiological studies have demonstrated that this increase in rates of depression is real (e.g. Hagnell et al 1982: CNCG 1992; Murphy et al 2000).

Widespread evidence exists for geographical variation in the prevalence rate of depression. Prevalence is defined as 'The proportion of individuals in a population having a disease...a statistical concept referring to the number of cases of a disease that are present in a particular population at a given time' (Webster's New World Medical dictionary 2008). Large scale epidemiological studies reliably report that the prevalence rate for depression is significantly lower in rural areas than urban areas (Brown and Harris 1978; Jenkins et al 1997; Biji 1998; Ayuso-Mateous et al 2001) with rates across rural areas similar and stable compared to the huge variation in prevalence found in urban areas (Brown and Harris 1978; Andrews et al, 1999).

Significantly, Ayuso-Mateous et al (2001) established that the female/ male difference in depression distribution is greater in urban settings compared with rural areas.

3.3.i Sex linked difference in depression: incidence and prevalence

Epidemiological studies consistently suggest that depressive disorder is, on a global scale, the principal cause of morbidity in women (Murray and Lopez 1996) with the female/male prevalence ratio around 2:1 (e.g. Kessler 2003; Nolen-Hoeksema and Grigus 1994; Parker et al 2001). This female/male difference in the prevalence of depression is reported to first manifest with the onset of puberty, typically between 11 and 14 years of age (Angold et al 1998), although no statistically significant difference exists in the mean age of onset between males and females (Weissman et al 1993).

Investigating the incidence of depression, Kessler et al (1993) reported a higher lifetime rate of *first onset* in women compared with men, but not for recurrence or chronic progression. Previously it has been suggested that postnatal women contribute significantly to rates of first onset of major depression (Wisner et al 1993: Gotlib et al 1989) but only where there is a strong family history of depression (Sichel 2000). Data from the Stirling County Study (Murphy et al 2000) contributes to a more comprehensive understanding of this picture, demonstrating that rates of depression between men and women were comparable in cohorts of 1952 and 1970. By 1992, Murphy et al (2000) identified a two fold increase in the prevalence of depression in those less than 45 years, when compared with subjects from 1952 and 1970 cohorts. Importantly, this increase has occurred specifically in women.

Overall, there are no consistent findings to support a discrete biological, sex linked difference in depressive phenomenology, chronicity or recurrence (Murphy et al 1988; Young et al 1990; Kessler et al 1993; Weissman et al 1993; Breslau et al 1995). However, the reporting of a sex linked-difference in female/ male rates of depression has inevitably stimulated investigation into the potential impact of a range of variables.

Previously, the role of sex hormones in depression has assumed an important focus given that the onset of the sex difference in prevalence occurs with puberty (Angold 1998). Also, changes in levels of sex hormones such as the menstrual cycle, menopause, pregnancy and use of the oral contraceptive pill have been associated with fluctuations in mood (Campbell 1992). Whilst a relationship between oestrogen and progesterone and nueurotransmitter function is acknowledged there is little reliable evidence of their role in major depressive disorder (Yonkers et al 2000).

Findings for the influence of gender roles in depression suggest that women have a higher risk of *first onset* depression but not of staying depressed when compared to men, supporting the absence of role-induced chronicity (Kessler and McLeod 1984). Arguments for a self-report bias in the female/ male ratio in the prevalence of depression, suggesting that women are less resistant to reporting depressed mood than men (Gater et al 1998) are also unsupported (Kessler 1994; Kendler et al 1997; Young et al 1990). Overall, such findings are interpreted to point to the potential of other causal factors such as the modulating effects of environment and biography in the female/ male rates in affective disorder (Mullen et al 1996; Kessler 1997; Lehtinen et al 2003; Dowrick et al 2002).

3.4 Postnatal depression

Postnatal depression has the reputation of being atypical (Pitt 1968) and is widely proposed to represent the most commonly diagnosed mood disorder in the puerperium (Kumar and Robson 1984; Cox 1987; Cox, 1989; Holden 1991; Affonso et al 2000). The prevalence of postnatal depression is typically cited at between 10%-15% of all postpartum women (Pitt 1968; Kumar and Robson 1984; Cox 1987; Holden 1991) although rates as high as 73% have been reported (Affonso et al 2000).

Findings from the most recent systematic review conducted by Gavin et al (2005) suggests that the point prevalence for combined minor and major depression rose until the 3rd month postpartum to 12.9% following which it declined; the estimate for major depression alone at 2 months postpartum, which is reported as one of three peak times postpartum, is reported at 5.7% (Gavin et al 2005). This rate is lower than estimates in primary care samples (9.5%) and only marginally higher than for the general population (4.5%) as reported by Maire et al (1997). Although there were few estimates of period prevalence it was found that the rate of minor and major depression combined during the first 3 months postpartum was as high as 19.2% of all postnatal women with the rate for major depression alone estimated at 7.1% (Gavin et al 2005).

Incidence of postnatal depression is defined as the *first onset of* depression occurring within the first year postpartum (Cooper et al 1988; Hannah et al 1992; Cox et al 1993; Yamashita et al 2000). Gavin et al (2005) reported on the relatively limited findings for the incidence of postnatal depression, which was estimated to be raised in the first 3 months after delivery with the rate of first onset minor and major depression combined reported to be as high as 14.5%. However, a number of studies

which investigated postnatal depression using a control group report no significant difference in estimates of first onset, recurrence, and clinical features, between postpartum women and non-child bearing groups (Cooper 1998, O'Hara 1999).

Overall, findings for postnatal depression are inconsistent with reports of *lower rates of depression* detected in the postpartum period compared with the 3rd trimester (Josefsson et al 2001; Evans et al 2001; Ross et al 2003); higher rates detected postpartum compared with the antenatal period (Watson et al, 1984; Kumar and Robson 1984; Gavin et al 2005), and higher rates at nine months postpartum than at three months (Nott 1987). Furthermore, Affonso et al (2000) in an explorative, descriptive study examined rates of depression symptoms in postnatal women across 5 continents and found some cultural variation in prevalence. (Although *cultural variation* was not defined, based upon the variables investigated in the study (Affonso et al 2000) the term is interpreted here to represent differences between the groups studied, in their shared attitudes, values, goals, and practices). Adding to this conflicting picture, studies investigating postnatal depression that included a control group of non-postnatal women established *lower* depression rates in postnatal subjects (Blazer 1994; Whiffen and Gotlib 1993; Eberhard-Gran et al 2002). There has been a recent increase in interest into maternal anxiety as a specific feature of *postnatal depression.* Ross et al (2003) argues that the presence of an EPDS sub-scale that has been interpreted to represent pathological anxiety provides '*...important evidence for postpartum depression as a specific diagnostic construct, distinct from major depression'.*

The findings of Gavin et al (2005) were generally lower than prior systematic reviews because studies investigating only self-report, which tend to yield higher rates of depression, were excluded (Gavin et al 2005). Overall Gavin et al (2005) found that the estimates for point and period rates and the incidence of postnatal depression were inconsistent over time with wide confidence intervals leading to a '*considerable amount of uncertainty'*. Furthermore, findings did not support the hypothesis that the rate of perinatal depression was higher than for depression at other times of life (Gavin et al 2005).

Gelder et al (2009) adopts a strident approach to clarifying the position of depression postpartum noting that "*postnatal depression' has the danger of introducing...the mirage of a homogeneous disorder... Not surprisingly, research into its causes has found that they are the same as those that cause depression at all ages—heredity, a history of previous or prepartum depression, 'neuroticism', adverse events or social conditions, difficult relationships, and social isolation'*.

Perinatal psychiatric disorders are reported to be the leading cause of maternal death since 1995 with over half of these due to suicide (NICE 2007). Maternal depression in particular is associated with the greatest number of suicides with the majority of these occurring between 34 weeks gestation and 12 weeks postpartum (NICE 2007). However, the rate of perinatal suicide is estimated to be lower than the rate of suicide in the female population as a whole (Appleby 1991; Appleby et al 1998).

3.5 Aetiology of depression: the interaction between biology and environment

At any time of life individual propensity to depression is mediated by the interaction between genetic and environmental factors (for example Tsigos and Chrousos 2002;

Kendler 2006; Goldberg 2006) particularly the experience of trauma in the formative years (for example, Gunnar and Donzella 2002; Heim et al 2000; Heim and Nemeroff 2001). In humans the hypothalamic-pituitary-adrenal axis (H-PA) is central to the regulation of mood, governs the body's reaction to stress (Tsigos and Chrousos 2002) and has been shown to undergo changes in pregnancy and the puerperium (Magiakou et al 1997; Wadhwa et al 1996). Fundamentally, the H-PA mechanism controls the ratio of activation between the parasympathetic nervous system (controlling rest, recovery and energy storage) and the sympathetic nervous system (controlling arousal, emergencies and expending energies) (Bownds 1999 p 237). H-PA reactivity is genetically pre-set with a number of independent genetic pathways responsible for individual vulnerability to depression (Goldberg 2006). The genotype is reported to account for 40% of the variance in men and women (Thapar and McGuffin 1998; Kendler and Prescott 1999) with the heritability of depression higher in females (42%) compared with men (29%) (Kendler et al 2001; Hong and Tsai 2003; Kendler 2006).

It is now widely accepted that the interaction between genes and environmental factors such as poor emotional and social support (Kumar, 1990; Kendell et al, 1981; Paykell et al, 1980; Cox et al, 1989) and death of a mother before the age of 15 years (Harris, 1986; Shrout et al, 1989; Tweed 1989) mediate the risk of depression and anxiety (for example Brown and Harris 1978; Sullivan et al 2000; Ebmeier et al 2006) at any time of life. Even so, the exact aetiological mechanism remains unclear (Goldberg 2006). Most significant in H-PA reactivity to stressors is the quality of infant-maternal attachment; *good* attachment decreases responsiveness whilst maternal deprivation increases responsiveness (Hofer 1984; Shekhar et al 2001).

Notably, emotional deprivation and maltreatment in childhood have been shown to increase H-PA responsiveness (Gunnar and Donzella 2002) resulting in increased risk of depression in adulthood (Heim et al 2000; Heim and Nemeroff 2001).

Findings from a 10 year longitudinal study conducted by Brown and Harris (1978) established depression as a *social phenomenon* which manifests in the presence of poverty, social and emotional deprivation and negative life events (Brown 1989). Urban, working class women were more likely to develop a psychiatric disorder (predominantly depression) than their rural counterparts. Brown and Harris (1978) also confirmed an increased risk of psychiatric disorder in association with motherhood. However, far from identifying any causal factor for depression specifically associated with childbearing, their findings suggest an increased risk *only* in the presence of ongoing difficulty, for example, the lack of a close confiding relationship and poor economic circumstances (Brown and Harris 1978). More recently, findings from the ODIN Study (Lehtinen et al 2003; Dowrick et al 2002), a cross-sectional, multinational, community survey of five European countries, endorsed those of Brown and Harris (1978). Furthermore, the difference found between rural and urban rates of depression in women are reported to be readily explained by the socio-demographic factors studied (Lehtinen et al 2003; Dowrick et al 2002).

Overall, the accumulative evidence confirms that, regarding depression, there is no clear difference between childbearing and non-childbearing populations, in relation to cause, onset, symptom profile, chronicity and disease course, (e.g. Kumar et al 1982; Cooper et al 1988, O'Hara 1990; Whiffen 1992; Whiffen and Gotlib 1993; Eberhard-Gran 2002). This similarity in depression profile, between postnatal and non-

childbearing groups, is reflected in the current absence of any diagnostic classification for postnatal depression. However, arguments for a specific diagnostic classification for postnatal depression exist, based upon the reported prominence of anxiety in *postnatal depression* (Cooper and Murray 1995; Ross et al 2003).

3.6 Anxiety disorder: classification and definition

As with depression, the categorical model of disorder determines the threshold for pathological anxiety. According to DSM-1V (APA, 1994) the classification coding of anxiety sub-types represent distinct anxiety disorders in contrast to the sub-types of unipolar depression.

3.6.i Characteristics of anxiety

In view of the fact that a range of anxiety disorders are classified, the purpose of this thesis is adequately served by a fundamental description of pathological anxiety: The ICD-10 (WHO 1994), in its general introduction to anxiety disorders, identifies symptoms of anxiety that are common across the classified sub-types;
'As in other anxiety disorders the dominant symptoms are highly variable, but complaints of feelings of nervousness, trembling, muscle tension, sweating, light headedness, palpitations, dizziness and epigastric discomfort are common' (ICD-10, WHO 1994). In contrast to postnatal depression neither the ICD-10 (WHO 1994) nor the DSM-1V (APA 1994) considers any specifier for anxiety disorders in association with childbearing.

3.7 Anxiety disorders: incidence and prevalence

Data from large scale epidemiological studies estimate the overall lifetime prevalence for anxiety disorders at almost 15% (LePine 2002) with 1 in 4 of the general

population proposed to suffer (Kessler 1994; Reiger 1990). Data for anxiety sub-types suggest an estimated lifetime prevalence of general anxiety disorder of 5-6% (Boyd 1986; Kessler 1994); panic disorder, 1.5% - 2% (Eaton et al 1991), with rates of social anxiety disorder varying from relatively rare (3%) (Boyd 1990; Schneier 1992) to widespread (13%) (Kessler 1994; Biserbe1996; Kessler 1998). A high, lifetime prevalence has also been shown for obsessive compulsive disorder with rates of 2%-3% (Karno 1988; Weissman 1994). Twenty five per cent of individuals who have experienced traumatic events are suggested to develop post traumatic stress disorder with a substantially increased risk of onset in women compared with men (Breslau 1994).

3.7.i Sex linked difference in anxiety: Incidence and prevalence

A number of studies confirm that female sex is associated with an increased risk of anxiety (Breslau 1995; Murphy et al 1988) with a lifetime prevalence of any anxiety disorder of 25% and 43% for males and females, respectively (Breslau 1995). The lifetime prevalence, female: male ratio is estimated of approximately 2:1 for panic disorder (7.7% V's 2.9%) (Keyl et al 1990), simple phobia (13.9% V's 7.2%) and post traumatic stress disorder (12.5% V's 6.2%) (Kessler1994; Kendler1992). Less sex difference has been established in rates of social anxiety (3.1% V's 2.3%) (Turk et al 1998) and obsessive compulsive disorder (OCD) (3.1% V's 2.0%) (LePine 2002). Interestingly, the sex difference in OCD is not maintained across all age groups. Although females are one and a half times more likely to suffer a lifetime prevalence of OCD compared with men a higher mean age of first onset (25 years V's 20 years) is experienced by females (Nesiroglu et al 1994; Thomsen 1995). Women are also twice as likely to suffer generalised anxiety disorder compared with men (Boyd 1986;

Kessler 1994). Despite these findings, population based studies have generally produced less meaningful information about anxiety than depression, a finding perceived to be particularly true for sub-clinical anxiety and those with co-morbid conditions (Brown et al 1994; Judd, 1994).

3.8 Anxiety and childbearing

Exploration of the available literature suggests that some important differences exist between the scientific treatment of anxiety *and* childbearing on one hand, and depression *and* childbearing, on the other. These raise a number of points that have direct bearing upon the interpretation of findings for *postnatal depression.*

Firstly, this difference is highlighted by a recognised lack of clarity in the reporting of anxiety as either a distinct, clinical entity or as a feature of primary depression in perinatal mood (Pitt 1968, 1985; Stuart et al 1998; Green et al 1998; Ross et al 2003). Furthermore, Goldberg (1984) defines *"...the principle of diagnostic parsimony"* where it is proposed that hierarchical diagnostic custom determines that depression, as a label, takes precedence over anxiety. Secondly, Shears et al (1995) noted that until the mid-1990's there were no reviews of assessment, treatment and progression of perinatal anxiety disorders reflecting their secondary status in relation to perinatal depression. Ross et al (2006), in the most recent systematic review of perinatal anxiety suggests that the status quo has been maintained. Specifically, the review was *'limited by the quality of research evidence'* (Ross 2006). In particular there were inconsistencies in the methods of psychometric assessment of anxiety disorders that failed to conform to DSM diagnostic criteria and with no studies found to have included appropriate non-perinatal control groups (Ross 2006). Although Ross (2006)

found that anxiety disorders were common in the perinatal period, few prospective, longitudinal studies were available to estimate whether anxiety onset pre-dated childbirth. It was noted that one consequence was the difficulty in assessing from the available evidence whether causal factors relating to pre-existing anxiety differed to perinatal onset: Conceptually, there is no anxiety equivalent to postpartum depression. Thirdly, and finally, non-pathological *separation anxiety* is recognised to represent a *normative* experience (Hock and Schintzinger 1992; Feldman et a 1999; Nesse 2000), a status not commensurate with features of depression.

Reports suggest that anxiety symptoms can occur in pregnancy and the puerperium and may be divided into those meeting the diagnostic threshold (e.g. panic) and those that do not meet clinical significance (e.g. maternal separation anxiety). A review of the literature for the relationship between childbearing and existing anxiety disorders is outside the scope of this thesis. Therefore the following sections consider findings for the relationship between anxiety and childbearing where anxiety is reported as *secondary* to childbearing.

3.8.i Non–pathological anxiety and childbearing

Pigott (1999) in a review of studies investigating anxiety and childbearing suggests that women can experience a degree of psychological distress and somatic symptoms without past or existing mental health problems and without representing serious psychiatric disturbance. In a longitudinal study of healthy pregnant women between the 31st and 33rd week gestation, Areskog et al (1981) established that hypochondrial concerns were common and increased as pregnancy progressed; 6% expressed severe fear of labour and delivery with 17% expressing moderate fear.

Anxiety complaints such as fear of dying have been found to occur frequently during the third trimester, at a higher rate than the control group of non-pregnant women (Fava, 1990).

3.8.ii Maternal separation anxiety

Hock and Schintzinger (1992) describe maternal separation anxiety as a normative *'unpleasant emotional state evidenced by worry, sadness or guilt and…. linked to the mother-child separation experience'*. On the other hand high levels of maternal anxiety are suggested to erode maternal confidence, contribute to some difficulty in adapting to the maternal role and to result in the poor psychosocial development of offspring (Barnett and Parker 1986). Barnett et al (1990), in a naturalistic cohort study, applied a battery of psychometric tests to assess how first time mothers adapted to parenthood. It was found that highly anxious mothers continued to show greater pathology at both one year and five years after childbearing. Such mothers were likely to report more problems, more distress and less confidence in themselves and their circumstances (Barnett and Parker 1986). Subjects also perceived their attachments and social integration to be less satisfactory with limited social networks that were inadequate and unavailable on both an intimate as well as a more casual level (Barnett and Parker 1986).

It remains unclear whether a pathological level of maternal separation anxiety can occur independently of other psychiatric disorders. Whilst a relationship has been shown between maternal separation anxiety and psychological variables such as spouse support, infant temperament (Mayseless 2000), employment and child care (Houndoumadi 1996) no studies could be found to have investigated an association

between normal maternal separation anxiety and the presence of psychiatric disorders. In turn, the role of anxiety as a contributor to functional maternal adaptation has generally received scant attention. From an ethological perspective, separation anxiety is believed to ensure that the mother institutes highly responsive care for her infant and diminishes as the mother feels confident that the infant will be safe in her absence (for example, Bowlby 1969; Feldman 1999).

3.9 Aetiology of anxiety: the interaction between biology and environment

As outlined in the section addressing the aetiology of depression (see Chapter 3, pg. 32-35), the stress response in humans is controlled by the H-PA axis, the reactivity of which is genetically predetermined and mediated by early life experiences (for example, Goldberg 2006; Heim et al 2000). The same neurobiological mechanisms are implicated in both the normative and pathological anxiety response in humans. In addition, the unified functions of the hypocampus and the amygdyla are concerned with the formation of emotional memories and with evaluating emotional stimuli (Bownds 1999 p 247 - 248). Specifically, the hypocampus is involved in cognition and learning and the amygdyla is concerned with the processing of images and words, associating them with positive or negative affect and with the formation of specific reactions to anxious stimuli before they reach the level of awareness (Bownds 1999 p 251). At a psychotherapeutic level anxiety disorders are considered to arise from cognitive dysfunctions processing maladaptive schema and behaviours (Hindmarch 1998).

Similar to depression, genetic and environmental factors are considered to mediate individual risk to anxiety (e.g. Kendler and Neale 1996). In a study of 1,033 female

twin pairs from a population registry, Kendler and Prescott et al (1999) found that, for example, General Anxiety Disorder (GAD) is moderately familial (approximately 30%). It was hypothesised that specific and distinct environmental factors mediate the development of anxiety disorders but only where the genotype is present (Kendler and Neale 1992). Although psychosocial stress arising for factors such as poverty, inadequate housing and poor social support are known to predispose to anxiety disorders in the general population, no systematic investigation could be found into the relationship between these variables and anxiety disorders in pregnancy and the postnatal period.

3.9.i Aetiology of anxiety: endocrine theories

The critical role played by oestrogen and progesterone in the regulation of neurotransmitters suggest that they are implicated in the anxiety response. Consequently, their significance in female reproduction has been considered to provide a possible explanation for the greater susceptibility of women to anxiety disorders compared with men (Pigott 1999; Seeman 1997; Arpels 1996; Fink 1996). Both oestrogen and progesterone are considered neuroprotective. Progesterone in particular is thought to exert an anxiolytic effect in women (Seeman 1997; Halbreich 1997; Jenswold 1996; Sherwin 1996) although it is also linked to dysphoria and thought to oppose the neuroprotective influence of oestrogen (Halbreich 1997).

3.10 Section summary: Depression and anxiety; women and childbearing

In summary, the reporting of depression and anxiety as single disorders highlights a female/ male ratio of 2-3:1 with women reported to experience increased heritability of both, compared with men. Whilst the sex-linked difference in depression is shown to

emerge with puberty, findings for anxiety are less precise. Even so anxiety and depression are recognised to have a shared diathesis with individual risk of disorder mediated by the interaction between neurobiological and environmental factors with the latter exerting the greatest influence.

Similar rates of depression have been shown to exist between postnatal and non-childbearing populations, with no difference found between men and women, in aetiology, symptom profile or disease course. Furthermore, in contrast to the pure pathology of depression in relation to childbearing, anxiety is understood to represent a defence mechanism with maternal separation anxiety viewed as a *normative* response to childbearing. Unlike depression postpartum, only bipolar disorder is considered to be precipitated by the endocrine event of pregnancy.

Depression and anxiety; evidence for concomitance

3.11 Co-morbid anxiety and depression: classification constructs

Evidence for the relationship between depression *and* anxiety (co-existing and temporal sequence) has emerged chiefly through longitudinal epidemiological investigation, concerned with distinguishing between *incidence, prevalence, recurrence and chronicity.* In turn, *definitions, diagnosis* and *incidence* are factors proposed to be central to the interpretation of epidemiological data (Murphy et al 1988). However, variations in the theoretical constructs and idioms used to express concomitant depression and anxiety are considered to represent important dilemmas for the interpretation of psychiatric epidemiological data (Murphy et al 1988; Clark and Watson 1991; Murphy et al 2000).

As a clinical concept the history of co-existing depression and anxiety has been complex and controversial. For example, from a philosophical and measurement viewpoint Judd et al (1996) considered co-morbid depression and anxiety as a *nuisance* variable whilst the association, according to Goldberg (1984), is an artefact of diagnostic categories. From a clinical perspective depression has been considered to represent a complication of anxiety (Angst et al 1990) or a marker of the severity of anxiety in youth (Kovacs 1990).

Previously, Clark and Watson (1991) succinctly summarised the diversity of views regarding the relationship between depression and anxiety, in that (a) *they represent different points along the same continuum, (b) are alternative manifestations of a common underlying diathesis, (c) are heterogeneous syndromes that are associated because of a shared subtype, (d) are separate phenomenon, each of which may develop into the other over time, and (e), are conceptually and empirically distinct phenomena.*

The traditional and categorical view (point (e) above) is largely responsible for shaping diagnostic criteria (Sthal 1997), and identifies co-morbid depression and anxiety as distinct and unrelated disorders (Claghorn 1970; Downing and Rickels 1974; DSM-III, APA 1989). However, the sheer volume of evidence to support that mixed depression and anxiety is more common than pure disorders (for example, Rapport 2001; Kessler 1998;Stein 1995) has led classification systems to respond with specific definitions of co-occurring anxiety and depression, namely *Co-morbid Anxiety and Depression* (CAD) (DSM-1V-Tr, APA 2000) and *Mixed Anxiety and Depression* (MAD) (ICD-10, 1994). The CAD perspective requires that *both* anxiety and depression meet full DSM-1V (APA, 1994) criteria where neither is considered

part of the other but acknowledges their concomitance. DSM-1V (APA 1994) criteria do not consider co-existing depression and anxiety below the diagnostic threshold of clinical significance (Schotte and Cooper 1999), recommending that researchers construct their own criteria for sub-threshold disorders where no specifier exists (DSM-1V-Tr, APA 2000).

In contrast, MAD (ICD-10, WHO 1994) criteria represent a cluster of depression and anxiety symptoms with neither required to meet the diagnostic threshold for disorder if considered separately. MAD is referred to as *sub-syndromal* (Sthal 1997) or *subthreshold* (Helmchem and Linden 2000). Definitively, a diagnosis of MAD cannot be given where the patient meets the full criteria for any anxiety disorder or depression.

In summary, classification constructs for concomitant depression and anxiety and their epidemiological definitions are inconsistent leaving research outcomes open to interpretation.

3.12 Co-existing anxiety and depression: incidence and prevalence

More recently it is recognised that the frequency of co-existing anxiety and depression is such that it is likely to be the *rule rather than the exception* (Rapport 2001). When measured according to different classification criteria prevalence rates for co-existence have been shown to vary. Such findings illustrate caveats around the use of definitions and diagnosis of psychiatric morbidity which are important factors in epidemiological studies and clinical practice (Murphy et al 1988; Murphy et al 2000). For example Stein (1995) found rates of MAD (42.3%) and CAD (19.2%) varied

significantly; rates for both were higher versus a single anxiety disorder (12.8%) and a single depressive disorder (10.3%).

Between 1952 and 1970 The Stirling County Study conducted by Murphy et al (1988) examined the incidence and prevalence of psychiatric disorders in relation to age in the adult population, using a longitudinal investigation that consisted of repeated cross sectional surveys and cohort follow-up. Findings demonstrated that subjects diagnosed with depression frequently met the full criteria for anxiety with the overall *incidence* of both being low in relation to prevalence, supporting the role of *chronicity* in rates of psychiatric morbidity rather than *new case* (Murphy et al 1988; Keller 1999). The incidence of aggregated anxiety and depression for any of the follow-up years was established at 9 per 1000 (male: female ratio 1:3) with point prevalence for 1952 and 1970, 12.5% and 12.7%, respectively. Although the aggregated prevalence for women was higher than for men this higher rate was due to anxiety and mixed affective disorder with rates of depression comparable between men and women (Murphy et al 1988).

3.12.i Sub-threshold co-existing anxiety and depression: incidence and prevalence

Studies consistently show that co-existing anxiety and depression, *below* the diagnostic threshold for either is more common than single disorders (Zinbard et al 1994; Stein et al 1995; Wittchen and Essau 1993; Von Korf et al 1987; Angst et al 1997; Brown et al 1996; Roy-Byrne et al 1994; Zung et al 1990; Helmchem and Linden 2000). Pini (1999) investigated psychiatric disorders in primary care and found a prevalence rate of sub-threshold disorders of 30.4% compared with major

depression (13.4%), dysthymia (2%), general anxiety disorder (8.4%), panic disorder (1.6%) and agoraphobia (2%) with similar findings reported by others (Papassotiropoulos and Ptok 2000; Papassotiropoulos and Heun 1999). Comparing primary care and general population samples Maire et al (1997) found rates for sub-threshold syndromes (ICD-10 1994) of 32.1% and 21.8%, respectively.

In summary, co-existing depression and anxiety, above and below the threshold for disorder, is more common than single disorders and is suggested to represent the norm rather than the exception.

3.13 Anxiety and depression: temporal sequence, co-morbidity and sex linked difference

The consistently higher rate of depression in women, compared with men, is well documented (e.g. Kessler et al 1993, Weissman 1993; Nolen-Hoeksema and Girgus 1994). However, findings from the extensive review of epidemiological and clinical studies conducted by Maser and Cloniger (1990) suggest that this picture might be misleading. The available evidence supports both a high lifetime *co-occurrence* of anxiety and major depressive disorder and a predominant temporal sequence with *anxiety more likely to precede rather than to follow depression* (see Maser and Cloniger 1990).

This temporal sequence was evident both in terms of episode onset (Alloy et al 1990) and incidence (Kendell et al 1974; Angst et al 1990; Kessler et al 1998). Parker et al (1997) specifically identified anxiety in the form of social inhibition or avoidance as likely to precede or to predispose to early onset, non-melancholic depression. Anxiety, co-morbid with major depression, has been found to be prevalent, whilst *pure*

depression, that is depression without a lifetime history of anxiety disorder, is rare (Alloy et al 1990; Reiger et al 1990).

Using data from a longitudinal epidemiological study, Breslau et al (1995) was the first to investigate the interaction between sex, prior anxiety and female vulnerability to major depression. Breslau (1995) proposed that the association between anxiety and depression might be significant in the sex-linked difference in depression for a number of reasons; firstly, the higher rate of major depression in women might reflect their greater propensity to depression following an anxiety disorder. Secondly, even if there was no difference in the risk of depression following anxiety between women and men, the higher rate of major depression in women might be explained by their greater propensity to anxiety disorders. Thirdly, the increased risk for women of developing additional anxiety disorders with the onset of a first, that is, developing co-morbid anxiety disorders, is relevant in the sex linked difference in depression.

In relation to this thesis Breslau et al's (1995) work regarding sex difference in anxiety and depression is important and therefore worth presenting in some detail. Outcomes reported by Breslau et al (1995) suggest that the lifetime prevalence of major depression was 12.6% in males and 22.8% in females. The cumulative percentage for a history of major depression by 14 years of age was found to be 1.3% in boys and 3.1% in girls with the difference in incidence growing more sharply in females than males in the 3rd decade.

In contrast, the lifetime prevalence of *any* anxiety disorder was 25% and 43% in males and females, respectively; by 10 years of age, the cumulative percentage of *any anxiety disorder* was found to be 8% in males and 17.9% in females. It was also

found that the gap between incidence rates of any anxiety disorder in males and females increased slowly in the 2nd and 3rd decades.

Overall the key findings of Breslau's et al (1995) study support;

1. The lifetime prevalence of anxiety disorders and depression were twofold higher in women than in men.

2. Both men and women incurred a similar, increased susceptibility to major depression in association with any anxiety disorder. *However, more than one anxiety disorder posed a greater risk for males than for females.*

3. The earlier onset of anxiety disorders in female accounted considerably for the observed sex difference in the prevalence of major depression.

4. The higher risk of major depression in females might be secondary to the higher prevalence of anxiety.

5. The higher lifetime prevalence of depression in females versus males was primarily in major depression *co-morbid* with anxiety disorders. These findings were similar to those of Murphy et al (1988).

6. No evidence was found to support that females suffered more depressogenic affects from anxiety than men.

7. When a history of prior anxiety disorder was controlled for, the sex-linked difference in major depression was reduced by 50 per cent.

In summary, Breslau's et al (1995) findings show that anxiety disorders are associated with a marked increase in subsequent onset of major depression in both males and females. The sex difference in the earlier onset of anxiety disorders experienced by females was, in the main, largely responsible for the sex difference in major depression.

Similarly, Parker et al (2001) investigated prior anxiety and sex-difference in depression and dysthymia according to DSM-11-R criteria (APA 1987) using data from the National Co-morbidity Study database. Outcomes were comparable to those of Breslau (1995) supporting that both female sex and prior anxiety disorder made significant contributions to early and late onset depression with anxiety contributing the strongest influence. Investigating the interaction between prior anxiety disorder and sex showed that there was no difference between males and females in the risk of subsequent depression, supporting the conclusion that anxiety serves as a diathesis factor in depression (Parker et al 2001). Parker et al (2001) concluded that a proportion of the female propensity to major depression and dysthymia in the general population is most likely determined by a primary sex difference in anxiety.
Study limitations were considered to arise from inconsistent definitions of anxiety and depression and for early and late onset depression; the sequencing of disorder onset and *scarring* effects are also factors noted to impact outcomes in psychiatric epidemiology (Parker et al 2001).

The World Health Organisation Collaborative Study on Psychological Problems in General Health Care (Gater et al 1998) investigated rates of anxiety and depression (ICD-10, WHO 1994) by sex and the influence of a range of psychosocial variables. The outcomes support the conclusion that, with the exception of GAD, women

experienced higher rates of depression, agoraphobia and panic than men (Gater 1998). No association was found between the psychosocial variables studied and the female: male ratio in rates of anxiety and depression which was interpreted to suggest that biological and psychosocial factors, either interacting or working alone, exerted a similar effect across cultures (Gater et al 1998). Gater et al (1998) acknowledged difficulties in interpreting variations in detection and prevalence and also the potential of a sex difference in reporting and conceptualization of anxiety and depression, rather than any real difference in rates.

3.14 Postpartum women and co-existing anxiety and depression

There is little investigation into the relationship between anxiety and depression in postpartum women (Altshuler et al 1998; Wenzell 2001) in contrast to the level of investigation in non-childbearing groups. Regarding the diagnostic dominance of depression Musik et al (2000) notes that not all anxious parents were depressed but that detection of anxiety symptoms in the postnatal period are likely to contribute to a diagnosis of postnatal depression. Green (1998) also acknowledged that anxiety is generally subsumed within the diagnosis of depression which, consequently, might limit our understanding of maternal distress.

Examination of the literature provides limited information regarding the relationship between depression *and* anxiety in postpartum women. That which is available is based on diagnostic thresholds for discrete disorders and overall, demonstrates a similar picture to non-postnatal samples. Studies investigating self-rated anxiety and depression postpartum, based upon diagnostic thresholds, have proved relatively inconsistent. Stuart et al (1998) found co-existing rates of anxiety and depression

symptoms, at 14 and 30 weeks postpartum, of 8.7% and 16.8%, and 23.3% and 15.7%, respectively; the period prevalence was 10.28% for anxiety and 7.48% for depression with comparable findings established by others (Najman 1990; O'Hara 1991).

Misri et al (2000) established that women with a clinical diagnosis of depression in the puerperium were 30-40% more likely to suffer co-morbid anxiety and also to exhibit *more* anxiety symptoms than women suffering with depression at other times in life. Conversely, mothers suffering from co-morbid anxiety and depression are reported to have significantly elevated levels of depression symptoms compared to those diagnosed solely with depression and were more difficult to treat than for depression or anxiety alone (Emmanuel et al, 1998). Co-morbid sufferers are also reported to experience poorer acute and long term outcomes (O'Hara, 2000; Appleby, 1997) with an increased risk of suicide (Fawcett, 1997).

Using DSM-1V (APA 1994) criteria Wenzell et al (2001) investigated a sample of non-help seeking, dysphoric postpartum women all of whom were reported to experience sub-syndromal symptoms of anxiety and depression; 19.8% of these were diagnosed with panic and /or OCD. In the total sample, major depression was detected in 44.3%, with co-morbid rates of panic and OCD of 2.3% and 5.4%, respectively (Wenzell et al 2001). Similarly, postpartum women with a pre-existing OCD have been found to suffer high rates of co-morbid depression (Williams and Koran 1997). Overall these findings for co-morbid depression and anxiety disorders do not differ significantly from those in the non-childbearing populations.

3.15 Section summary: Depression and anxiety; evidence for concomitance

Inconsistencies in methodological approaches and epidemiological definitions are considered to confound investigation into the relationship between anxiety *and* depression. These factors create difficulties in interpreting data outcomes and their application in clinical practice. Very long term longitudinal investigation has demonstrated a higher female, sex linked risk in anxiety rather than depression, compared with men (Breslau 1995). Furthermore, a temporal sequence has been shown in which anxiety typically precedes depression. Therefore the higher rate of anxiety in women is proposed to be responsible for the 2:1 female/ male ratio in depression, with the risk of depression following anxiety, similar in men and women (Breslau 1995). *This temporal sequence suggests that anxiety acts as a diathesis factor for depression with prior anxiety rather than female sex shown to more reliably predict the onset of depression in both females and males (*Parker 2001*)*. Overall, pure depression, that is depression in the absence of any anxiety disorder, is almost non-existent, whereas co-existing anxiety and depression below the diagnostic threshold is most common (Murphy 1988, 2000).

Although evidence is limited, maternal mood has been shown to exhibit features of both anxiety and depression without necessarily meeting the threshold for disorder.

3.16 Chapter summary

The reporting of depression and anxiety as discrete disorders suggests female/ male ratios of 2:1 and 2-3:1, respectively. Regarding aetiology, anxiety and depression are regulated by the interaction between neurobiological and environmental factors with no difference shown in depression rates, symptomotology, disease course or

chronicity, between postpartum and non-child bearing groups. There is no anxiety equivalent to the clinical concept of postnatal depression although the detection of perinatal anxiety is likely to contribute to a diagnosis maternal depression. In contrast to the pure pathology of depression separation anxiety is seen to represent a *normative* maternal response to childbearing. Furthermore, contrary to the reporting of *postnatal depression* findings suggest that bipolar depression is the only psychiatric disorder precipitated by childbearing.

Concomitance studies highlight important methodological issues regarding psychiatric epidemiology such as the need for very long term investigation in order to confidently discriminate between *incidence* and *prevalence.* Methodological approaches in relation to discrete depression and anxiety, such as the use of cross sectional data and retrospective self-report for psychiatric history are recognised as unreliable. Also, continuous measures such as the EPDS are frequently used in prevalence studies with cut-off scores interpreted as diagnostic and reported as prevalence without clinical confirmation of disorder.

Far from being discrete, unrelated disorders, long term epidemiological studies demonstrate a temporal relationship in which anxiety typically precedes first and episode onset in depression. Furthermore, the 2:1 female/ male ratio in depression is suggested to be secondary to the female, earlier onset, sex linked risk of anxiety. Importantly, prior anxiety, rather than female sex is the most significant predictor of depression with men and women shown to incur a similar risk for depression following any anxiety disorder. Consequently, depression without a history of anxiety is rare with co-existing anxiety and depression below the diagnostic threshold *more* common than single, threshold disorders in both the clinical and general populations.

Evidence for the relationship between childbearing and concomitant anxiety and depression is limited and presents a somewhat unclear picture. From a clinical perspective postpartum women diagnosed with depression are reported to suffer more anxiety symptoms than those suffering depression at other times of life. Co-morbid anxiety and depression is associated with greater morbidity, poorer treatment response and an increased risk of suicide with outcomes in the general and perinatal populations similar. Even so rates of perinatal suicide are altogether lower than for the female population as a whole.

3.17 Conclusion

There is little justification for considering *postnatal depression* as a discrete disordert. This assertion is based upon findings which support that no difference exists between postpartum and non-childbearing groups regarding depression rates, symptomotology and disease course. In addition, long term, prospective epidemiologic investigation into concomitant depression and anxiety, seen to represent the ideal methodological approach to distinguish between incidence and prevalence in psychiatric disorder, have not identified the perinatal period (ante and postnatal) as high risk for the first or episode onset of clinical depression and/or anxiety. Conversely, the puerperium is associated with a higher rate of first onset bipolar disorder which is manifest as puerperal psychosis, but likely only where pre-existing vulnerability exists. However, it is the consistent occurrence of elevated but transient symptoms of depression and anxiety in the first three months postpartum that are interpreted as diagnostic of postnatal depression.

From the perspective of this thesis it is significant that females have been shown to experience an increased primary propensity to anxiety rather than depression, compared with males. Although this female/ male difference is demonstrated in the higher female rate of anxiety disorders it is also evident in *normative* maternal separation anxiety which is aimed at promoting close proximity and ultimately infant survival. Furthermore, co-existing symptoms of depression and anxiety below the diagnostic threshold, that is, experienced *without* significant impairment or distress, is reasonably characteristic in humans. (The suggestion that anxiety and depression can exist both below the diagnostic threshold *and* at a level associated with significant impairment or distress is a contradiction). *Therefore in the postpartum period co-existing symptoms of depression and anxiety, below the threshold for disorder, should be anticipated.*

Chapter 4 explores the third theme which reviews the evidence and theoretical basis for maternal attachment and the potential for depression and anxiety as normative features of postnatal mood.

CHAPTER 4

THEME THREE: *SYMPTOMS OF DEPRESSION AND ANXIETY AS NORMATIVE FEATURES OF POSTPARTUM MOOD*

4.1 Synopsis

Chapter 4 discusses the third theme of the literature review which considers evidence from ethology, psychology and evolutionary theory regarding the utility of anxiety and depression. The aim is to examine evidence and hypotheses for the normative potential of depression and anxiety as features of non-pathological postpartum mood and their role in maternal-infant attachment. Theme three is presented in two sections;

- **Maternal – infant attachment**

This section explores evidence for the neurobiological mechanisms and early parental preoccupations and behaviours that support attachment in the mother-infant dyad. The impact of maternal psychiatric illness on child development is described together with the mechanisms that are proposed to be responsible for the transmission of mother related risk. The intention is to promote some distinction between maternal cognitions and behaviours in relation to childbearing that are functional on one hand and pathological on the other.

- **Evolutionary hypotheses for the utility of depression and anxiety**

This section examines evolutionary hypotheses for the utility of depression and anxiety in general and then in relation to childbearing. The aim is to justify consideration of symptoms of anxiety and depression as phenotypical features of the childbearing experience.

4.2 Attachment theory

Classic attachment theory (see Bowlby 1969, 1973) provides a working model of the biological and psychological mechanisms of *infant-parent* interactions (Holmes 1993). Early attachment experience shapes the *internal working model of interactive patterns* between individuals and attachment figures (Holmes 1993) with this *'attachment dynamic'* operational throughout life *(Heard and Lake 1986). Hofer (2005) states that* the central hypothesis of clinical attachment theory predicts the impact of *'...non-verbal features of the early mother-infant interaction...'* form'*...lasting mental representations of maternal behaviour in the adult....'* through which the *'...trans-generational effects of early experience of maternal behaviour, vulnerability to stress, fear responses and their underlying gene expression'...is transmitted.*

Feldman et al (1999) notes that a*ttachment* has typically been investigated from the infant perspective with maternal bonding behaviours viewed only as facilitators of infant attachment (e.g. Ainsworth et al 1978). One corollary is that little emphasis has been placed on the unique mental representations and internal processes that accompany the formation of a mother's selective and enduring bond with her baby (van IJzendoorn and Tavecchio 1987; Feldman et al 1999). Underpinned by the hypothesis that maternal attachment rests upon the same mechanisms as those underlying infant attachment, Feldman et al (1999) investigated *maternal–infant attachment* through the components of proximity, separation and loss. Data for maternal thoughts and worries (for example, over infant safety, comfort, dependency, vulnerability and something bad happening), affiliative behaviours (for example, repetitive caretaking behaviours and interactions with the infant) and attachment

representations (for example "*finding him the most beautiful baby*") identified two distinct global paradigms of maternal attachment (Feldman 1999). The first relates to pre-occupations with infant safety and wellbeing, and the second to the building of a selective bond (Feldman 1999). Both were shown to include bio-behavioural and mental components with the former precipitants of bonding behaviours in mammals (Instel 1997) whilst the latter is unique to the human adult (van Ijzendoorn and Tavecchio 1987). The construct of "attachment" is therefore proposed to define the mother-infant relationship and is not a characteristic exclusive to infants (Bridges, Connell & Belsky 1988; Hinde 1989; Feldman 1999).

4.2.i Bio-behavioural and mental components of mother –infant attachment

Mother-infant attachment in humans cannot be experimentally manipulated (Feldman 1999). The necessary consequence, as noted by Taylor et al (2000), is the lack of a coherent model for *mother-infant attachment*, with the literature for neuroendocrine mechanisms informed mainly by rodent studies and observed bonding behaviours informed by primate and human studies. Although findings from animal studies require a degree of caution in their application to humans they have provided valuable evidence for the relationship between the biophysical and behavioural component of attachment in both infants and mothers. For example, the work of Hofer (e.g. 1973, 1984, 1995), using rat pup-dam dyads, showed mother–infant proximity to be moderated by complex behavioural regulators which underpin primary attachment behaviour and patterns of attachment.

Biophysical changes in reaction to separation occurred in both the dam and pup, demonstrating the increase/ decrease attachment pattern predicted by Bowlby

(1969), where initial separation led to increased autonomic and behavioural arousal with prolonged separation resulting in reduced levels of activation. Studies involving voles, conducted by Instel (1992, 1997) suggest that mammalian maternal bonding behaviours such as nursing, grooming, licking and touch, which are components of mother-infant proximity, may be related to activation of a specific neurobiological system in which oxytocin plays a central role in maternal affiliative behaviours.
In animal studies investigating the female response to stress Taylor et al (2000) found that *flight or fight* was the primary physiological response to stress in both males and females. Even so females were found to demonstrate a more marked pattern of *'tend and befriend'* compared with males. This *tend and befriend* response pattern is proposed to involve behaviours that are shaped by nurturant activities, designed to *'protect self and offspring, promote safety and reduce stress'*, and are suggested, in part, to rest upon oxytocin (Taylor et al 2000).

A number of human studies provide more direct evidence for the mechanisms involved in maternal-infant attachment. In particular, it is accepted that both primate and human females demonstrate an almost exclusive left handed cradling preference when holding infants (e.g. Borgen et al 1984; Sieratzki and Woll 2002). The weight of evidence suggests that left sided cradling is linked to right hemisphere dominance in a range of bio-behavioural mechanisms that underpin social attachment (e.g. Borgen et al 1984; Sieratzki and Woll 2002; Reisland 2000). Findings for the function of left sided cradling/ right hemisphere processing suggest that the left visual field confers an evolutionary advantage by promoting maternal sensitivity and response to infant cries (Best 1994) and for monitoring infant facial cues for distress (Sieratzki and Woll 2002). Maternal vocal pitch has also been shown to be lower during left sided

cradling, compared with cradling on the right and is suggested to promote infant soothing (Reisland 2000) although findings for vocal significance are conflicting (Lucas et al 1993; Todd and Butterworth 1998). Ochsner et al (2002), investigating differences between males and females in response to emotional stimuli, found that female subjects, compared with males, responded more reliably and strongly to emotional stimuli. Such characteristics are proposed to be valuable in nurturing (Ochsner et al 2002).

A number of studies have investigated the neurobiology of human maternal attachment behaviours, demonstrating the activation of complex neurocircuitry. Nitschke et al (2004) found bilateral activation of the orbito-frontal cortex was associated with positive affect when maternal subjects viewed pictures of their own infants as opposed to unfamiliar infants and adults. More recently, Noriuchi et al (2008) investigated the functional neuroanatomy of *maternal love* as a factor of maternal-infant attachment. Functional Magnetic Resonance Imaging (fMRI) demonstrated '*highly elaborate neural mechanisms mediating maternal love and complex maternal behaviours for vigilant protectiveness'* when mothers observed video clips of their infants smiling and crying (Noriuchi et al 2008). Similarly, Swain et al (2007) found that the infant stimulus activated regions of the basal forebrain in parents, which is responsible for specific nurturing responses such as emotion, motivation, empathy and attention. Swain et al (2007) acknowledged that these were essential components of effective parenting and argues that an integrated understanding of the neurobiology of parenting has profound implications for mental health.

4.2.ii Early parental preoccupations and behaviours

Winnicott (1956) was amongst the first to observe that human mothers exhibited an obsessive-like involvement with their infants and expressed thoughts that were characterised by anxiety and mental exclusivity. This unique mental repertoire, collectively termed '*primary maternal preoccupations,* 'was proposed to achieve close physical and psychological proximity between mother and infant during bonding (Winnicott 1956). *Significantly, Winnicott (1956) maintained that at any other time of life such levels of obsessiveness would indicate a mental disorder, whilst during bonding it is not only typical but critical to the formation of maternal attachment.*

In a study of 41 parent pairs Leckman et al (1999) investigated early parental preoccupations and behaviours (EPPB). The central hypothesis being tested was that these would peak close to the birth of a new infant and included the *a priori* hypothesis that anxious intrusive thoughts with harm avoidant behaviours (AITHAB) would resemble features of OCD. Using semi-structured interviews and self report measures parents were interviewed separately in the home at 8 months antenatal, 2 weeks and 3 months postpartum. Data were gathered using a battery of measures that were described to possess *adequate psychometric and conceptual properties* (Leckman 1999):

As predicted by Leckman et al (1999) parents reported a high level of pre-occupation with infants that reached a peak 2 weeks after the birth with the maternal frequency of infant focused thoughts (approximately 14 hrs per day) uniformly higher than for fathers. Parents of firstborns reported being preoccupied between 1.4 and 2.4 hrs longer per day than parents with more than one child. The content domains of parental thoughts and actions were examined with a particular focus on AITAB.

Leckman et al (1999) found that at 2 weeks and three months postpartum virtually all parents (>95%) reported recurrent thoughts and worries about infant health and development, appearance, vulnerability, physical safety and anxiety about something bad happening to the baby. Outcomes demonstrated the stable content of EPPB, with ratings for mothers, in all but worries over maternal health, consistently higher than for fathers.

Leckman (1999) established that parents frequently expressed concerns about infant harm arising from parental negligence, pets and abhorrent thoughts of themselves or others harming the infant. A minority of parents confirmed such thoughts to be a definite source of interference that could cause moderate to severe distress. In addition a number of parents reported that they performed various actions in response to anxious or unpleasant thoughts, including checking, talking to others and making an effort to distract themselves. Similar findings were replicated by Feldman et al (1999), in a study investigating mother-infant proximity. Virtually all parents (>90%) reported checking on the baby usually in response to infant cues with a majority reporting that they checked even though they knew everything was OK. A number reported compulsive checking either *frequently* or *very frequently*, despite some acknowledging such checking to be *unnecessary* or *silly*. A majority of parents (83% of mothers) stated that they would be distressed if they were prevented from checking on the child with a proportion stating that they would feel guilty (59% of mothers) or panicky (37% of mothers) if they slept through the night.

None of the parents involved in Leckman's et al (1999) study were diagnosed with OCD. Even so 95% reported the presence of at least one repetitive thought or behaviour unrelated to the care of the infant during the course of the study, such as

cleanliness and repetitive washing (79%) and *arranging* behaviours (79%) although these were not generally associated with parental distress. Leckman et al (1999) proposed that anxious feelings experienced by parents provide *'potent stimuli that facilitate the establishment of selective bonds'* as well as prompting *'immediate care giving behaviour'* whilst close physical proximity diminishing parental anxiety. Importantly, outcomes confirmed the discriminant validity of EPPB for mothers and fathers, separate to perinatal mood states (depression, state and trait anxiety), level of childcare responsibilities, direct physical contact and time spent away from the infant, at 2 weeks and 3 months postpartum.

In summary, despite questions over the generalisability of study findings Leckman et al (1999) established that normative parental obsessions, characteristic of EPPB, are frequent and intense, '*almost like an illness'* as Winnicott (1956) described.

4.2.iii Mother-infant synchrony

Parental sensitivity and responsiveness to infant cues influences the ability of infants to tolerate states of heightened arousal, which promotes early infant self-regulation and later adjustment (Rogman 1991; Jaffe et al 2001, Isabella and Belsky 2001). *Normal* mother-infant interactions are typically characterised by reciprocal, synchronous or coherent, affect and behaviours (Tronick 1989). According to Feldman (2007) synchrony emerges in the '*temporal relationship between parent – infant interactions and relatedness', which in its optimum formation 'involves a parent-infant match in affective states, behaviour and biological rhythms as well as a matching in the direction of change, to form a single relational unit'. Parent-infant synchrony therefore provides the foundation for the child's later capacity for intimacy, symbol use, empathy and the ability to read the intentions of others*' Feldman (2007).

Synchrony originates in the repertoire of maternal postpartum bonding behaviours, which include close infant proximity, gaze at the infants face, facial expressions and affective displays, high pitched vocalisations (motherese) and affectionate touch that is akin to licking and grooming in mammals and shape the life-long stress reactivity of offspring and the cross generational transmission of parenting (Champagne et al 2001; Feldman 2007). As such maternal bonding behaviours form the *building blocks of a regulatory process* in the first months of life that play a formative role in the organisation of infant neurobiological systems continuous with social, emotional and self-regulatory skills in childhood and adolescence (Feldman 2007).

4.2.iv The transmission of maternal psychiatric disturbance

There is considerable evidence that maternal psychiatric disorder such as depression, general anxiety disorder and eating disorders adversely affect the health and development of offspring (Yarrow et al 1984; Field 1984, 1988, 1995; Radke-Yarrow 1985; Weissman 1986; Troncik 1989; Dunham and Dunham 1990; Fonagy 1991; Cohn and Campbell 1992; Murray et al 1992; Beardslee et al 1993, 1998; Cicchetti 1998; Brennan et al 2000; Carter et al 2001; Elgar et al 2004; Stanley et al 2004; Stein 2009).

Maternal depression

Depression has received the most empirical attention regarding mother-related risk for infant outcome (Feldman 2007). The severity and chronicity of maternal depressive symptoms, especially when persisting through the first 6 months postpartum is generally associated with greater impairment of functioning, less emotional availability and poor quality of maternal care (Belsky and Vondra1989) compared to those with episodic depression only (Teti et al 1995). Depressed

mothers tend to show more flat or negative facial expressions (Field et al 1985; Raage et al 1997), fewer expressions of interest (Cohn et al 1986; Flemming et al 1988; Murray et al 1993), less accurate matching of happy facial expressions to happy vocal expression (Murray and Cooper 1997; Field 2002; Lundy et al 1996) and demonstrate more hostility and intrusive behaviours and lower responsivity to infant stress or social signals during interactions (Field et al 1992; Lovejoy et al 2000). Depressed mothers are also suggested to provide less affectionate touch (Feldman et al 2004, 2003) with poor levels of stimulation or appropriate social response during interactions (e.g. Murray et al 1997). Depression also adversely influences maternal attachment representations of the infant (Hart et al 1999; Feldman et al 1999).

Maternal anxiety

There has been less investigation into the relationship between clinical levels of maternal anxiety and adverse infant outcomes compared with depression. However, anxious mothers have been shown to exhibit *shorter and quick paced interactive sequences* with their infants (Feldman 2007). The interactive style of mothers with anxiety is not sensitive to the micro-signals of infants and tends to inhibit moments of quiet, neutral affect or gaze aversion and infant *'self-refuelling* prior to the *next sequence of shared relatedness'* (Feldman 2007). In addition anxious mothers are likely to report more fear and worries regarding infant safety and growth and are more preoccupied by an attention to environmental threats that leads to over protectiveness and potentially intrusive parenting (Stein 2009; Bringen 1990; Feldman et al 1997). Feldman et al (1999) found that clinical anxiety in mothers impairs relationship building behaviours and interferes with the balance between fear and hedonic components in maternal representations. Although it is suggested that anxious mothers are not dissimilar to non-disordered mothers regarding the amount of

motherese and displays of positive affect they are more likely to maintain a highly stimulating style, matching less frequently the infant's state and signals (Feldman et al 2005). For example anxious mothers have been shown to maintain high pitched sing-song vocalisations regardless of whether the infant was socially responsive, gaze averting or showing signs of fatigue (Feldman et al 2005).

Preoccupation in psychiatric disorder

Evidence for the relationship between the interactive style of mothers with psychiatric disorder and disrupted maternal-infant synchrony emerge from observational studies. Stein (2009) suggests that maternal cognition, in the form of preoccupations, is likely to be a key mechanism in the transmission of maternal psychiatric disorder. In contrast to the normative EPPB described by Leckman (1999), preoccupations that are associated with a broad range of prevalent psychiatric disorders (Harvey 2004) such as depression (Noelen-Hoeksema 2000), general anxiety disorder (Borkvec et al 1983) and eating disorders (Troop 1997) are dominated by recurrent, negative, intrusive thoughts that are difficult to control. Specifically, preoccupation in depression is strongly associated with a narrowed internal or self-focused attention (Ingram 1990; Mor 2000). In general anxiety disorder the attentional bias can be internal, scanning the body for threat (Ehlers 1995) or external, scanning for environmental threat (Williams 1997). Preoccupations in eating disorders tend to be associated with eating, weight and body shape (Cooper et al 1993; Rosen 1995).

Preoccupations in psychiatric disorders are represented by a self perpetuating, narrowed or self-focused attention (Stein 2009), which is suggested to impair the processing of environmental stimuli, consume attentional capacity and adversely impacts the speed of performance and performance of a secondary task including

social interactions and problem solving (Teti and Gelfand 1997). Thus maternal preoccupation associated with mental illness is proposed to act as a mechanism in the transmission of psychiatric disturbance by reducing maternal responsiveness and sensitivity (Papousek 1987) that impacts infant learning and attention, joint attention in mother-infant interactions and the infant's capacity for emotional self-regulation (Stein 2009).

4.3 Section summary: Maternal-infant attachment

Notably, there is a lack of any *coherent model* for mother-infant attachment (Taylor et al 2000), which is characterised by both bio-behavioural *and* mental components, with the latter unique to humans (Leckman et al 1999; Feldman et al 1999; van IJzendoorn and Tavecchio 1987). These components of mother-infant attachment are normative in function. They promote close parent-infant proximity (Leckman et al 1999; Bowlbly 1973; Ainsworth et al 1978) and optimum synchrony in the mother-infant relationship which contributes to optimum emotional and cognitive development in offspring (Feldman 2007). In turn, maternal preoccupations associated with psychiatric illness induce low maternal sensitivity and responsiveness to infant cues and results in disrupted mother-infant synchrony, which adversely impacts infant health and development. Importantly, understanding of the neurobiology that underpins the mental domain of maternal attachment is seen to harbour profound implications for mental health in humans (Swain et al 2007).

The following section continues this line of reasoning by drawing upon evolutionary hypotheses for the utility of symptoms of depression and anxiety.

Evolutionary hypotheses for the utility of depression and anxiety

4.4 Proximate and evolutionary explanations for illness: two halves of a whole

Nesse (1999) advocates the need to consider both the proximate (What?) and evolutionary (Why?) explanations for illness in order to define the boundary between pathology and what is *normal*. Proximate (medical) explanations of disorder describe the pathological process of illness primarily as a means of developing appropriate treatments; evolutionary explanations are complimentary and account for *why* humans exhibit some apparently dysfunctional traits, *why* they are regulated as they are and *why* we are susceptible (Nesse 1999).

4.4.i The utility of a trait

Darwinian Theory (Darwin, 1872, Chapter 5) proposes that traits become common through the promotion of characteristics advantageous to existence i.e. evolutionary adaptation. Whether common disorders such as anxiety and depression are adaptive and are therefore defences, *dysregulated* defences or defects remains the focus of ongoing debate (Nesse 2000). Nesse (1989) elucidates that the utility of *emotions* such as depression and anxiety are best viewed as '*...co-ordinated systems of physiology, cognition and behaviour... the function of which only makes sense with consideration of the specific characteristics of that situation by which they are aroused,...increasing Darwinian fitness'.*

Defences, Nesse (2000) clarifies, are aversive states such as nausea, fever, pain and fatigue, regulated by cues associated with situations in which they are useful; *defects*, for example, epilepsy and hypo-anxiety, are not. Defences may appear pathological because they are readily activated, therefore seeming over defensive and

dysfunctional; typically a *false alarm* is *less costly* than the potential harm associated with a failure to respond in unpropitious circumstances (Nesse 1999). In contrast a dysregulated or maladaptive defence is said to represent *chronic over activation or inappropriate evocation of mechanisms (in the affect system)* because we now live in conditions very different to the environment in which they evolved (Bowlby 1969; Irons 1998; Gilbert 2006).

In view of the global prevalence of sub-threshold and threshold categories of depression (for example, Kendler et al 1999) and anxiety (Marks and Nesse 1994) the potential of depression and anxiety as defences are worthy of consideration.

4.4.ii Low mood and sub-types of depression: features and functions

'Depression is well recognised to be rooted in the down regulation of positive affect systems' (Gilbert 2006). Opinions on whether or not depression meets the criteria for a defence range from its absolute pathology (Nettle 2004), to its status as a dysregulated defence (Gilbert and Alan 1998) to its functional utility as an adaptation shaped by selection (Price 1994). Mid-ground, Wakefield (1997) concedes that it is both the severity and context of low mood that determines whether depression is *normative* or *pathological.*

A detailed consideration of the psychological models and rationales for the evolutionary utility of depression is beyond the scope of this thesis (for example Gilbert 2006). The emphasis therefore, is not upon the function of *clinical depression* but how its various characteristics, said to be common within the range of normal experience (Nesse 1999), can be argued to confer a selective advantage (Nesse 2000; Gilbert 2006): Succinctly, extreme low mood is global (Hill and Martin 1997;

Weissman et al 1996) with peak prevalence occurring during childbearing years which is an *unprecedented* feature of most other diseases (Kessler 1993; Nesse 2000). Reanalysis of Brown and Harries' (1978) data suggests that severe enough situations can cause depression in almost everyone (Monroe and Simons 1991).

Importantly, Coryell et al (1994) found that patterns of low mood are stable across situations rather than intrapersonal. Specific feature of depression are *precipitated by cues associated with specific fitness-losses* and subside when the situation resolves (Coryell et al 1994). Such characteristics are suggested to represent those that distinguish defects from designs (Nesse 1989).

Keller and Nesse (2005) propose that that the following features or sub-types of low mood have evolved in response to different environmental cues that might signal the risk of loss;

- *Sadness* can be triggered by actually or imagined *fitness losses* and is suggested to motivate future avoidance of situations that might incur loss.

- *Crying* is proposed to signal the need for help (Lewis et al 1934), solicit empathy and comforting behaviours from others (Labott et al 1991; Cornelius and Labott 1997) and strengthen social bonds (Frijda 1986). Crying is therefore anticipated to increase in response to situations involving social losses or a lack of social support (Keller and Nesse 2005).

- *Self –reproach,* characterised by feelings of worthlessness and guilt (self-blame) is proposed to represent the mental search for understanding; it signals

culpability to others, thus avoiding loss of social bonds and motivating avoidance of similar, future actions (Keller and Nesse 2005)

- *Fatigue* motivates energy conservation through reduced exertion and goal pursuit in unpropitious situations and signals depletion of resources.

- *Pessimism* is the tendency to think negatively about the success of future outcomes and diminishes goal pursuit through motivating disengagement when the likelihood of success is perceived to be low (Klinger 1975).

- *Appetite* is affected by unpropitious circumstances. Low mood may manifest in decreased appetite resulting in a temporary reduction in foraging where situations of uncertainty might incur risk. Increased appetite might be adaptive in the face of diminishing food reserves (Keller and Nesse 2005).

- *Changes in sleep* typically manifest as nocturnal wakefulness is proposed as a useful safeguard against adversaries in the *'adaptively relevant environment'* (Irons 1998).

4.5 Anxiety and sub-types: features and functions

'Nearly everyone recognises that anxiety is a useful trait that has been shaped by natural selection' (Marks and Nesse 1994).

Anxiety and panic as conserved responses can both be defined to incur and unpleasant feeling associated with increased physiological arousal in the absence of any objective threat (Marks 1987). The utility of fear and anxiety is well recognised with the aversiveness of these involved in hypervigilance and harm avoidance (Marks

1987). Similar to evolutionary hypotheses for depression, anxiety sub-types are hypothesised to have originated in response to specific cues for threat. Anxiety therefore increases fitness in situations that threaten reproductive resources which include life, health and interpersonal living (for example, Marks and Nesse 1994; Nesse 1999).

Normal anxiety, as an evolved response, is similar to low mood in that it is *low cost* and prone to false alarms. This is because failing to respond to a perceived threat may result in significant loss (Marks and Nesse 1994). Natural selection is therefore proposed to have evolved a nervous system in humans that predisposes us to (1) *prepotency* or *salience,* that is an innate attention to specific cues (Marks 1969; Ohman and Dimberg 1984) and (2) *preparedness* (Seligman 1970), signifying specific patterns of reaction to specific cues.

Prepotency underpins *prepared* reactions to particular patterns of stimulation, leading to an *organised* distribution of fear; general anxiety, inducing vigilance and physiological arousal, is considered probably to have evolved in response to ill defined threats (Marks and Ness 1994); proximity seeking in infants is triggered by separation anxiety (Bowlby 1973; Marks 1987); threat from predators provokes flight, fight, or freeze (Cannon 1929); high places evoke freezing whilst fear communicates danger (Darwin 1872). It is important from the non-pathological perspective of this study, regarding the relationship between childbearing women, mood and anxiety, that females are recognised to experience heightened levels of *prepotency* and *preparedness*, compared with males (Craske 2003, pg 192-199, 201-203, 210).

4.6 Evolutionary theories for postnatal depression

To date there has been little consideration of the potential for symptoms of depression and anxiety to represent adaptive mechanisms in relation to childbearing, shaped by fitness needs in ancestral environments. In a comprehensive analysis Hagen (1999) explored the potential evolutionary purpose of postnatal depression as a response to adverse circumstances. Hagen (1999) addressed three related, adaptive functions for postpartum depression that are consistent with the evolutionary *defection* hypothesis, based upon the assumption that postnatal depression is a mechanism for maternal withdrawal from reproductive investment:

- ***Defection Hypothesis Part 1*** (Hagen 1999) examines the association between *negative affect*, that is *sad* or *depressed mood,* and circumstances that were reproductively costly in ancestral environments with correlates of net fitness costs. The proven association between negative affect and, for example, poor environmental factors and low infant viability leads Hagen (1999) to conclude that strong evidence exists to support that circumstances associated with *increased fitness costs in our ancestral environment are etiological factors for negative affect in modern mothers.*

- ***Defection Hypothesis Part 2*** (Hagen 1999) anticipates that *loss of interest* is associated with reduced maternal investment in the infant, partner, other children or non-infant related responsibilities. Hagen (1999) concludes that *loss of interest* predicts the defection hypothesis for postnatal depression. Further maternal *loss of interest* is considered to extend the defection hypothesis to renegotiation or defection from others in attempts to increase investment by others or reduced the social costs of defecting from childrearing.

- ***Defection Hypothesis Part 3*** (Hagen 1999) proposes that postpartum depression amounts to a *negotiation* strategy based upon evolutionary models of reciprocal co-operation (Axelrod and Hamilton 1981). In a later study investigating postnatal depression as *bargaining*, Hagen (2002) suggested that the outcomes provided good support for this hypothesis but acknowledged that they were not conclusive with further longitudinal studies required.

Overall, Hagen (1999) proposed that evidence for the defection hypothesis, although not proven, suggests that *postnatal depression* is an adaptation (1) shaped to motivate a reduction in or withdrawal from maternal postpartum investment and (2) represents resistance to external control over female reproductive decision making. Importantly, Hagen (1999) utilised reports for the incidence of postnatal depression, based upon *detection (*Cox et al 1993; Campbell and Cohen 1991; O'Hara 1984*)* although did acknowledge it to be indistinguishable from depression at other times of life (Hagen 1999).

4.7 Section summary: Evolutionary hypotheses for the utility of depression and anxiety

Evolutionary explanations for disorder consider what advantage might be conferred and needs to be understood alongside the proximate, medical explanations as a means of identifying the boundary between health and illness (Nesse 1999). Depression in particular is unprecedented amongst disorders in humans as it reaches peak prevalence in the reproductive years, a hallmark of a defence (Keller 1993: Nesse 2000). In turn, normative anxiety is generally recognised to be protective, with females demonstrating a heightened predisposition to *prepotency* (innate attention to specific cues) and *preparedness* (specific reactions to specific cues) compared with

males (Craske 2003). Evolutionary theory proposes that sub-sets or symptoms of depression and anxiety confer a fitness benefit when cued by circumstances that threaten reproductive success.

The limited investigation into the evolutionary purpose of *postnatal depression* suggests that it is a mechanism for defection in adverse or unpropitious circumstances although it is recognised to be no different to depression at other times of life (Hagen 1999).

4.8 Chapter summary

Evidence for maternal attachment and hypotheses for the utility of depression and anxiety are relevant to understanding normative postpartum mood. Currently there is lack of a *coherent model* for mother-infant attachment (Taylor et al 2000) with attachment generally considered from the infant's perspective only (Feldman 1999). Even so maternal bonding behaviours are observed in all mammals (e.g. Hofer 1995; Instel 1997; Borgen et al 1984; Sieratzki and Woll 2002; Ochsner et al 2002; Nitschke et al 2004) whilst human parents exhibit obsessive preoccupations and behaviours (EPPB) (Leckman et al 1999) in relation to newborns, with the formation of a selective bond (Feldman 1999). The purpose of these conserved physical and mental mechanisms is to establish emotional bonds and close proximity with the infant which ultimately promote evolutionary survival. Far from being pathological, it is suggested that in the context of the sensitive postpartum period primary maternal preoccupations and bonding behaviours are *normative* even if reaching conventional thresholds for disorder (Leckman et al 1999). In contrast preoccupations that are associated with a range of psychiatric disorders adversely impacts the health and

development of offspring by reducing maternal sensitivity and responsiveness and disrupting synchrony in the mother-infant relationship.

Evolutionary hypotheses for the sheer prevalence of depression and anxiety during childbearing years (Kessler 1993; Nesse 2000) and their manifestation in response to specific fitness-losses (Zimmerman and Coryell 1994; Keller and Nesse 2005) suggest that they are hallmarks of a defence (Marks and Nesse 1994; Nesse 2000; Gilbert 2006). Importantly, *prepotency* (innate attention to specific cues) and *preparedness* (specific patterns of reaction to specific cues), features associated with *normative* anxiety, are heightened in females compared with males and confer a selective advantage (Marks and Nesse 1994).

The adaptive purpose of postpartum features of depression and anxiety has been considered only from the perspective of clinical disorder. The evolutionary theory for *postnatal depression* suggests that it is a mechanism for maternal defection from infant investment or resistance to external control over female reproduction (Hagen 1999). Whist it is acknowledged that no difference exists between postnatal depression and depression at other times of life Hagen's (1999) evolutionary hypotheses for postnatal depression overall suggest that it is a conserved response to childbearing that is concerned with maternal survival.

4.9 Conclusion

In humans the context of specific features of subjective depression and anxiety that are cued to promote evolutionary survival represent periods of vulnerability. Parental cognitions and behaviours (EPPB), which are neurobiological in origin and characteristic in healthy mother-infant attachment, resemble features of psychiatric

illness that are cued to promote optimum infant development and ultimately survival. Descriminant features of *normative* rather than psychiatric pre-occupation in mothers emerge in an attentional bias that is infant focused and associated with anxious intrusive thoughts and harm avoidant behaviours (AITHAB). Maternal pre-occupation associated with psychiatric disorder are intrusive and persistent. These pathological preoccupations inhibit maternal sensitivity to the infant by focusing the mother's attention on internal or environmental stimuli in a manner that distracts attention away from the infant. However, maternal personality disorder and attachment style have been shown to impose the same adverse influence upon the health and development of offspring that is typically associated with psychiatric disorder. In the absence of enduring psychopathology it is possible that EPPB and AITHAB underpin the transient and elevated symptoms of depression and anxiety that are more typically regarded as *postnatal depression*.

Evolutionary hypotheses for the function of *postnatal depression* are unconvincing as amongst other things, the context of the discussion is contemporary rather than evolutionary. More telling is the higher frequency of symptoms of depression in the reproductive years, compared with other times of life, a feature that points to their potential utility as defences. Examples of sub-types of depression typically associated with the postpartum experience are *guilt* or *self-reproach* (*blame*), *loss of interest in the external world* and *tearfullness*. Their utility in the vulnerable postpartum period, based upon an evolutionary rationale, lies in the motivation of harm avoidant behaviours (guilt/ blame), the inhibition of investment away from the infant (transient loss of interest in external events) and the signalling of the need for additional support and resources (crying). Anxiety is typically a *subjective* appraisal of threat, which

induces hyper-vigilance and harm avoidance. As a consequence such symptoms are phenotypical features of childbearing and should therefore be anticipated.

Overall, the association between the early attachment experience and later psychopathology provides leverage for the need to develop a cohesive model of *normative postpartum mood*.

STUDY RATIONALE

The rationale for this study is founded upon the reviews undertaken in Chapters 2, 3 and 4. Examination of the relevant literature confirms that there has been very little investigation into the mental domain of women after childbirth. Consequently, there is no cohesive model of *normative* postpartum mood. Conversely, postnatal mental health is defined by *postnatal depression*, a medical construct, although it is not recognised by any classification system. As a consequence the lens through which postpartum mood is observed and understood is pathological, with childbearing considered to pose an inherent risk for the *onset* of depression in women (Pitt 1968; Cox 1987; Cox 1993; Gavin et al 2005). Throughout its short history postnatal depression has been defined, critically, as '*atypical*' (Pitt 1968), a misnomer that continues to attract intense scientific interest. The development of the EPDS (Cox et al 1987), a tool specifically designed to screen postpartum women, has arguably served to substantiate the clinical status of postnatal depression. However, throughout the scientific literature messages about *what* postnatal depression actually *is* are mixed. This position is illustrated by the following points.

1. Postnatal depression is investigated as a discrete psychiatric disorder although does not meet any classification criterion (DSM-V, APA 1994; ICD-10, WHO 1994). Postnatal depression fails to meet the relevant criteria for a disorder (Wilson and Jungner 1968; UK NSC 2010).

2. No difference has been evidenced in depression regarding rates of onset, disease course, chronicity and symptom profile, between childbearing and non-childbearing populations (e.g. Kumar et al 1982; Cooper et al 1988; O'Hara 1990; Whiffen 1992; Whiffen and Gotlib 1993; Eberhard-Gran 2002). The aetiology of depression *at any*

time of life is shown to lay in the interaction between genes and environmental experience, particularly early trauma (Tsigos and Chrousos 2002; Goldberg 2006; Bownds 1999 p 237; Gunnar 2000; Hong and Tsai 2003; Kendler 2006). Stressful life events prior to first onset depression are therefore precipitators rather than causal and childbearing does not emerge either as a specific cause or a factor of high risk. (see Murphy 1988, 2000; Parker 2001).

3. Estimates of *incidence* in postnatal depression are limited and need to be considered with caution. Whilst in general the incidence is reported to be three-fold higher at five weeks postpartum (Gavin 2005) the few studies that include control groups report no significant difference in rates of depression onset between childbearing and non-childbearing groups (Cooper et al 1988, O'Hara 1990; Whiffen 1992; Whiffen and Gotlib 1993; Gelder 2009).

4. The EPDS is validated as a single construct measure of depression (Cox et al 1987) although includes anxiety and depression items with ratings for both contributing to a total depression score (Brouwers et al 2001; Ross et al 2003). The EPDS fails to meet the relevant criteria as a screening tool (UK NSC 2010).

5. In contrast to the interest in postnatal depression less attention has been paid to anxiety postpartum (see Chapter 3, pg. 37- 40, Ross et al (2006)). Whilst perinatal anxiety disorders are common, there is no anxiety equivalent to concept of *postnatal depression*.

Examination of relatively disparate areas of literature regarding depression and anxiety, relevant to the question of women and childbearing, indicates that the position of postnatal depression as a discrete disorder is, at best, tenuous.

The temporal sequence revealed by concomitance studies in which anxiety typically *precedes* depression in females *and* males (Alloy et al 1990: Murphy et al 1888, 2000; Breslau et al 1995; Parker et al 2001) suggests an overemphasis on the prevalence of depression in women which has been distracting. Further, the suggestion that females experience greater propensity to anxiety rather than depression compared with males (Breslau 1995; Murphy 1988; Parker 2001) is likely to represent a fruitful line of investigation both in terms of the aetiology of depression and the significance of anxiety in women and childbearing.

Findings that show co-existing anxiety and depression below the threshold for disorder is more common than single disorders both in community and psychiatric samples (Zinbard et al 1994; Stein et al 1995; Wittchen and Essau 1993; Von Korf et al 1987; Angst et al 1997; Brown et al 1996; Roy-Byrne et al 1994; Zung et al 1990; Helmchem and Linden 2000) support that this should be anticipated for postpartum women.

The female primary propensity to anxiety holds further significance in light of the role played by anxiety in maternal-infant attachment (Leckman et al 1999, Feldman 1999), which promote harm avoidant behaviours and attachment representations of the infant. These ultimately promote optimum safety and development and in offspring. Similarly, evolutionary hypotheses offer a compelling argument for the utility of for parental *guilt (self-blame), also* present *in normative maternal cognitions and harm avoidant behaviours in response to childbearing.* The characteristic presence of these specific symptoms of depression and anxiety suggest their potential as phenotypical features in postnatal women, the purpose of which is to support attachment in the mother/ infant dyad and ultimately survival.

Hypotheses and main study aim

Two hypotheses emerge from evaluation of the wider literature in relation to non-pathological postpartum mood;

- Hypothesis 1: Maternal postnatal mood is characterised by elevated symptoms of depression and anxiety that may or may not reach the threshold for risk of disorder, and remit between 4 and 16 postnatal weeks.
- Hypothesis 2: Maternal postnatal mood is characterised in particular by feelings of anxiety and guilt.

In response, the main aim of this thesis is to investigate how features of depression and anxiety might be organised to suggest a characteristic pattern in postpartum mood, in a population sample of postnatal women using the EPDS, Beck Depression Inventory (Beck 1961) and Beck Anxiety Inventory (Beck 1988).

This aim is addressed through the pursuit of two objectives which are underpinned by corresponding research questions;

Study objective 1 - To measure postpartum symptoms of depression and anxiety at two periods of time in a population sample of postnatal women using the EPDS, BDI and BAI.

Research question 1.1: What are the key features of postpartum mood?

Research question 1.2: How do rates of postpartum symptoms of depression and anxiety change over time?

Study objective 2 - To examine the relationship between rates of anxiety and guilt vis-à-vis other EPDS symptoms.

Research question 2.1: What is the prevalence of anxiety, blame and panic compared with other symptoms according to the EPDS?

Research question 2.2: What contribution do rates of anxiety and guilt make to the overall depression score according to the EPDS?

Chapter 5 to follow provides an overview of the study design, describes the main study methods and the subject profile, and summarises the statistical analysis plan.

CHAPTER 5

METHODS

5.1 Synopsis

Chapter 5 explains the methods used to explore *symptoms* of depression and anxiety and change over time, in a sample of postpartum women. The study design, setting, sample population and measures used, are described. The research questions are outlined and the statistical analyses are summarised. Subsequent chapters 6, 7 and 8 report the results of the study.

5.2 Study aim and objectives

The overall aim of this study is to investigate the frequency and distribution of postpartum symptoms of depression and anxiety and how these may be organised in women after childbirth. The study objectives and key research questions are;

Study objective 1 - To measure postpartum symptoms of depression and anxiety at two period of time in a population sample of postnatal women using the EPDS, BDI and BAI.

Research question 1.1: What are the key features of postpartum mood?

Research question 1.2: How do rates of postpartum symptoms of depression and anxiety change over time?

Study objective 2 - To examine the relationship between rates of anxiety and guilt vis-à-vis other symptoms of depression and anxiety using the EPDS, BDI and BAI.

Research question 2.1: What is the prevalence of anxiety, blame and panic compared with other symptoms according to the EPDS?

Research question 2.2: What contribution do rates of anxiety and guilt make to the overall depression score according to the EPDS?

The study methods are reported with consideration for the '*Strengthening the Reporting of Observational Studies in Epidemiology*' (STROBE) statement (Vandenbroucke et al 2007). The STROBE criteria (Vandenbroucke et al 2007) provides guidance to authors on how to improve the reporting of observational studies; the STROBE guidelines (Vandenbroucke et al 2007) make recommendations for best practice in writing the title, abstract, introduction, methods, results and discussion sections.

5.3 Design Overview

A field study was conducted using a pragmatic sample of eligible postnatal women, recruited from the caseloads of generic health visitors employed by Cardiff and Vale NHS Trust, Rhondda Community NHS Trust and North Glamorgan NHS Trust, in Mid and South Glamorgan. Consenting postnatal subjects completed the Edinburgh Postnatal Depression Scale, the Beck Depression Inventory and the Beck Anxiety Inventory to assess the presence of *symptoms* of depression and anxiety on two occasions, between 4 - 8 and 12 - 16 postnatal weeks. Total scores for the measures used were investigated to provide point and period rates, below and above validated cut-off scores for risk of disorder. Data were not collected and/or submitted for those subjects who refused to participate, withdrew, or met the exclusion criterion.

5.4 Study setting

The study was conducted in South Wales, within the counties of South and Mid Glamorgan, yielding 3 geographical cohorts. Health visiting provision was met by Cardiff and Vale NHS Trust (Trust 1), Rhondda Community NHS Trust (Trust 2) and North Glamorgan NHS Trust (Trust 3). In some cases geographical boundaries for health visiting overlapped both NHS Trust and Local Authority boundaries. At the time of data collection Trust 1 provided health visiting services to Cardiff and the Vale of Glamorgan; Trust 2 provided health visiting services to Rhondda and Taff Ely although the Local Authority boundary incorporated the Cynon Valley, identifying its remit as *Rhondda Cynon Taff*. Health care provision for the *Cynon Valley* was serviced by Trust 3, identifying its geographical service remit as *Merthyr and Cynon*.

It is an important consideration that there was no difference between areas in the provision of generic health visiting services to postnatal women which could influence study outcomes. In particular there was no systematic screening program for postnatal depression or routine provision for psychological intervention by health visitors such as the six *listening visits* proposed by Holden et al (1989).

5.5 Participants

Research participants were generic health visitors and postnatal women from populations within the service remit of three NHS Trusts in South and Mid-Glamorgan.

5.5.i Health visitors

Generic, case holding health visitors employed by the 3 collaborating NHS Trusts were eligible to recruit and screen postnatal women from their caseloads.

Health visitors are qualified nurses and/ or midwives whose professional remit is public health, registered as *Specialist Community Public Health Nurse* (Nursing and Midwifery Council 2004). Health visitors were ideally positioned to participate in this study as their sphere of practice engages them with postnatal women and because they are the primary professional discipline charged with screening women for postnatal depression.

5.5.ii Postnatal women

The sample population was postnatal women within the first 16 weeks following childbirth, recruited at the primary birth visit.

Postnatal women - inclusion criterion

The inclusion criterion identified *eligible* postnatal women as:

- those who had delivered a live, healthy infant at full term (i.e. 37 – 42 weeks gestation) between March 2003 and May 2004,
- were recruited from the caseload populations of participating, generic health visitors and
- provided written, informed consent.

Postnatal women - exclusion criterion

Postnatal women were excluded from the study if they

- were below the age of legal consent, i.e. 16 years of age,
- had a current experience of stillbirth, neonatal or infant death including cot death,
- delivered a premature infant (i.e. before 37 weeks gestation) or

- infant in need of specialist care.

Women diagnosed with a major mental illness such as bi-polar disorder, psychotic depression or schizophrenia, were also excluded.

5.5.iii Recruiting and screening among ethnic minority mothers

Participating health visitors with high numbers of ethnic minority mothers in Trust 1 reported a range of difficulties that had not been anticipated prior to inception of the study. Notably, eligible ethnic minority mothers were reported to refuse to participate or withdraw more frequently than their white, English speaking counterparts. Reasons that were suggested by health visitors included the reluctance of other family members, language difficulties, levels of literacy, cultural influences and concepts of health. In addition, the need to use interpreters imposed increased financial and workload demands, with both proving prohibitive.

Importantly, relevant health visitors expressed concern that efforts to gather data from ethnic minority groups could adversely impact the potentially fragile relationships between them. Furthermore these health visitors questioned the validity of the measures used in that they represent western concepts of psychological health that might not apply to ethnic minority groups. This was somewhat borne out by interpreters who reported difficulties in accurately translating scale items. The problem extended even to the EPDS which has been translated into a number of languages and validated accordingly. As a result health visitors either opted not to recruit women from ethnic minorities or withdrew from the study as a whole.

Following discussion with the main study supervisor it was deemed ethically appropriate to exclude ethnic minority clients. This decision applied to the Cardiff cohort only as the ethnic minority population in Trust 1 was not matched by Trusts 2 and 3.

5.6 Study Sample

A pragmatic sample of convenience was recruited by generic health visitors based upon birth sequence and consent to participate. In order to examine the research questions a sample of 1,275 postnatal women was estimated. The sample size was based on estimating a 95% CI for prevalence of postnatal depression within 2 percentage points of a predicted prevalence of 15% in the sample population. This figure represented approximately 10% of the annual birth rate for the collective postnatal cohort for the year 2001- 2002, preceding the study period.

The total sample size together with the annual birth rate and number of participating health visitors in each Trust determined the number of postnatal women individual health visitors were required to recruit.

5.7 Bias

A number of factors were acknowledged to potentially increase *bias*, that is, the systematic deviation of the study statistics from the population value (Vandenbrouke et al 2007; De Vas 2002 pg 70 & 229). The risk of *selection bias,* which can affect the internal validity of a study (Vandenbrouke et al 2007) was considered to emerge from a range of sources.

In particular, the use of a pragmatic sample of convenience as opposed to a simple random or systematic sampling method was acknowledged to increase the risk of sampling error from the outset. It was also considered that this method of sampling might lead some health visitors to sub-consciously select subjects or influence subject self-report in either direction. However, this risk was deemed low in view of the large number of participating health visitors with each required to recruit only between 5 and 9 postnatal women depending upon Trust/ geographical area. Furthermore, the study protocol required health visitors to approach all postnatal women at the birth visit to ask if they would consent to take part in the study. Health visitors were explicitly discouraged from selecting women who were considered most likely to consent.

Similarly, the sampling method was acknowledged to potentiate *response* bias, a type of selection bias (Vandenbroucke et al 2007). Health visitors were asked to record reasons why some eligible postnatal women refused to participate as these were not observably different to subjects. It was not possible to determine whether these two groups differed significantly particularly regarding risk, past history or current levels of depression and anxiety as clinical interviews were not conducted and medical records were not accessed. Furthermore, the exclusion of ethnic minority women, which was not anticipated at the beginning of the study, suggests that outcomes predominantly reflect Welsh, white, English speaking postnatal women. This reduces the representativeness of the sample and raises questions about the generalisability of study outcomes.

Social data such as educational attainment, marriage status and the number of employed adults in the household were not gathered. This omission represents a significant study limitation and was recognised by the researcher at the outset to introduce a risk of *information bias* (Vandenbroucke et al 2007), a factor that reduces confidence in the representativeness of the sample. Health visitors who participated in the study voiced strong opposition to gathering social data from clients which would not usually be sought in the course of routine health visiting work. An important consideration was that confidentiality could not be guaranteed regardless of the research context. One consequence of this position is that social data gathered by case holding health visitors assumed greater risk for the client as information shared could be subject to later challenge or action. This position contrasts greatly to information shared during limited contact with an *anonymous* researcher. Health visitors expressed concern that clients could feel compromised by a requirement to disclose social data and risked undermining the client/ health visitor relationship.

Overall, the need to gain personal data was weighed against the potential loss of health visitor and subject participation. It was decided to pursue a representative sample by maximising the participation of both groups through limiting personal information to relevant demographic data. A number of additional factors were taken into consideration in support of the decision not to collect social data: Health visiting is universal in Britain meaning that the provision is offered to *all* mothers with new born infants. Although data for the number of perinatal women who refuse health visiting input was not available, anecdotally the numbers are small to the point of being rare. Health visitors therefore have access to virtually all mothers with new born infants. The fact that postnatal subjects were recruited from the caseloads of generic health

visitors suggests that participants were without any special needs and representative of the Caucasian postnatal population.

There was a general consensus amongst health visitors that even in the course of routine work only tentative reliance can be placed upon the accuracy of clients' self-reported, social information. Clients have no mandate to share personal information. Anecdotally, in health visiting practice, clients are discerning in their disclosures with accounts of their circumstances often evolving, not least because of the surveillance role of the health visitor. In efforts to meet the study requirements Welsh Assembly Government statistics for 2003-2004 were accessed (WAG 2005) to provide on overview of social data relevant to the three geographical cohorts.
The influence of selection and information bias was acknowledged to increase the risk of sampling error from the outset. In the main the factors involved were addressed through the pursuit of a large sample size.

5.8 Funding

Preceding the study it was identified that financial support was required to pay for research materials, travel expenses and ring fenced study time for the research manager. Funding was secured from Cardiff and Vale NHS Trust Research and Development (R+D) Small Grant Scheme; following submission of the application, which included the research proposal and costing, the sum of £6,000 was awarded in April 2002.

5.9 Administrative conduct of the study

The author of this thesis was the sole study manager. The author was responsible for (a) applying for and achieving Local Research Ethical Committee (LREC) and Trust *in-house* ethical approval, (b) securing agreement for Trust collaboration and ongoing liaison at managerial level, (c) briefing and instructing health visitors regarding the study protocol and use of measurement tools, (d) recruiting health visitor participants, (e) distributing research materials, (f) coding data, (g) supporting and monitoring the data collection process across all geographical areas, (h) meeting and communicating with health visitor managers and health visitors to ensure compliance with the study protocol, (i) personally collecting completed data sets from external Trust sites and (j) general administrative responsibilities.

The Division of General Practice, College of Medicine, Cardiff University provided clerical assistance to organise study materials and for data entry. All subject data were anonymous at the point of data entry and housed in a secure store room.

5.10 Ethical approval

To ensure research integrity and professionalism and above all, protection of the public, the study was subject to several stages of ethical approval and agreement, both formal and informal.

5.10.i Provisional Trust agreement

Once funding for the study had been secured directorate managers with responsibility for health visiting in the three NHS Trusts were approached by the study manager. A provisional, conditional agreement was obtained for their collaboration in the study with the recruitment and screening of eligible postnatal women by generic health

visitors. This conditional agreement was secured prior to seeking ethical approval as participating trusts needed to be identified for the relevant application. Any initial agreement made by individual Trusts to participate in the study would be subject to further scrutiny by their Research and Development Ethics Committee's (R&DEC) once full ethical approval had been secured.

5.10.ii Local Research Ethics Committee approval

In 2002, Area Health Authorities (AHA) had jurisdiction over research undertaken within their geographical service remit. Under each AHA a Local Research Ethics Committee (LREC) existed to scrutinise and approve research studies prior to their inception. For this study, an initial application for ethical approval was made to Bro Taff AHA LREC, which was granted in July 2002.

5.10.iii Trust Research and Development Ethics Committee approval

LREC approval occasioned formal submission of the research proposal to the R&DEC of Trusts 2 and 3, undertaken between August and October 2002; copies of the study protocol and confirmation of LREC approval were also submitted. Ethical approval at Trust level facilitated direct contact between the study manager and health visitor managers in preparation for the recruitment and data collection process.

Scrutiny and approval of the research rationale and protocol by Cardiff and Vale NHS Trust R&DEC (Trust 1) had been achieved with the application for the small grant scheme for novice researchers.

5.11 Recruitment of health visitors

Following full ethical approval of the study a strategy was formulated to facilitate transition of the study from a position of managerial agreement to the recruitment and screening of postnatal subjects by health visitors in the field. Between September 2002 and February 2003 a total of twelve meetings occurred between the study manager and generic health visitors across the three participating NHS Trusts. These were undertaken to present the study rationale and protocol, and to explain the use of measurement tools and the data collection process.

5.12 Research materials

In advance of the recruitment process, study materials and variables were subject to numeric coding in order to

- identify geographical cohorts,
- coordinate subjects, measurement tools and screening intervals,
- manage the data collection process, and
- ensure confidentiality through anonymity, at the point of data analysis.

Research materials included;

- A copy of the research protocol (see Appendix I)
- Patient information sheet (see Appendix II)
- Consent pro-forma (see Appendix III)
- *Subject demographics* pro-forma (see Appendix IV)
- Edinburgh Postnatal Depression Scale (EPDS) (see Appendix V)
- Beck Depression Inventory (BDI) (see Appendix VI)
- Beck Anxiety Inventory (BAI) (see Appendix VII)

- *Subject log* to assist health visitors in co-ordinating 1st and 2nd screenings for each subject (see Appendix VIII)

Study materials were assembled into individual packs which were identified by a numeric *specifier* for each health visitor and relevant NHS Trusts, i.e. 1, 2 or 3, along with a health visitor name.

Measurement scales were duplicated for the first and second occasions of screening and conveniently arranged as booklets. Each s*creening booklet* was identified with the numeric specifiers (health visitor and Trust). Consenting postnatal women were allocated a number by the recruiting health visitor according to their chronological order of recruitment, i.e. 1, 2, 3, etcetera, with this number recorded along side the health visitor numeric specifier. Health visitors maintained a confidential log (see appendix VIII) recording the subject name and recruitment number together with the date measurement scales were completed in order to co-ordinate the 1st and 2nd episodes of data collection.

All data were named and labelled according to SPSS format.

Relevant managers for each Trust agreed with the study manager appropriate mechanisms to distribute the research packs to health visitors.

5.13 Recruitment and screening of postnatal women

In accordance with the research protocol (see Appendix I), health visitors informed and/or recruited postnatal women about the study during the primary birth visit. Those who expressed an interest or agreed to participate in the study were provided with the *patient information sheet (*see Appendix II*)*. This explained the study

rationale, screening process and, importantly, the client's rights to refuse or withdraw from the study at any time. The patient information sheet also clarified that participation in the study would not effect the core health visiting provision, including regard for appropriate confidentiality. Duplicate consent pro-forma (see Appendix III) were signed by postnatal women who agreed to participate and the recruiting health visitor. One copy was retained by the subject with the second copy filed in the formal family health record. Data were not gathered from postnatal women who refused to participate or did not provide written consent.

Data for postpartum symptoms of depression and anxiety were gathered using the EPDS (see Appendix V), BDI (see Appendix VI), and BAI (see Appendix VII), which were completed by subjects between 4-8 and 12-16 postnatal weeks. These time frames reflected the EPDS screening guidelines that existed in Trust 1 at the time of data collection. Health visitors were present when measures were completed with the intention to provide an opportunity to debrief subjects if necessary and to address matters arising as in routine practice. If required, health visitors were able to access EPDS total scores from the study data providing subjects' had agreed to completing the scale as a matter of routine care. Demographic data were recorded on the relevant pro-forma (see Appendix IV) for subject age, parity and the number of weeks postpartum that measures were completed.

5.14 Study maintenance and support

A strategy to support health visitors in the data collection process was established. Health visitors were encouraged to communicate with the research manager whenever necessary. Relevant contact details (telephone number, work and email

address) were provided with the research protocol. The study manager disseminated information to health visitors on a monthly basis through newsletters (17 in total; for example see Appendix IX) emails and/ or telephone contact, regarding the study progress, as well as any comments, queries and concerns raised by professionals and subjects. In addition to the initial twelve briefing sessions, when requested the study manager met with individuals or small groups of health visitors across three geographical sites.

5.15 Data collection and management

Health visitors were required to return research materials following completion of the 2nd screening. Data sets were returned either through the internal mailing system (Trust 1) or collected by the researcher from designated liaison personnel (Trusts 2 and 3). A '*Head Count*' pro-forma (see Appendix X) was issued to health visitors in June and July 2003 to strategically assess the progress of the study. Data for the number of postnatal women who had refused to participate or had withdrawn from the study were also gathered together along with any reason given. Data collection ceased in August 2004.

5.16 Measurement scales

Quantitative data for postpartum symptoms of depression and anxiety were gathered using three self-rating measures:

5.16.i Edinburgh Postnatal Depression Scale

The 10-item Edinburgh Postnatal Depression Scale (EPDS) (Cox et al 1987) (see Appendix V) is a continuous self report inventory specifically developed to assess

women for *postnatal depression*. Each EPDS item is rated according to 4 statements indicating symptom severity, scored 0 (least), 1, 2, and 3 (most). The total EPDS score ranges from zero to 30 points and is calculated by adding together the scores for each of the 10 items. The original validation study (Cox et al 1987) compared EPDS total scores with psychiatric assessment of subjects using the Clinical Interview Schedule (Goldberg et al 1978). A validated cut-off score of ≥13, identifying at least *risk* of depression, was established using the *diagnostic threshold for depression* according to the Research Diagnostic Criteria (RCD) (Endicott and Spitzer 1978). The EPDS demonstrates good sensitivity (86%) and specificity (78%) with a positive predictive value established at 73%.

Despite its validated single construct status the EPDS comprises two *anxiety* items notably items 4 (*anxiety*) and 5 (*panic*), which contribute to the total EPDS depression score. Depression and anxiety variables have been shown to form sub-scales that have been interpreted to operate as discrete measures of depression and of anxiety (Brouwers et al 2001; Ross et al 2003) (see chapter 6, pg. 118 – 123).

5.16.ii Beck Depression Inventory

The 21-item Beck Depression Inventory (BDI) (Beck 1961) (see Appendix VI) is a continuous, single construct measure of depression, developed '*...to provide a quantitative assessment of the intensity of depression*', for use in adolescent and adult populations and in clinical research (Beck et al 1961). Each BDI item corresponds to behavioural symptoms or attitudes specific to depression, measured according to 4 self-evaluating statements indicating symptom severity, scored 0 (least), 1, 2, and 3 (most). The total BDI score ranges from zero to 63 points and is calculated by adding together the scores for each of the 21 items. A validated cut-off

score of ≥13 indicates *risk* of depression (Beck 1987). The BDI (Beck 1961) demonstrates good internal reliability and validity with a statistically significant relationship between all items and total scale scores (sig. = <0.001); the split half-reliability showed a strong correlation between the odd and even categories (Pearson r = *0.86).*

5.16.iii Beck Anxiety Inventory

The 21-item Beck Anxiety Inventory (BAI) (Beck et al 1988) (see Appendix VII) is a continuous self report inventory. The BAI is designed to measure both the severity of anxiety symptoms and to discriminate between anxiety and depression in the psychiatric population, for use in clinical practice and research. Each BAI item refers to a specific anxiety symptom measured according to 4 self-evaluating statements indicating symptom severity, scored 0 (least), 1, 2, and 3 (most). The total BAI score ranges from zero to 63 points and is calculated by adding together the scores for each of the 21 items. A validated cut-of score (≥19) (Beck 1990) is reported to represent the diagnostic threshold (DSM -111 -Tr; APA 1987).The BAI demonstrates high internal consistency ([alpha] = 0.92) and test – retest reliability over 1 week (r(81) =.75) with good factorial validity.

5.17 Operational score categories

To assist the interpretation of findings for the frequency and co-existence of postpartum symptoms of depression and anxiety operational score categories were generated. Validated cut-off scores for the EPDS (≥13) (Cox 1987), BDI (≥13) (Beck 1987) and BAI (≥19) (Beck 1990) were retained providing a category for risk of

disorder. For all three measures the score range *below* cut-off scores was further divided by selecting the median score to yield two additional categories;

- A *sub-threshold* range proposed to represent symptom severity associated with some impairment and/or distress, below the validated risk criteria (BDI and EPDS = 7-12 points; BAI = 10 -18 points).
- A *sub-clinical* range proposed to represent symptoms experienced without impairment and/or distress (BDI and EPDS = 1-6 points; BAI = 1-9 points).

Operational score categories for period rates were defined according to the highest scored category achieved by subjects across the two episodes of screening, combined.

Understanding the implication of zero scores (EPDS, BDI and the BAI) was not a focus for this study. Further, no studies were found to have discussed the significance of *zero scores* thus precluding any meaningful interpretation of *zero* data. However, for lucidity, because *zero* scores contribute to the score profiles of the three measures, relevant data are reported alongside outcomes for the operational score categories.

5.18 Statistical analysis

The statistical analyses used in this study are described in detail in Chapters 6, 7 and 8. The preliminary analysis (Chapter 6) examines descriptive data for health visitor and postnatal subject participation, recruitment and screening and categorical data for subject demographics, using mean, standard deviations, minimum and maximum values. Internal reliability of the EPDS, BDI and BAI was investigated using a range of

analyses; scale performance was explored using rotated principal component analysis.

Analyses for *Research Questions 1.1 and 1.2* (Chapter 7) investigated cross sectional data for EPDS, BDI and BAI total scores, according to the operational score categories, using mean (standard deviation) and median values, with 95% CI's, applied to point and period rates for discrete and co-existing symptoms of depression and anxiety. Point rates were estimated for depression and anxiety symptoms at each occasion of screening. Period rates were estimated based on the highest operational score category achieved by subjects *across* both screening episodes.

Analyses for *Research Questions 2.1 and 2.2* (Chapter 8) investigated the frequency and severity ratings for individual EPDS items using percentages, mean (standard deviations) with 95% confidence intervals (CI's).

Correlation analysis was used as a preliminary investigation of the relationship between total scores for the EPDS, BDI and BAI at single time point and across time and to investigate the relationship between covariates and total scores (dependent variables) for the three scales. One way between groups analysis of variance (ANOVA) was computed to examine differences in mean score variance and between the three geographical cohorts at single time points and over time.

Non-parametric techniques were used to investigate change in total scores and for the risk score categories for the EPDS, BDI and BAI, between the 1st and 2nd episodes of screening and for the change scores.

5.18.i Missing data

For each subject, measurement scales that were less than 80% completed were excluded from analysis. Where at least 80% of a scale was complete the mean score for existing data points was calculated with this value entered for the missing data. Where subject data was investigated across the two screening intervals the '*exclude cases pairwise'* option was used for the relevant analysis.

5.19 Chapter summary

Following ethical approval (regional and Trust) consenting postnatal women were recruited by generic, case holding health visitors. Subjects completed the EPDS, BDI and BAI to assess the presence of symptoms of depression and anxiety on two occasions in the early postpartum period. Sample demographics and scale performance were examined; rates for point, period and co-existence in symptoms of anxiety and depression were assessed according to operational categories together with change in symptoms and over time (Research Question 1.1 and 1.2); analyses of ratings for individual EPDS items was undertaken (Research Question 2.1 and 2.2).

To follow, Chapter 6 presents pre-analysis findings describing the health visitor and postnatal participants and the psychometric performance of the EPDS, BDI and BAI in the study population.

CHAPTER 6

SAMPLE DEMOGRAPHICS AND PERFORMANCE OF MEASUREMENT SCALES IN THE POSTNATAL COHORT

6.1 Synopsis

Chapter 6 describes the pre-analysis findings for health visitor and postnatal subject participation for the two occasions of screening. The performance of the Edinburgh Postnatal Depression Scale (EPDS) (Cox et al 1987), Beck Depression Inventory (BDI) (Beck et al 1961) and the Beck Anxiety inventory (BAI) (Beck et al 1988) in postnatal women is also investigated.

- **Section 1 – Sample demographics**

Section 1 presents descriptive statistics for health visitor and postnatal subject participation and the baseline demographic profile of postnatal subjects. In addition, data for recruitment and screening are explored for the collective and geographical cohorts.

- **Section 2 – Performance of measurement scales in the postnatal cohort**

Section 2 reports outcomes for factor analysis undertaken to test the underlying structure and performance of EPDS, BDI and BAI in postnatal subjects.

Section 1 – Sample demographics

6.2 Participants: health visitor and postnatal subject profiles

Research participants were generic health visitors and eligible postnatal women, so defined by the research protocol, from three NHS Trusts within South and Mid-

Glamorgan. The pre-analyses investigated a range of categorical, ordinal and continuous data for both the health visitor and postnatal cohorts; rates of participation, recruitment and screening are explored together with a small number of demographic variables for the postnatal subject groups.

6.2.i Health visitors

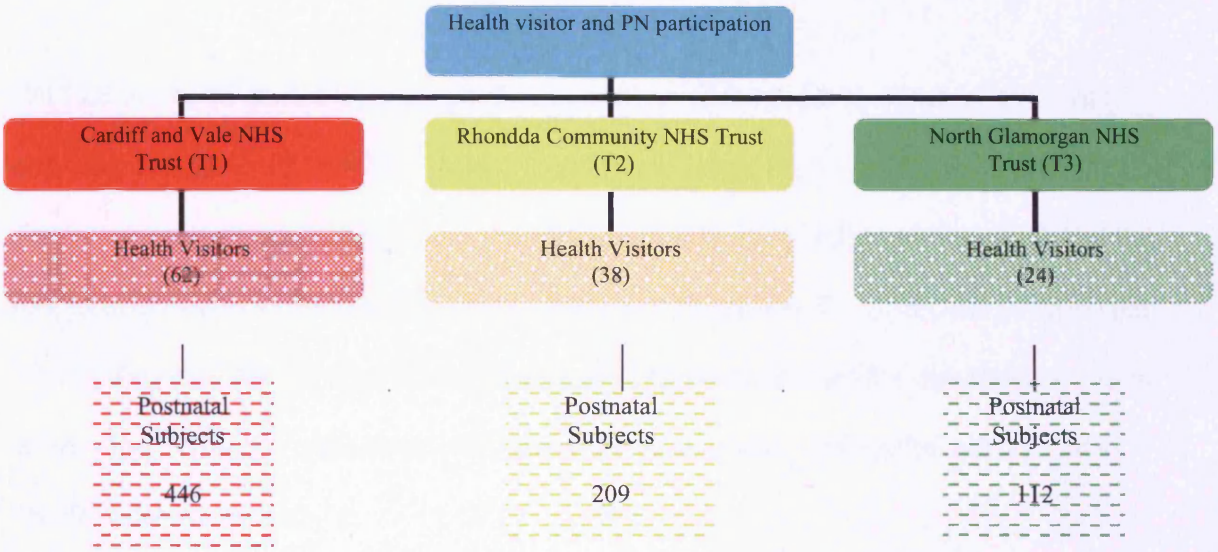
Across the three NHS Trusts a total of (N) 226 generic health visitors were eligible to participate in the study. Of these (n) 144 health visitors were employed in Trust 1 with 43.1% (n = 62) of these participating; (n) 44 were employed in Trust 2 with 86.4% (n = 38) participating; (n) 38 were employed in Trust 3 with 63.2% (n = 24) participating. In total of 54.9% (n = 124) of eligible health visitors (N = 226) agreed to participate.

6.2.ii Health visitors and non-participation

A total of (n) 102 eligible health visitors did not participate with four categories of non-participation evident; (a) health visitors who refused to take part from the outset without any reason (n = 40) and (b) with a reason given (n = 14), (c) those who declared an intention to participate but withdrew before data collection commenced (n =15) or after (n = 2) and (d) those health visitors who agreed, received research materials, did not screen subjects and failed to communicate their withdrawal from the study (n = 31).

Figure 6.1 provides an overview of health visitor and postnatal subject participation according to individual Trust cohorts.

Figure 6.1 Health visitor and postnatal subject participation



6.2. iii Postnatal subjects; geographical cohorts

Subjects were recruited from the postnatal populations of Mid and South Glamorgan between March 2003 and August 2004. Welsh Assembly Government statistics for 2003 -2004 (WAG 2005) relevant to the study needs were sourced to provide a general socio-demographic profile for the geographical subject cohorts. The geographical boundaries for these statistics relate to electoral divisions and not those for each NHS Trust. Figures for 2004 are presented in brackets.

- *Trust 1*

Cardiff - population profile

In 2003 (2004), the rate of live births in Cardiff was 3,708 (3,694), representing approximately 49 live births per 1000 women aged between 15 and 44 years; 1,678 (1,667), approximately 45% (45.1%) of live births, occurred outside marriage.

With 28 electoral divisions that ranked from some of the least to some of the most deprived areas, Cardiff rated a higher deprivation score than for Wales as a whole. Ten electoral divisions were in the top 40% most deprived, with one quarter (7) of the electoral divisions in the top 20% most deprived; Butetown, Ely and Caerau were the 3 most deprived electoral divisions in Cardiff. One third of Cardiff's electoral divisions were rated in the 5% least deprived for Wales as a whole. Of families with children 16.6% were workless.

Vale of Glamorgan – population profile

In 2003 (2004) the number of live births was 1,303 (1,262), representing 55 live births per 1000 women aged between 15 and 44 years; 626 (595) occurred outside marriage, representing 48% (47%) of live births.

The 22 electoral divisions of the Vale of Glamorgan were overall less deprived than the rest of Wales and were not higher than Wales as a whole for any poverty indicator domain. Five electoral divisions were in the top 40% most deprived in Wales. Of families with children approximately 17.1% were workless.

- Trust 2

Rhondda Cynon Taff - population profile

The number of live births for Rhondda Cynon Taff in 2003 (2004) was 2,675 (2,741), representing 56·7 live births per 1000 women aged between 15 to 44 years; 1,569 (1,621), representing 58.7% (49.1%) of live births, occurred outside marriage.

The 53 electoral divisions in Rhondda Cynon Taff ranked from some of the least to some of the most deprived in Wales. Levels of deprivation for health and employment and to a lesser extent, income and education, were higher than for Wales as a whole. Thirty eight electoral divisions were rated amongst the top 40% most deprived in Wales, with one half of the electoral divisions rated within the top 20% most deprived. Pen-y Waun in Cynon ranked as the second most deprived area in Wales. Of those families with children approximately 22.1% were workless.

- *Trust 3*

Merthyr Tydfil- population profile

In 2003 (2004), the number of live births was 639 (649), representing 49 live births per 1000 women aged between 15 and 44 years; 417 (414), representing 65.3% (63.8%) of live births, occurred outside marriage.

The 11 electoral divisions of Merthyr Tydfil rated in the top third for deprivation, with the majority in the top 20% most deprived. Deprivation ranks for health and employment were the highest in Wales and higher than Wales as a whole and amongst the highest for income and education. Of families with children approximately 30.2% were workless.

6.2.iv Eligible postnatal women

Eligible postnatal women were recruited from the caseload populations of participating health visitors. A total of (N) 767 postnatal subjects completed measurement scales across the 1st and 2nd screening intervals; subject ages ranged from 16 - 47 years (mean = 28.42 years, sd = 5.9); parity ranged from 1 - 8 children.

Table 6.1 reports data for age, parity (number of children) and total number of postnatal (PN) weeks women completed measurement scales.

Table 6.1: Postnatal subject profile (N = 767)

	Age (yrs)	Parity	1st screen PN weeks	2nd screen PN weeks
min.	16	1	3	10
max.	47	8	12	23
mean (sd)	28.42 (5.9)	1.76 (1.0)	5.7 (1.3)	14 (1.8)
mode	-	1	-	-

6.2.v Postnatal subjects and non-participation

Health visitors recruited postnatal women from their caseloads at the primary birth visit as a pragmatic sample of convenience based upon consent. Fifty five eligible postnatal women refused; (n) 32 (Trust 1, n = 19; Trust 2, n = 6; Trust 3, n = 7) gave no reason although anecdotally, '*returning to work*' was most commonly cited by the health visitor. The remaining 23 postnatal women who refused to participate (Trust 1, n = 18; Trust 2, n = 4; Trust 3, n = 1) cited reasons such as '*not having the*

time...moving to a different area...not wishing to discuss personal matters...believing themselves to be under scrutiny' and '*partner with a brain tumour'*.

6.3 Recruitment and screening profile

The number of postnatal women each health visitor was required to recruit was influenced by a range of factors. These factors included the target sample size (1,275) and, in relation to the geographical cohorts, the annual birth rate for 2000 - 2001 (WAG 2002) and the number of health visitor participants.

A total of (N) 767 postnatal subjects completed all or some of the measurement tools for symptoms of depression and anxiety; 58·1 % (n = 446), 27·4% (n = 210) and 14·5% (n =111) of subjects came from Trusts 1, 2 and 3, respectively.

Trust 1

In *Trust 1 (n) 62* health visitors participated in the study, representing 50% of the total health visitor cohort (n =124). *Trust 1* health visitors were intended to recruit eight or nine postnatal subjects with 64.5% (n = 40) doing so. The remaining 35.5% (n = 22) of health visitors recruited between one and seven subjects. One health visitor, the study co-ordinator, recruited 10.

Trust 2

In *Trust 2* (n) 38 health visitors participated in the study representing 30.6 % of the total (n = 124). *Trust 2* health visitors were required to recruit 6 or 7 postnatal subjects (one health visitor recruited 8 subjects) with this outcome achieved by 73.7% (n = 28). The remaining 26.3% (n =10) recruited between 2 and 4 postnatal subjects.

Trust 3

In *Trust 3* (n) 24 health visitors participated in the study representing 19.4% of the total (n =124). These health visitors were intended to recruit 5 or 6 postnatal subjects with this outcome achieved by 62.5% (n =15). The remaining 37.5% (n = 9) recruited between 1 and 4 postnatal subjects.

The number of participating health visitors provided some safeguard against clustering of those subjects who met measurement criteria for risk of disorder. The maximum number of subjects who met the criteria for risk of disorder per health visitor was three, with only two health visitors screening this number.

6.4 Screening intervals

In order to ascertain point and period rates for postpartum symptoms of depression and anxiety and change over time cross sectional data were collected on two occasions between 4 - 8 (1st screen) and 12 - 16 (2nd screen) postnatal weeks. Measurement scales completed during, before and after these designated screening intervals are recorded as *target,* early and late, respectively.

6.4.i 1st screen (4-8 postnatal weeks)

During the 1st screening interval a total of (n) 759 postnatal subjects completed measurement scales. Outcomes showed that 98·8% (n = 750) of postnatal subjects met the *'target'* period; 0.3 % (n = 2) were screened *'early'* and 0.9% (n = 7) were screened *'late'* (mean number of postnatal weeks 5.3 (7.9)). Of those subjects who completed measurement scales at the 1st screening interval 6.9% (n = 53) did not complete the 2nd screen.

Table 6.2 reports data for the number of subjects who completed measurement scales for the 1st screen.

Table 6.2: Subjects completing 1st screen (n = 759)

	Target 4-8 pn wks (n)	*Early* ≤3 wks (n)	*Late* ≥9/< *12 wks (n)*	Total (n)
Trust 1	57.31% (435)	0.13% (1)	0.53% (4)	57.97% (440)
Trust 2	27.40% (208)	0	0	27.40% (208)
Trust 3	14.10% (107)	0.13% (1)	0.40% (3)	14.63% (111)
Total	98.81% (750)	0.26% (2)	0.93% (7)	100% (759)

Additional analysis showed that at 1st screen the greatest proportion of subjects completed the EPDS (n = 759) followed by the BDI (n =742) and BAI (n = 757).

6.4.ii 2nd screen (12–16 postnatal weeks)

During the 2nd screening interval a total of (n) 714 postnatal subjects completed measurement scales. Outcomes showed that 93·84% (n = 670) of postnatal subjects met the '*target'* period; 1.26 % (n = 9) were screened '*early'* and 4.90% (n = 35) were screened '*late'* (mean number of postnatal weeks 14.0 (1.8)); 1.12% (n = 8) of subjects completed measurement scales at the 2nd screen without undertaking the 1st screen.

Table 6.3 reports data for the number of subjects who completed measurement tools for the 2nd screen.

Table 6.3: Subjects completing 2nd *screen* (n = 714)

	Target 12-16 PN wks (n)	*Early* ≥9/ ≤12wks (n)	*Late* ≥17 *wks* (n)	Total (n)
Trust 1	52.24% (373)	0.14% (1)	3.50% (25)	55.88% (399)
Trust 2	27.73% (198)	0.84% (6)	0.42% (3)	28.99% (207)
Trust 3	13.87% (99)	0.28% (2)	0.98% (7)	15.13% (108)
Total	93.84% (670)	1.26% (9)	4.9% (35)	100% (714)

For the 2nd episode of screening a total of (n) 714 subjects completed the EPDS, with (n) 695 and (n) 714 completing the accompanying BDI and BAI, respectively.

6.5 Summary: Section 1–Sample demographics

Data for symptoms of depression and anxiety in postnatal women were gathered by generic, case holding health visitors from 3 NHS Trusts (n = 124) in Mid and South Glamorgan. The number of subjects each health visitor was required to recruit varied for the geographical cohorts and was determined overall by the estimated sample size, the number of participating health visitors and annual birth rate in each area.

In total (N) 767 postnatal subjects completed measurement scales to assess symptoms of depression and anxiety between 4-8 and 12-16 postnatal weeks with the majority doing so during these designated screening intervals; a small number of

subjects were screened on one occasion only. The most common reason cited by health visitors for women who did not complete the 2nd screen was returning to work.

Section 2 – Performance of measurement scales in the postnatal cohort

6.6 Assessing scale performance

The primary measurement tools used to investigate symptoms of depression and anxiety in the postpartum sample were the Edinburgh Postnatal Depression Scale (EPDS) (Cox et al 1987), Beck Depression Inventory (BDI) (Beck 1961) and Beck Anxiety Inventory (BAI) (Beck 1988). Evidence from studies using factor analysis to investigate the psychometric properties and underlying structure of these scales illustrate the presence of multiple symptom domains (BDI; BAI) and sub-scales (EPDS). In this section a range of analyses has been undertaken in efforts replicate outcomes from validation and other studies, to confirm the performance of measurement scales in the post partum sample. In addition, outcomes from factor analysis have been used to inform the interpretation of analyses for co-existence in particular (objective one, Chapter 7, pg. 139-184). Outcomes for EPDS factor analysis serves to underpin the second objective of this study (see Chapter 8, pg. 185- 201) which examines the composition of the EPDS total scale score as well as the relationship between the range of EPDS item/ symptoms.

6.7 Outline of analyses to assess performance of the EPDS, BDI and BAI

In this section outcomes from the original validation studies and examples of other studies that have investigated the internal reliability and underlying construct of the EPDS, BDI and BAI are summarised (only studies conducted in English are

presented). The following analyses undertaken in these earlier studies have been repeated to explore the comparability of scale functioning in the postnatal sample.

- Split half reliability (EPDS, Cox et al 1987; BDI, Beck et al 1961)
- Cronbach's alpha (EPDS, Cox et al 1987; BAI, Beck et al 1988)
- *Kruskal- Wallis Non-Parametric Analysis of Variance by Ranks* (BDI, Beck et al 1961)
- Pearson Correlation Analyses (EPDS, Cox et al 1987)
- Inter-item and item-total correlations (undertaken only for the current data)
- Rotated PCA (EPDS, Brouwers et al 2001; BDI, Steer et al 1987; BAI, Beck at al 1988). Rotated principal component analyses have been repeated for both episodes of screening in this study to assess whether the measurement scales performed consistently over time in the postnatal sample.

Reliability of the EPDS, BDI and BAI, indicating how free the scales are from random error, that is the unpredictable fluctuation in measurement outcomes (Pallant 2002, p. 6-7), has been widely reported. Internal reliability, measuring the degree to which items consistently measure the latent variables of a scale (De Vaus 2002, pg. 198) provides one means of assessing random error. The analyses undertaken to confirm the internal reliability of the EPDS, BDI and BAI in relation to their application in this postpartum cohort is recognised to represent an important step for interpreting the research results (Politt et al 1957).

The reference studies have, in all cases, applied principal component analysis to the EPDS, BDI and BAI, to confirm hypotheses regarding the underlying structure of the scales. Convention in factor analysis makes the distinction between *exploratory*

principal component analysis (PCA) and *confirmatory* factor analysis (FA) methods. Broadly, the former reduces the size of large data sets to a smaller number of latent variables whilst the latter tests hypotheses about underlying scale constructs (for example, Dancey and Reidy 2002, p. 408-409). PCA investigates both the shared and unique variance and assumes no error in contrast to FA which investigates only the *shared* variance (Dancey and Reidy 2002, p. 409; Norman and Streiner 2000, p. 163). Differences between PCA and FA are generally not considered to be sufficiently important, with the result both are commonly used in practice to explore and test hypothetical constructs (Dancey and Reidy 2002, p. 408-409; De Vaus 2002, p.186-191; Norman and Streiner 2000, p. 167-168; Palant 2005, p. 172-173).

Components extracted using PCA were subject to mathematical rotation which aids their interpretation by changing the distribution of the variance across factors (Norman and Streiner 2000, p.170). Both the EPDS and BDI were subject to orthogonal (uncorrelated) varimax rotation replicating the analyses of Ross et al (2003) and Steer et al (1987), respectively. In contrast, the BAI was subject to oblique (correlated) promax rotation similar to Beck et al (1988). Norman and Streiner (2000, p.172) propose that both methods are likely to yield similar outcomes; the latter assumes some degree of correlation between components which Norman and Streiner (2000, p172) argues is likely to be more realistic than the completely unrelated factors of varimax rotation. However, promax rotated data are more difficult to interpret compared with the more commonly used varimax rotation (De Vaus 2005, p. 190) where high correlations are emphasised and low ones minimised (Dancey and Reidy 2002, p. 421; Norman and Streiner 2000, p.172).

6.7.i Assessing the suitability of measurement scales for factor analysis

Preliminary assessment of data for the EPDS, BDI and BAI (1st and 2nd screen) was undertaken to investigate scale suitability for Factor Analysis. Both the large sample size (N = 767) and the many strong inter-correlations (majority of coefficients ≥0.3) between scale variables for the EPDS and the BDI, although fewer between BAI items, suggest factor analysis to be appropriate. The factorability of all three scales was further evidenced with the Kaiser-Mayer-Olkin (KMO) value exceeding >0.6 and Bartlett's Test of Sphericity statistically significant (p = < .01). Throughout, *goodness-of-fit* was found to be statistically significant (p = < .001) for all scales at both occasions of screening.

Table 6.4: Results for Kaiser-Mayer-Olkin (KMO) Bartlett's Test of Sphericity applied to the EPDS, BDI and BAI (1st and 2nd screen).

Table 6.4: EPDS/ BDI/ BAI - K-M-O value and Bartlett's Test of Sphericity

	EPDS 1	EPDS 2	BDI 1	BDI 2	BAI 1	BAI 2
coefficients	>0.3	>0.3	>0.3	>0.3	>0.3	>0.3
K-M-O value	0.9	0.92	0.93	0.96	0.96	0.93
Bartlett's Test of Sphericity	p≤0.01	p≤0.01	p≤0.01	p≤0.01	p≤0.01	p≤0.01

Coefficients <0.3 have not been reported (De Vaus 2002, p190); items that cross loaded have been assigned to the component with the highest loading and excluded from interpretation where they failed to adequately discriminate between components (De Vaus 2002, p190). The current data set met the recommended requirement for

Factor Analysis with a minimum of 100 participants and at least 5 participants for each scale variable (Dancey and Reidy 2002, p 409).

In contrast to the original EPDS and BDI validation studies (Cox et al 1987; Beck 1961) postpartum subjects in this study were not subject to clinical interviews therefore clinical data was not available for comparison with the total scales scores. Where possible, rotated PCA components and scale variables have been interpreted and labelled according to the terminology used in the reference study.

6.8 Assessing the performance of the EPDS

The EPDS (Cox 1987) was originally validated as a single construct measure of *postnatal depression* and is the foremost scale used to assess depression in postpartum women both in community practice and research (Gibson 2009). Consequently, the universal validity and widespread use of the EPDS has been influential in promoting postnatal depression as a diagnostic construct. Even so, at face value the EPDS includes both depression and anxiety items despite its status as a one dimensional measure of depression (Cox et al 1987). During development of the EPDS (Cox et al 1987) the specific model used to conceptualise depression was not defined and it might be the case that anxiety was interpreted as a feature of depression postpartum rather than a distinct clinical phenomena (see Clark and Watson 1991).

The influence of the EPDS anxiety items is revealed in a number of outcomes. Findings from more recent studies suggest that the scale is sensitive to anxiety with the total scale score correlating well with some specific measures of anxiety (Stuart et al 1998; Brouwers et al 2001) such the State-Trait Anxiety Inventory (Spielberger et al

1970). Significantly, studies investigating the underlying structure and psychometric properties of the EPDS have demonstrated sub-scales that have been interpreted to represent discrete depression and anxiety (Pop 1992; Brouwers et al 2001; Ross et al 2003). Ross et al (2003) concluded that the EPDS performance was consistent throughout the perinatal period.

The EPDS validation study (Cox et al 1987) compared EPDS total scores for postpartum subjects with a clinical diagnosis of depression based upon Research Diagnostic Criteria (RDC) (Spitzer et al 1975) to determine diagnostic accuracy; the scale was found to demonstrate high internal consistency with split-half reliability and the standardised ά coefficients of 0.88 and 0.87, respectively (Cox et al 1987).

Ross et al (2003) investigated a varimax rotated PCA three component solution for the EPDS in a 6 week postpartum sample, in efforts to replicate earlier findings established by Brouwers et al. (2001). Components were interpreted to represent discrete sub-scales for depression (items 1, 2, 6, 8 and 9) and anxiety (items 3, 4 and 5) which explained 27.0% and 25.4% of the total variance, respectively (Ross et al 2003) (see appendix XI).This outcome was interpreted to justify postnatal depression as a distinct clinical construct based upon the suggested prominence of anxiety (Ross 2003).The third component included item 10 (*self harm/ suicide*) only, which Brouwers et al (2001) previously assessed to be *'...clearly different from the other items'*

Ross et al (2003) failed to report on the internal consistency of the EPDS. However, in a sample of pregnant women at 24 weeks gestation Brouwers et al (2001) found good internal consistency for the 10-item EPDS and the depression and anxiety

subscales with Cronbach's alpha coefficients of *0.80*, *0.79* and *0.60,* respectively. Brouwers et al (2001) established a moderate strength relationship between the depression and anxiety sub-scales using Pearson correlation (coefficient *0.37)* with '*highly significant correlations'* (*P* = <.001) between the 10-item EPDS and each of the sub-scale's although coefficients were not reported

6.8.i EPDS: internal reliability

At 1st and 2nd screen split-half reliability for the EPDS demonstrated high internal consistency with coefficients of *0.74* and *0.81*, respectively, similar to the outcomes reported by Cox et al (1987); inter-item correlations ranged from *0.18 - 0.71* (1st screen) and *0.23 - 0.72 (*2nd screen); item-total correlations ranged from 0.45 - 0.81 (1st screen) and *0.47 - 0.84* (2nd screen). For both episodes of screening all EPDS variables were strongly correlated (standardised ά coefficient = *0.9)*.

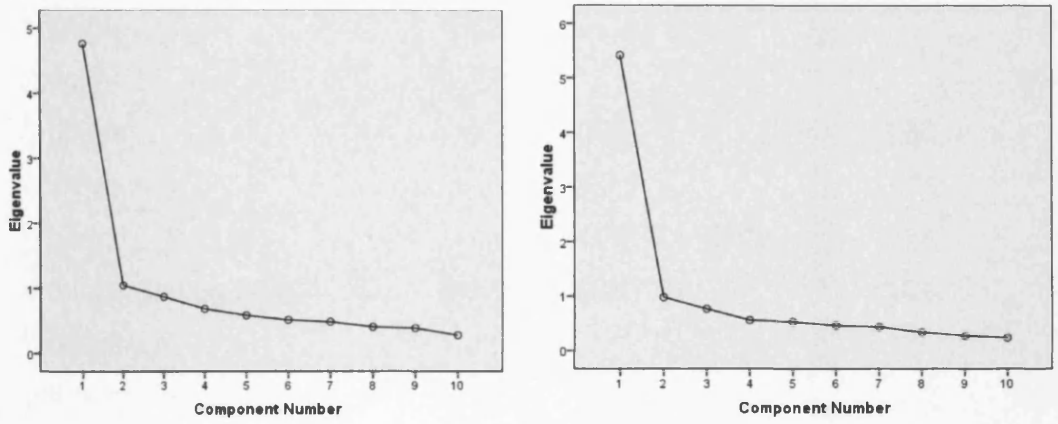
6.8.ii EPDS: rotated PCA

PCA applied to the EPDS (1st and 2nd screen*)* extracted a two component solution in this study based on the scree test (Cattell 1966).

Figure 6.2: EPDS scree plots for the 1st and 2nd screen which show a clear break at the 2nd component for both occasions of screening suggesting two components are relevant.

Figure 6.2: EPDS Screeplots

1st screen **2nd screen**



EPDS data (1st and 2nd screen) were consequently forced to fit a PCA three factor model for subsequent varimax-rotation in order to replicate the findings of Ross et al (2003). Components demonstrated two subscales that have been previously reported to represent discrete measures of depression and anxiety (Brouwers et al 2001; Ross et al 2003) with the third component defined by item 10 (*self-harm/ suicide*). Outcomes for the initial two factor solution established in this study differed from the three factor solution mainly in relation to item 10 which loaded on the depression sub-scale in the two factor solution.

Table 6.5: EPDS varimax-rotated PCA 3 component matrix (1st and 2nd screen).

Table 6.5: EPDS – varimax-rotated PCA 3 component matrix

EPDS items	1st screen			2nd screen		
	Dep.	Anx.		Anx.	Dep.	
	1	2	3	1	2	3
1 laugh	0.80				0.86	
2 enjoy	0.77				0.86	
3 blame		0.78		0.80		
4 anxious		0.76		0.78		
5 panic		0.72	0.30	0.74		
6 things getting on top	0.65	0.42		0.66		0.66
7 unhappy/ difficulty sleeping	0.36	0.35	0.60		0.52	
8 sad/ miserable	0.66	0.47		0.64		
9 unhappy/ crying	0.63	0.45		0.59		
10 suicide/ self-harm			0.90			0.90
Total variance - rotated	27.90%	24.40%	14.30%	33.10%	27.10%	11.60%

At 1st screen the depression sub-scale *(component one* - EPDS items 1, 2, 6, 8 and 9*)* demonstrated high internal consistency *(*Cronbach's alpha coefficient *0.*85*),* explaining 27.9% of the total variance (rotated solution). Similarly, high internal consistency (alpha coefficient *0.74*) was confirmed for the EPDS anxiety sub-scale (*component 2* - items 3, 4, and 5), explaining 24.4% of the total variance (rotated solution), and for the EPDS 10-item scale *(*alpha coefficient *0.87).* Pearson correlation coefficients demonstrated strong relationships between the anxiety and depression sub-scales (*r0.61)*, and between the 10-item EPDS and anxiety sub-scale (*r0.86)* and the depression sub-scale (*r0.91).*

At 2nd screen Cronbach alpha again demonstrated high internal consistency for the anxiety sub-scale (*component 1*), which explained 33.1% of the total variance (rotated solution), the depression sub-scale items (*component 2*), which explained 27.1% of the total variance (rotated solution), and the total EPDS scale, with coefficients of *0.78, 0 .79* and *0.9,* respectively. High Pearson coefficients demonstrated a strong relationship between the depression and anxiety sub-scales (*r0.61*), and between the 10-item EPDS and anxiety sub-scale (r0.89) and depression sub-scale (*r0.85*).

Component loadings varied somewhat between screenings. At 1st screen item 7 was excluded from the final interpretation as it failed to discriminate between components 1 and 2 although this item loaded strongly on component 3. Even so item 10 (*self harm/ suicide*) loaded more strongly than item 7 and overall was interpreted to define component 3. In contrast, at 2nd screen, item 7 loaded on the depression-sub-scale (*component 2*). At the 2nd screen EPDS depression items 6, 8 and 9 were omitted from the final interpretation of components despite relevant loadings on the depression sub-scale at 1st screen. This decision was taken because, at the 2nd screen, item 6 failed to adequately discriminate between the first and third *components* whilst items 8 and 9 loaded on the anxiety sub-scale (*component 1*) but were not considered to influence the overall interpretation.

6.8.iii Summary: Assessing the performance of the EPDS

Good internal consistency was established for the EPDS total scale (1st and 2nd screen) similar to outcomes reported by Cox et al (1987) and Brouwers et al (2001). Rotated PCA of the two and three component solutions investigated in this study replicated the findings of Brouwers et al (2001) and Ross et al (2003), confirming the presence of two sub-scales which have previously been interpreted as discrete

measures of depression and of anxiety (Brouwers et al 2001; Ross et al 2003). A strong relationship was demonstrated between the depression and anxiety subs-scales and between the sub-scales and 10-item EPDS.

6.9 Assessing the performance of the BDI

The 21-item BDI, similar to the EPDS, was validated as a one-dimensional measure of depression '*...to provide a quantitative assessment of the intensity of depression*' in psychiatric outpatients (Beck et al 1961). Of the analyses that could be repeated for the current data Beck et al (1961) investigated the internal consistency of the BDI using 2 methods; *Kruskal- Wallis Non-Parametric Analysis of Variance by Ranks* was used to assess the relationship between the ranked score for each of the 21 BDI items and the total scale score. A statistically significant relationship was established between all BDI items and the total scale score (sig. = <0.001) (Beck 1961). Split half-reliability using odd and even categories showed strong correlations between the two halves (Pearson r = *0.86)* indicating good internal reliability; a Spearman-Brown correction, use to predict reliability if the BDI was extended, yielded a coefficient of *0.93* (Beck 1961).

Studies investigating the internal structure of the BDI found the number of factors to range from 1 - 9 with a consensus that the BDI represents a general measure of depression (Welch et al 1990). Steer et al (1987) established a BDI varimax-rotated PCA three component solution (see appendix XII) in a sample of clinically depressed outpatients based upon the scree test (Cattell 1966); component 1 was interpreted to represent *cognitive-affective and performance difficulties,* component 2, *cognitive*

distortions and component 3, *somatic complaints,* which explained *29.9%, 8.1% and* 6% of the total variance, respectively.

No studies could be found to have investigated the reliability and underlying structure of the BDI in a postpartum sample.

6.9.i BDI: internal reliability

At 1st screen *Kruskal- Wallis Non-Parametric Analysis of Variance by Ranks* suggested a statistically significant relationship (p = <.01) only between the BDI total score (n = 742) and items 11 (*irritated*), 12 (*lost interest in people*), 14 (*appearance), 15* (*work as well as before), 16* (*sleep as well); 17 (more tired), 18 (appetite),* 19 (*weight loss*) and 21 (*interest in sex*). At the 2nd screen a statistically significant relationship was shown between the BDI total score (n = 695) and items 11, 12 and 20 (*worried/ health*) (p = <0.5) and items 14, 17 and 21 (p = <.01). For both screening intervals outcomes failed to replicate the findings of Beck et al (1961). The significant relationships established for the current BDI data might suggest those *features* of depression that are characteristic of non-pathological postnatal mood, compared with clinical depression in a psychiatric population.

Similar to the report of Beck et al (1961) outcomes for the BDI in the postnatal sample (1st and 2nd screen) confirmed good reliability with the split half reliability (odd and even categories) yielding Pearson *r* coefficients of 0.79 (Spearman-Brown coefficient 0.88) and 0.86 (Spearman-Brown coefficient 0.92), respectively. Although not reported in the original validation or reference studies (Beck et al 1961; Steer et al 1987) Cronbach alpha was computed for the BDI (1st and 2ndscreen), demonstrating good internal reliability with coefficients of 0.88 *and* 0 91 respectively; inter-item

correlations ranged from 0.007 - *0.55* (1st screen) and 0.002 - 0.65 (2nd screen); item-total correlation ranged from 0.24 - 0.69 (1st screen) and 0.2 - 0.76 (2nd screen).

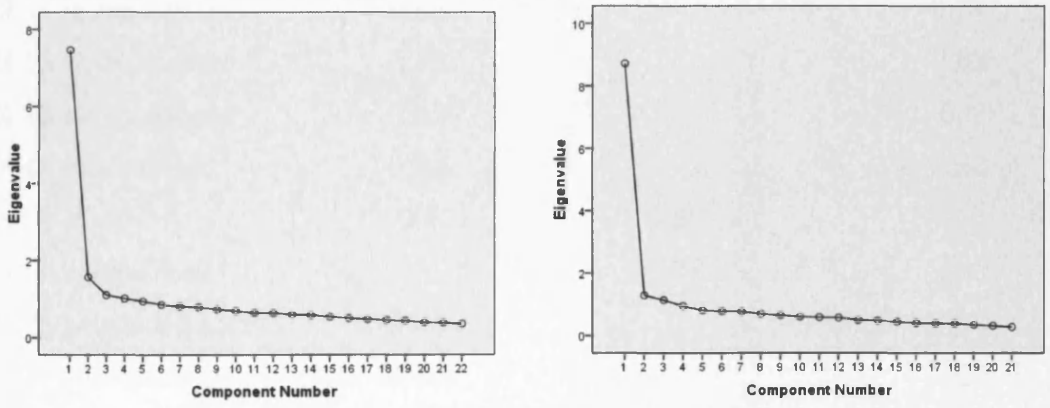
6.9.ii BDI: rotated PCA

PCA applied to the current BDI data at 1st screen (n = 742) and 2nd screen (n = 695) could not replicate the 3 component solution established by Steer et al (1987).The screeplots (Castell 1956) indicated only one component was relevant with a clear break demonstrated just before the second factor.

Figure 6.3: BDI scree plots for the 1st and 2nd screen. The scree demonstrate a clear break before the 2nd component for both occasions of screening, suggesting that only one component is relevant.

Figure 6.3: BDI screeplots

1st screen **2nd screen**



BDI data (1st and 2nd screen) were forced to fit a 3 solution model for subsequent varimax rotation; components were interpreted with regard to the labelling criteria applied by Steer et al (1987); variance for the rotated solution is reported.

Table 6.6: BDI varimax-rotated PCA 3 component matrix for the 1st and 2nd screen.

Table 6.6: BDI varimax-rotated PCA 3 component matrix

BDI items	1st screen			2nd screen		
	1	2	3	1	2	3
BDI 1 sad	0.43			0.70		
BDI 2 discouraged	0.3	*0.53*	0.39	*0.65*		
BDI 3 feel like a failure		0.77		0.78		
BDI 4 satisfaction	0.54	0.42			0.57	
BDI 5 guilty		0.68		0.72		
BDI 6 punishment		0.74		0.69		
BDI 7 disappointment		0.74		0.77		
BDI 8 self blame	0.38	0.59		0.62		
BDI 9 killing self		0.51		0.6		
BDI 10 crying	0.52	0.32		0.52		
BDI 11 irritated	0.62				0.72	
BDI 12 lost interest	0.66				0.66	
BDI 13 making decisions	0.64	0.33			0.55	
BDI 14 appearance	0.54	0.39			0.54	
BDI 15 work as well	0.65				0.63	
BDI 16 sleep as well	0.42				0.4	
BDI 17 more tired	0.62				0.56	
BDI 18 appetite	0.4		0.53			0.56
BDI 19 weight loss			0.73			0.73
BDI 20 worried/ health	0.49					0.43
BDI 21 interest in sex	0.59				0.56	
Total variance	21.10%	19.50%	6.30%	25.40%	20.90%	7.20%

At 1st BDI screen the varimax-rotated 3 component solution failed to adequately replicate the item/ components loadings reported by Steer et al (1987); component 1 was interpreted to represent mixed *cognitive affective* and *somatic domains of depression* and component 2, *cognitive distortions*, explaining 21.1% and 19.5% of the total variance, respectively. Component 3 loaded strongly for items 18 *(appetite)* and 19 (*weight loss*) and explained 6.3% of the total variance.

Rotated PCA outcomes for the 2nd screen failed to replicate the findings of Steer et al (1987) and those of the 1st screen; component 1 was interpreted to represent mixed *cognitive affective* and *cognitive distortions,* explaining 25.4% of the total variance. Component 2 was not adequately represented by the labelling criteria applied by Steer et al (1987) and was interpreted to best represent the interpersonal dimension of depression, explaining 20.9% of the total variance. The third component achieved the highest loading for item 19 (*weight loss*) and item 18 *(appetite*), explaining 7.2% of total variance.

6.9.iii Summary: Assessing the performance of the BDI

The BDI demonstrated good internal consistency in the postnatal sample (1st and 2nd screen) although failed to fully replicate the original validation findings for Kruskal-Wallis Non-Parametric Analysis of Variance (Beck et al 1961). In view of the PCA single solution model found in this study BDI data (1st and 2nd screen) were forced to fit a rotated PCA 3 component model in efforts to replicate findings reported by Steer et al (1987). Although the BDI was found to perform as a discrete measure of depression in the postpartum cohort, item/ component loadings were inconsistent over time and were not adequately represented by the labelling criteria of *cognitive-*

affective and performance difficulties, cognitive distortions and *somatic complaints* applied by Steer et al (1987).

Differences in outcomes are considered to reflect the performance of the BDI in the postpartum sample compared with the depressed psychiatric sample investigated by Steer et al (1987). The loading for BDI item 1 (*sadness*) was significantly lower for the 1st screen compared with outcomes reported by Steer et al (1987) (*0.43* versus *0.65*) although a higher loading was achieved at the 2nd screen (*0.70*).This might suggest that sadness is not a characteristic feature of early postpartum mood compared with clinical depression. BDI item 10 (*crying*) achieved identical loadings for the 1st and 2nd screen (0.52) although failed to adequately load on any component according to Steer et al (1987). This outcome may be specific to the postpartum sample with tearfulness recognised as a common feature of postpartum women, likely to be primarily associated with high levels of prolactin (Cornelius and Labott 2001), compared with the psychiatric population. BDI item 19 (1st and 2nd screen) achieved higher loadings compared with that reported by Steer et al (1987) (*0.73 versus 0.67*) and might reflect a preoccupation with weight gain associated with pregnancy.

6.10 Assessing the performance of the BAI

Postpartum anxiety was assessed using the 21-item BAI (Beck 1988), developed to measure both the severity of anxiety symptoms and to discriminate between anxiety and depression, in an American psychiatric population; two (Beck et al 1988; Steer et al 1993), three (Cox et al 1996; Leyfer et al 2006) and four factor models (Beck and Steer 1991; Steer at al 1993; Osman et al 2002) have been reported based upon the performance of the BAI in clinical and non-clinical populations. Although not

addressed in detail here, it is relevant that the BAI, whilst performing as a discrete measure of anxiety, has been argued to be *panic-centric (for example* Cox 1996), with scale items mostly representing the panic domain.

The BAI validation study (Beck et al 1988) demonstrated high internal consistency (ά = 0.92) with item-total correlations ranging from *0.30* to *0.71* (median *0.60*). BAI promax-rotated PCA revealed two correlated components (*r 0.56*, p<.001), based upon the scree test (Castell 1966), that were interpreted to measure *somatic (*component *1)* and *subjective anxiety and panic* symptoms (component 2) (Beck et al 1988) (see appendix XIII) according to DSM-III-R (APA 1987) classification; BAI item mean (SD) ranged from 0.61-1.89 (0.78-1.14).

Similar to the BDI (Beck 1961), and despite being used to assess rates of perinatal anxiety, no studies could be found to have investigated the performance of the BAI in a postpartum sample.

6.10.i BAI: internal reliability

At 1st (n = 757) and 2nd screen (n =714) the BAI demonstrated high internal consistency with Cronbach alpha coefficients of *0.89* (inter-item correlations 0.06 to 0.66, median 0.52) and *0.92* (inter-item-correlations 0.16 to 0.69, median 0.58), respectively; moderate to strong item-total correlations ranged from 0.41- 0.68 at 1st screen and 0.48 - 0.78 at 2nd screen.

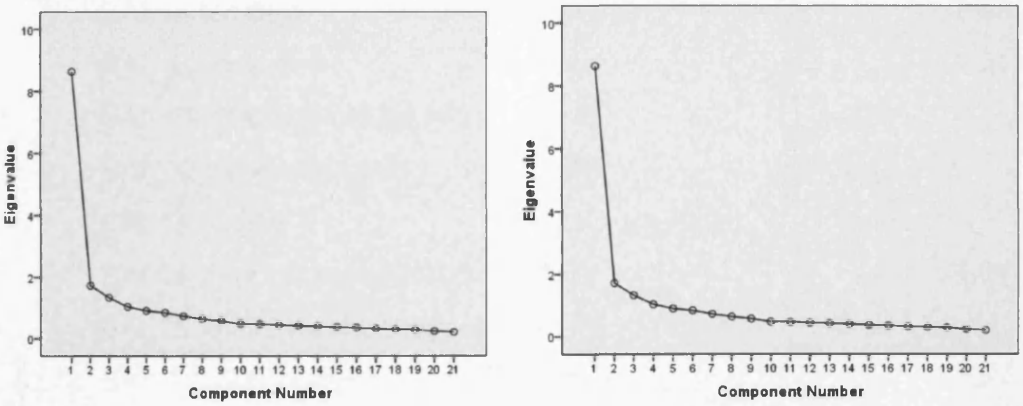
6.10.ii BAI: rotated PCA

PCA applied to the BAI 1st screen (n = 757) and 2nd screen (n = 714) could not replicate the two component solution established by Beck et al (1988) with the scree test (Cattell 1966) indicating only one component was valid.

Figure 6.4: BAI scree plots for the 1st and 2nd screen, which show a clear break before the 2nd component for both occasions of screening, suggesting that only one component is relevant.

Figures 6.4: BAI Screeplot

1st screen **2nd screen**



BAI data (1st and 2nd screen) were forced to fit a two component solution for promax-rotation. Outcomes overall replicated those established by Beck et al (1988); component 1(1st screen) (explaining 21.2% of the total variance) and 2 (2nd screen) (explaining 8.3% of the total variance) measured *subjective anxiety and panic symptoms*. BAI component 2 (1st screen) (explaining 20.4% of the total variance) and component 1 (2nd screen) (explaining 41.2% of the total variance), interpreted to represent *somatic anxiety;* BAI components were strongly correlated at the 1st (coefficient *r0 .61)* and 2nd screen (coefficient *r*0.63).

Table 6.7: BAI promax-rotated PCA 2 component matrix for the 1st and 2nd screen.

Table 6.7: BAI - promax –rotated PCA two component matrix

BAI Items	1st screen		2nd screen	
	1	2	1	2
BAI 1 numbness/ tingling			*0.58*	
BAI 2 feeling hot		*0.63*		*0.44*
BAI 3 wobbliness in legs		*0.53*	*0.76*	
BAI 4 unable to relax	*0.44*			*0.68*
BAI 5 fear of the worst	*0.55*			*0.91*
BAI 6 dizzy/ light headed		*0.70*	*0.84*	
BAI 7 heart pounding/ racing	*0.41*	*0.35*	*0.56*	
BAI 8 unsteady		*0.62*	*0.84*	
BAI 9 terrified	*0.86*			*0.66*
BAI 10 nervous	*0.77*			*0.80*
BAI 11 feelings of choking	*0.60*		*0.55*	
BAI 12 hands trembling	*0.50*		*0.52*	
BAI 13 shaky	*0.32*	*0.45*	*0.63*	
BAI 14 fear of losing control	*0.63*			*0.71*
BAI 15 difficulty in breathing	*0.40*		*0.67*	
BAI 16 fear of dying	*0.62*			*0.59*
BAI 17 scared	*0.84*			*0.84*
BAI 18 indigestion/ discomfort in abdomen		*0.54*	*0.46*	
BAI 19. faint		*0.76*	*0.83*	
BAI 20 face flushed		*0.70*	*0.36*	
BAI 21 sweating (not due to heat)		*0.71*		*0.49*
Total variance	*21.2%*	*20.4%*	*41.2%*	*8.3%*

6.10.iii Summary: Assessing the performance of the BAI

The 21- item BAI demonstrated high internal consistency in the postnatal sample (1st and 2nd screen) similar to findings reported by Beck et al (1988). PCA suggested a

single component model for the BAI on both occasions of screening. Data were subsequently forced to fit a rotated 2 component solution in efforts to replicate the findings of Beck et al (1988). BAI components are interpreted to measure discrete dimensions of *somatic anxiety* and *subjective anxiety with panic,* similar to Beck et al (1988).

6.11 Summary: Section 2 – Performance of measurement scales in the postnatal cohort

The EPDS (Cox 1987), BDI (Beck 1961), and BAI (Beck et al 1988) were the principal measures used to assess symptoms of depression and anxiety in postpartum women. The EPDS and BDI are validated as single construct measures of depression with the former investigated in a postnatal sample and the latter, a psychiatric sample; the BAI has been validated as a discrete measure of anxiety in a psychiatric sample. Earlier studies investigating the psychometric properties and underlying structure of these scales have demonstrated the presence of multiple symptom domains (BDI and BAI) (for example, Richter et al 1998; Steer et al 1987; Steer and Beck et al 1991; Cox et al 1996) or discrete sub-scales for depression and anxiety (EPDS) (Brouwers et al 2001; Ross et al 2003).

Investigation into reliability and performance of the EPDS, BDI and BAI (1^{st} and 2^{nd} screen) in this study was undertaken with reference to earlier studies (Brouwers et al 2001; Ross et al 2003; Beck 1960; Steer et al 1987; Beck et al 1988). The three scales were found to demonstrate high internal consistency using a range of analyses. For all scales the underlying structure was explored using PCA with the scree test supporting single solutions for the BDI and BAI and a two component

model for the EPDS. In efforts to replicate the findings of others EPDS and BDI data were forced to fit a PCA varimax-rotated 3 solution model (Brouwers et al 2001; Ross et al 2003; Steer and Beck et al 1991); with BAI data forced to fit a promax-rotated 2 solution model (Beck 1988).

Findings for the 10-item EPDS (1st and 2nd screen) revealed sub-scales for depression and anxiety, similar to Brouwers et al (2001) and Ross et al (2003). This outcome provides some challenge to the EPDS status as a single construct measure of postnatal depression. In contrast, whilst outcomes for the BDI varimax-rotated 3 component model (1st and 2nd screen) supported its status as a discrete measure of depression, components/ symptom domains were inconsistent over time and not adequately represented the labels applied by Steer et al (1987). These outcomes are interpreted to reflect differences between the postpartum sample used in this study and the psychiatric sample used by Steer et al (1987). The BAI promax-rotated 2 component solution (1st and 2nd screen) was found to replicate the findings of Beck et al (1988) and measured specific domains of *somatic* and *subjective* anxiety,

Overall, outcomes in the postpartum sample were interpreted to confirm that the BDI and BAI consistently performed as discrete measures of depression and anxiety, respectively. In contrast, the EPDS proved a consistent and reliable measure of depression and anxiety (Brouwers et al 2001; Ross et al 2003).

6.12 Chapter summary: Sample demographics and performance measurement scales in the postnatal cohort

Generic, case holding health visitors from 3 NHS Trusts in South Wales (n = 124) gathered data for symptoms of depression and anxiety in consenting postnatal women (n = 767). Individual health visitors were required to recruit between 5 and 9 postnatal subjects, with the number dependent upon the annual birth rate and number of participating health visitors per geographical cohort. Subjects completed self-rating scales between 4-8 and 12-16 postnatal weeks, with the majority doing so during these time frames; a small number were screened on one occasion only. The potential for bias introduced through the clustering of *risk* cases was minimised by the small number of subjects each health visitor was required screen.

Using analysis undertaken in previous studies the performance of the EPDS (Cox et al 1987; Brouwers et al 2001; Ross et al 2003), BDI (Beck et al 1960; Steer et al 1987) and BAI (Beck et al 1988) was investigated. All scales were found to demonstrate good internal reliability. Initial outcomes for rotated PCA demonstrated single solutions for the BDI and BAI with a two component solution for the EPDS. EPDS outcomes for a rotated three component solution replicated the findings of Brouwers et al (2001) and Ross et al (2003), revealing the presence EPDS sub-scales (1^{st} and 2^{nd} screen), interpreted to represent measures of discrete depression and anxiety.

Although outcomes for the BDI (1^{st} and 2^{nd} screen) were interpreted to confirm the scale as a discrete measure of depression, analyses failed to replicate the rotated 3 component solution reported by Steer et al (1987). In the postpartum sample BDI item/ component loadings were inconsistent over time and were not adequately

represented by the component labels used by Steer et al (1987) (component 1 *cognitive-affective and performance difficulties;* component 2, *cognitive distortions and* component 3, *somatic complaints)*. The BAI 2 two component solution (1st and 2nd screen) fully replicated the validation findings (Beck et al 1988), reflecting discrete dimensions of somatic and subjective anxiety.

6.13 Conclusion

The large postnatal sample was recruited from the caseloads of generic health visitors, with no notable difference in key variables, suggesting that they were representative of the postnatal population. Even so the broad socio-economic profile of each geographical subject cohort varies to include some of the most to some of the least deprived populations in Wales.

It can be argued that the EPDS, contrary to its validation status (Cox et al 1987), is not a single construct measure of depression and reliably measures anxiety. Findings for an EPDS sub-scale which consistently comprises of one depression item (blame) alongside items for two broad dimensions of anxiety (*anxious* and *panic*) may represent a combination of symptoms that is characteristic of non-pathological postpartum mood. Therefore the sub-scale interpreted in previous studies to represent a measure of pathological anxiety is defined here as the *postpartum sub-scale* with the rationale for this undertaking discussed fully in Chapter 9. In contrast, the BDI and BAI were found to perform as discrete measures of depression and anxiety, respectively, in the postpartum cohort.

Comparison between outcomes for rotated PCA in this and other studies suggest that the interpretation of the performance of the EPDS, BDI and BAI is subject to a

number of influences. These influences include not only the sample population and the conditions under which the scales are applied but the definitions/ labels that are used to identify the scale items, symptom domains and components, as well as the number of rotated components and the range of symptom domains harboured in the underlying scale construct. These influences have implications for the interpretation of study data. For example, the BDI can be interpreted to measure multiple symptom domains of depression (*negative affect, psychomotor, interpersonal, cognitive* and *motivational)* whilst the EPDS depression items are interpreted in this study to represent *cognitive*, *negative affect* and subjective dimensions. This suggests that even if comparing outcomes only between EPDS depression items and the BDI, such comparison is limited.

Bergin and Lambert (1978, p 172), cited by Steer et al 1987, note that regarding scale construct, '*...the main factors derived from data was more associated with the measurement method or observation used in collecting the data rather than being identified by some conceptual variable that would cut across techniques of measurement'.* Such factors represent important considerations for interpreting findings for the *Research Questions 1 and 2.*

Chapter 7 to follow reports outcomes for 1st objective of this study which examine point and period rates for symptoms for depression and anxiety, rates of co-existence and change in symptom rates, over time.

CHAPTER 7

RATES AND CHANGE OVER TIME IN POSTPARTUM SYMPTOMS OF DEPRESSION AND ANXIETY

7.1 Synopsis

This chapter reports analysis outcomes for the first objective of this study, to measure postpartum symptoms of depression and anxiety at two periods of time in a population sample of postnatal women using the EPDS, BDI and BAI.

Two research questions are considered;

Research question 1.1: What are the key features of postpartum mood?

Research question 1.2: How do rates of postpartum symptoms of depression and anxiety change over time?

Continuous self- rated measures such as the EPDS, BDI and BAI are frequently used to assess child bearing women for depression and anxiety, in clinical practice and research. Attention is typically focused upon validated cut-off scores that indicate at least *risk* of disorder whilst total scores below this threshold receive little if any attention.

Research question 1.1 examines cross sectional data for postpartum symptoms of depression and anxiety gathered at two time points, to estimate point and period rates and co-existence of symptoms. ANOVA was used assess variance in the geographical cohorts. Operational score categories were applied to the total score range for the EPDS (1-30 points), BDI (1-63 points) and BAI (1-63 points), providing

sub-clinical (*without impairment or distress*), sub-threshold (*limited impairment or distress*) and *risk* criteria (*at least risk of disorder*), which aided interpretation of findings. The period rate is defined according to the highest score category for the two occasions of screening. Zero scores are considered separately to the operational score categories. Relevant data are reported for clarity only as zero scores are not subject to interpretation in relation to outcomes for research questions 1.1 and 1.2 (see Chapter 9, pp. *216 - 221*). *Research question 1.2* examines spontaneous change over time in total scores and those at or above the threshold for risk of disorder for the three measures used.

Research question 1.1: What are the key features of postpartum mood?

7.2 EPDS - point and period rates

Findings for this and other studies (Brouwers et al 2001; Ross et al 2003) reported in Chapters 6 confirm the EPDS as a measure of depression *and* anxiety. In this section outcomes for the EPDS are reported for the total scale and are not intended to represent rates of discrete depression (see scale performance, Chapter 6, pp.114-138). Point and period rates for EPDS symptoms (depression and anxiety) are investigated according to the operational categories of sub-clinical (1-6 points), sub-threshold (7-12 points) and *risk* (≥13) scores.

7.2.i EPDS point rates

At 1st (n = 759) and 2nd screen (n = 714) the majority of subjects rated *some* EPDS items/ symptoms (range 1-28 points) (1st = 96.6%; 2nd = 90.3%) with most doing so below the threshold for risk of disorder (1st = 83.9%; 2nd = 79.0%). EPDS scores for

the sub-clinical, sub-threshold and risk criteria were rated by 45.7% (n = 347), 38.2% (n = 290) and 12.7% (n = 96), at 1st screen respectively; rates at 2nd screen were 50.4% (n = 360), 28.6% (n = 204) and 11.3% (n = 81), respectively.

The percentage of subjects who rated sub-clinical symptoms increased between screening episodes whilst rates of sub-threshold and *risk* scores fell. At both occasions of screening the percentage of subjects who rated EPDS total scores at the level for risk of disorder (≥13) was comparable with the reported prevalence of postnatal depression (for example Cox et al 1987; Ross et al 2003; Gavin 2005). Point rates for EPDS zero scores were 3.4% (n = 26) (1st screen) and 9.7% (n = 69) (2nd screen).

Figures 7.1 and 7.2: Bar charts show the frequency of total EPDS scores according to operational score categories for the 1st and 2nd screening intervals.

Figure 7.1: Bar chart - EPDS total scores/ operational categories (1st screen) (n = 759)

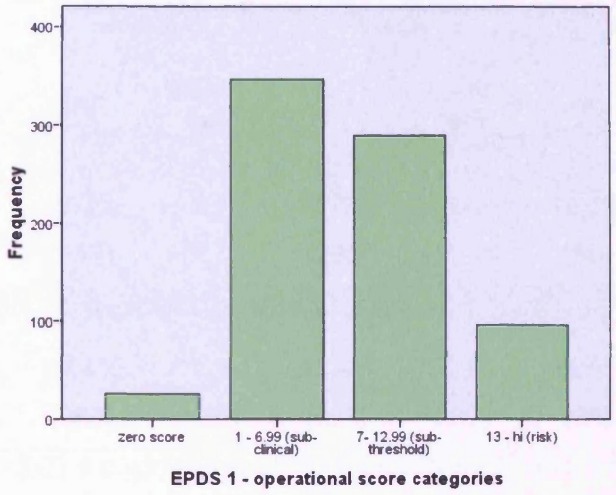


Figure 7.2: Bar chart - EPDS total scores/ operational categories (2nd screen) (n = 714)

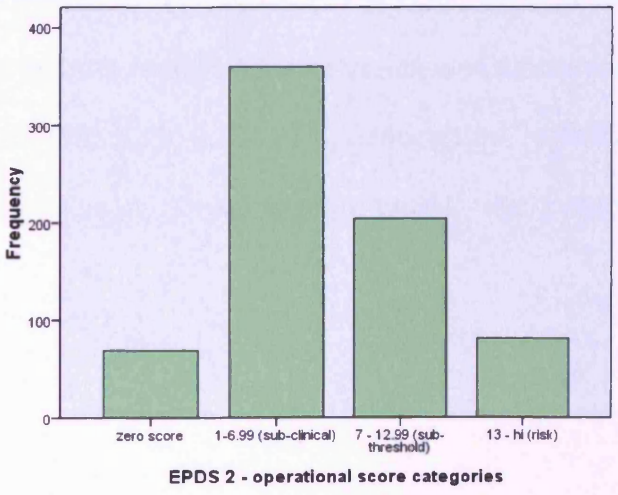


Table 7.3: EPDS percentage rates according to operational score categories and EPDS total mean (SD) scores for the 1st and 2nd screening episodes.

Table 7.3: EPDS point rates

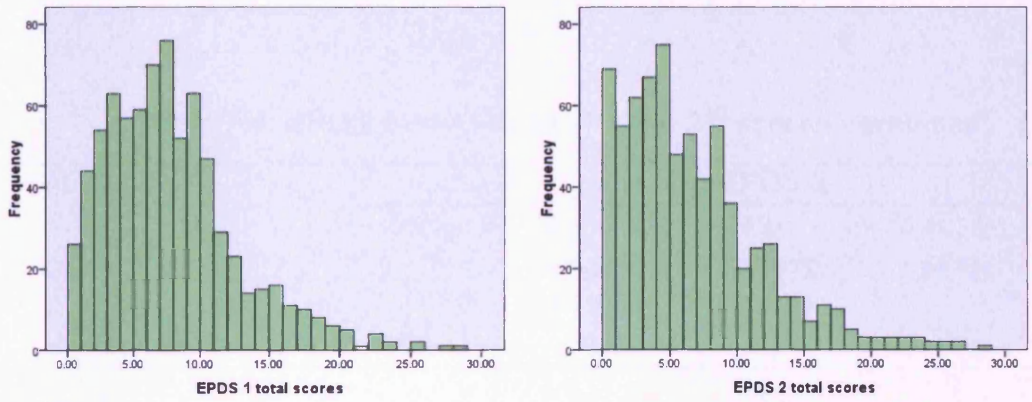
		Sub-clinical score 1-6		Sub-threshold score 7-12		Risk score ≥13		
	zero (n)	(n)	mean (sd)	(n)	mean (sd)	(n)	mean (sd)	*Total mean (sd)
EPDS 1st screen (n =759)	3.4% (26)	**45.7% (347)**	3.7 (1.7)	**38.2% (290)**	8.9 (1.6)	**12.7% (96)**	16.6 (3.3)	7.2 (4.8)
EPDS 2nd screen (n =714)	9.7% (69)	**50.4% (360)**	3.4 (1.6)	**28.6% (204)**	9.0 (1.7)	**11.3% (81)**	17.1 (3.8)	6.3 (5.2)

*Values indicate mean (SD) scores for the EPDS total score - 1st and 2nd screening intervals

The EPDS total mean score fell from 7.2 (4.8) (95% CI, *6.9 - 7.6*) (1st screen) to 6.3 (5.2) (95% CI, *5.8 - 6.6*) (2nd screen) whilst the total SD value increased. A paired sample t-test showed that the mean score reduction was statistically significant ($t(705) = 6.04$, p= <.001) (95% CI, 0.67 – 1.3) although the higher SD value at 2nd screen indicated that total scores became more broadly distributed around the mean, over time.

Figures 7.3 and 7.4: Histograms for the EPDS at 1st and 2nd screen, illustrating the non-normal distribution of data, which are positively skewed, demonstrating the greater concentration of total scores at the lower EPDS score range.

Figures 7.3 and 7.4: Histogram – EPDS total scores
1st screen (n = 759) 2nd screen (n = 714)



Mean (SD) score values for the EPDS sub-clinical and sub-threshold categories remained relatively unchanged between the 1st and 2nd screening intervals. In contrast, the mean (SD) score for EPDS *risk* category (>13) increased, reflecting the higher total SD value at 2nd screen. Outcomes for the Mean (SD) score values are interpreted to suggest that, overall, postnatal subjects experienced an improvement in those symptoms of anxiety and depression represented by the EPDS. However, some subjects who met the criteria for *risk* (>13) at 1st screen were shown to experience a worsening in symptoms over time.

7.2.ii EPDS period rates

The EPDS period rate (1st and 2nd screen combined) (n = 767) demonstrated a distribution of scores similar to EPDS point rates with 97.4% (n = 747), the majority of

subjects, rating *some* symptoms according to the EPDS; 79.8% (n = 612) rated symptoms *below* the threshold for risk (≥13).

Table 7.4: EPDS period rates according to the operational score categories. Data presented in bold represent rates for operational score categories achieved on two occasions of screening. Data for subjects who completed the EPDS on one occasion only are presented in the first column (1st screen only) and first row (2nd screen only).

Table 7.4: EPDS period rates (1st and 2nd screen combined)

		EPDS 2				
		Zero (n)	Sub-clinical (1 - 6) (n)	Sub-threshold (7-12) (n)	Risk (≥13) (n)	Total (n)
EPDS1	(n)	2	2	3	1	8
Zero	3	***15** (2.0%)	7	1	0	26
Sub-clinical (1-6)	24	47	***208** (27.1%)	61	7	347
Sub-threshold (7-12)	22	5	127	***105** (13.7%)	31	290
Risk (≥13)	4	0	16	34	***42** (5.5%)	96
Total	53	70	360	204	81	767 (100%)

* **(n) 706** subjects underwent screening with the EPDS at the 1st *and* 2nd episodes of screening. Period percentage rates were calculated according to the total number of subjects screened using the EPDS (n = 767) across both episodes of screening.

The EPDS sub-clinical rate (score 1 - 6.99) was 37.5% (n = 288) with (n) 24 undertaking the 1st screen only and (n) 2, the 2nd screen only; the sub-threshold rate (score 7-12.99) was 42.2% (n = 324) with (n) 22 undertaking the 1st screen only and (n) 3, the 2nd screen only. The EPDS period rate for risk of disorder (≥13) was rated by 17.6% (n = 135) with (n) 4 undertaking the 1st screen only and (n) 1, the 2nd screen only.

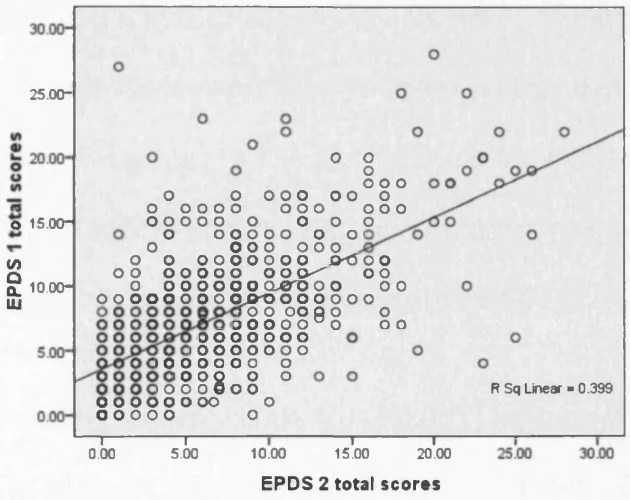
Sub-clinical, sub threshold and risk score criteria were achieved on *both* occasions of screening by 27.1% (n = 208), 13.7 % (n =105) and 5.5% (n = 42), respectively. The period rate for EPDS zero scores, assessed as those who rated zero on both occasions of screening, was 2% (n = 15).

The percentage of subjects who met the EPDS criteria for risk (≥13) on *both* occasions of screening (5.5%) reflects the prevalence of depression in primary care samples reported by Maire et al (1997); this rate was less than half the point rate for the 1st (12.7%) and 2nd (11.3%) screening episodes and three times lower than the total period rate (17.6%). A number of subjects were found to rate EPDS symptoms within the sub-clinical (n = 7) and sub-threshold (n = 31) categories at the 1st screen whilst meeting the criteria for risk (≥13) at the 2nd, suggesting a worsening in EPDS symptoms of depression and anxiety over time. Conversely, (n) 50 subjects progressed from the risk category at 1st screen to sub-clinical (n =16) and sub-threshold (n = 34) levels at the 2nd screen, suggesting their symptoms of EPDS depression and anxiety improved over time.

Pearson Correlation revealed a strong positive association between EPDS scores at 1st and 2nd screen (r 0.63, n = 706, $p<.001$).

Figure 7.5: Scatter plot demonstrating a moderately linear relationship between EPDS total scores for the 1st and 2nd screening intervals; scores are more tightly gathered at the lower score range, becoming more broadly distributed towards the higher score range.

Figures 7.5: Scatter plot - EPDS (1st / 2nd screen)



7.2.iii Summary: EPDS - point and period rates

Findings for the EPDS (1st and 2nd screen) reflect the presence of depression *and* anxiety symptoms and are not interpreted here to represent a discrete measure of depression. At single time points the majority of subjects rated *some* symptoms measured across the EPDS total score range with the greatest percentage within the sub-clinical score category (total score 1-6.99 points), proposed to be symptoms experienced *without impairment or distress*; the sub-threshold score range (7-12.99) represented the 2nd most frequently rated category. Point rates for the EPDS risk category (≥13) reflect findings for the reported prevalence of EPDS postnatal

depression (10-15%), with the period rate for this category slightly higher (17.6%). In contrast to the higher rate of sub-clinical scores at single time points the sub-threshold category (total score 7-12.99), proposed to be symptoms experienced with *limited impairment or distress*, was the most frequently rated period category (42.2%).

Importantly, the rate of EPDS scores within the *risk* category (≥13) at both episodes of screening (5.5%) suggest a sub-group of subjects with enduring symptoms of depression and anxiety and those most likely to suffer a clinical disorder. This was less than half the point prevalence (12.7% at 1st screen; 11.3% at 2nd screen) and three times lower than the period rate (17.6%) and reflected rates of clinical depression found in primary care samples (Maire et al 1997).

EPDS total scores and mean score values ($p = <0.001$) fell over time indicating an overall improvement in subjects' experience of EPDS symptoms. Conversely, an increase in total SD value and the mean (SD) score value for the risk category (≥13) at 2nd screen suggest that some subjects improved to non-risk levels (≤12) whilst a number experienced a worsening in symptoms over time; EPDS zero scores demonstrated a threefold increase.

7.3 BDI depression - point and period rates

Outcomes for the BDI (Beck 1961) are interpreted to reflect its status as a single construct measure of depression in the postpartum sample. Point and period rates are reported for BDI sub-clinical (1-6 points), sub-threshold (7-12 points) and risk (≥13) categories.

7.3.i BDI point rates

Point rates for symptoms of BDI depression revealed a similar distribution of total scores to the EPDS at the 1st (n = 742) (range 1 - 49 points) and 2nd (n = 695) (range 1- 46 points) screening intervals. According to the BDI the majority of subjects experienced *some* symptoms of depression (1st = 94.4%; 2nd = 87.6%) with most doing so below the threshold for risk of disorder (≤12) (1st = 78.8%; 2nd = 74.6%). At 1st screen BDI sub-clinical (1-6), sub-threshold (7-12) and risk categories (≥13) were rated by 48.9% (n = 363), 29.9% (n = 222) and 15.6% (n = 116), respectively; rates at 2nd screen were 56.8% (n = 395), 17.8% (n = 124) and 13.0% (n = 90), respectively. Point rates for BDI zero score were 5.6% (n = 41) (1st screen) and 12.4% (n = 86) (2nd screen).

Figures 7.6 and 7.7: Bar charts for the frequency of BDI total scores according to operational score categories for the 1st and 2nd screening intervals.

Figure 7.6: Bar chart - BDI total scores/ operational categories (1st screen) (n = 742)

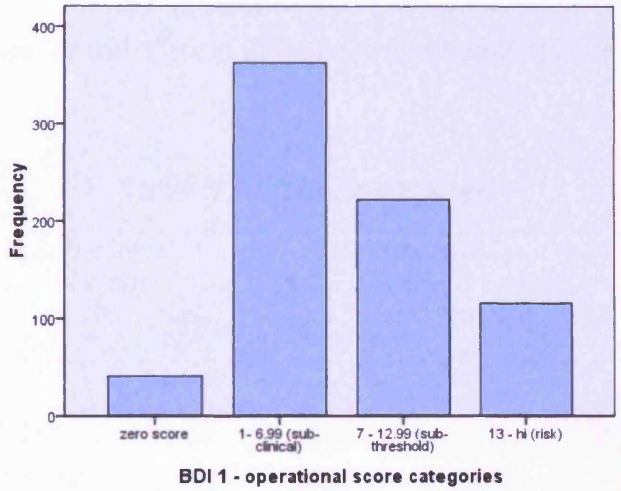
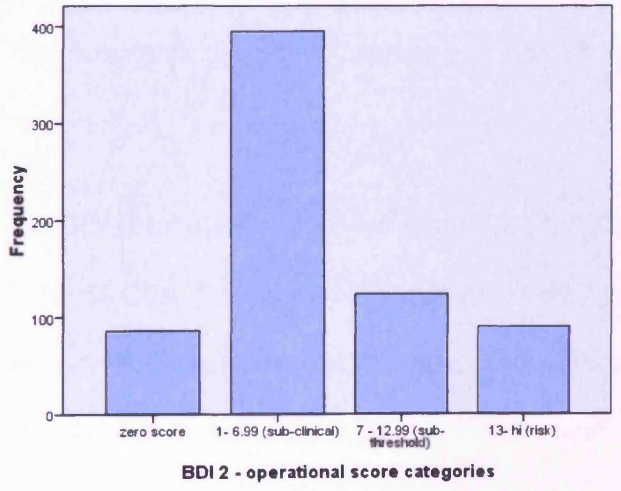


Figure 7.7: Bar chart - BDI total scores/ operational categories (2nd screen) (n = 695)



Over time the reduction in the percentage of BDI sub-threshold and risk scores was reflected mainly in the increased rate of zero scores with a small increase in sub clinical scores.

Table 7.6: BDI percentages rates according to operational score categories and BDI total mean (SD) scores for the 1st and 2nd episodes of data collection.

Table 7.6: BDI point rates

	zero (n)	Sub-clinical (1- 6) (n)	mean (sd)	Sub-threshold (7-12) (n)	mean (sd)	Risk (≥13) (n)	mean (sd)	*Total mean* (sd)
BDI 1 (n = 742)	41 (5.6%)	**363 (48.9%)**	3.32 (1.6)	**222 (29.9%)**	8.8 (1.7)	**116 (15.6%)**	19.4 (7.2)	7.3 (6.7)
BDI 2 (n = 695)	86 (12.4%)	**395 (56.8%)**	3.2 (1.7)	**124 (17.8%)**	9.0 (0.6)	**90 (13.0%)**	21.1 (8.9)	6.1 (7.2)

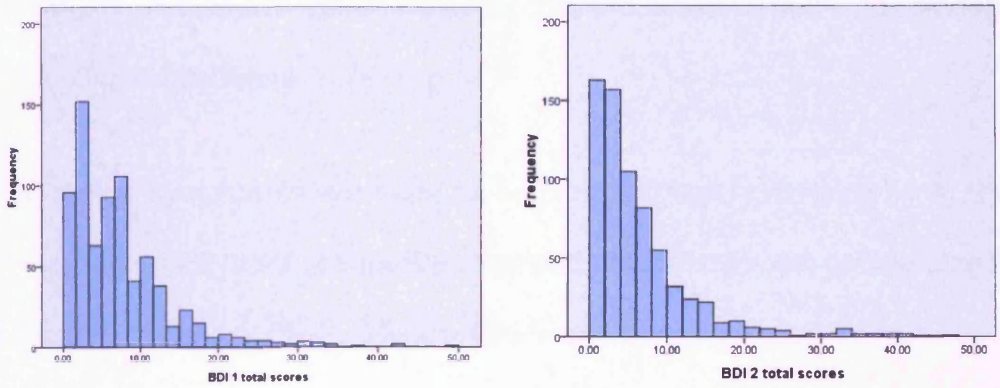
*Values indicate mean (SD) scores for the BDI total score - 1st and 2nd screening intervals

Similar to the EPDS, the BDI total mean score fell from 7.3 (6.7) (95% CI's, *6.8 - 7.8*)) (1st screen) to 6.1 (7.2) (95% CI's, *5.6 - 6.7*)) (2nd screen) whilst the total SD value increased. A paired sample t-test showed that this reduction was statistically significant ($t(677) = 5.4$, p=<0.001) (95% CI, *0.72 – 0.15*) although the increased SD value at 2nd screen indicated that BDI total scores became more broadly distributed around the mean, over time.

Figures 7.8 and 7.9: Histograms for the BDI at 1st and 2nd screen, illustrating the non-normal distribution of data, which are positively skewed, demonstrating the greater concentration of total scores at the lower BDI score range.

Figures 7.8 and 7.9: Histograms – BDI total scores

1st screen **2nd screen**



Mean (SD) score values for the BDI sub-clinical and sub-threshold categories remained relatively unchanged between the 1st and 2nd screening intervals. In contrast, the mean (SD) score for BDI *risk* category (>13) increased, reflecting the higher total SD value at 2nd screen. Outcomes for the Mean (SD) score values are interpreted suggest that, overall, postnatal subjects experienced an improvement in those symptoms of depression represented by the BDI. However, some subjects who met the criteria for *risk* (>13) at 1st screen were shown to experience a worsening in BDI depression symptoms, over time.

7.3.ii BDI period rates

The BDI period rates (n = 759) demonstrate a similar distribution of scores to those for single time points. Outcomes reveal that 96.0% (n = 729) of subjects rated *some* symptoms of BDI depression with 75.6% (n = 574) *below* the threshold for risk (≤12).

The BDI period rate for the sub-clinical score category (1 - 6) was 45.1% (n = 342) with (n) 29 of these screened on the 1st occasion only and (n) 8 on the 2nd occasion only; the period rate for the sub-threshold score category (7 - 12) was 30.6% (n = 232) with (n) 20 undertaking the 1st screen only and (n) 5 the 2nd screen only. The period rate for total BDI scores within the category for risk (≥13) was 20.4% (n = 155) with (n) 11 and (n) 2 of these subjects screened at the 1st and 2nd episodes of screening, respectively.

Sub-clinical symptoms were rated on both occasions of screening by 32.8% (n = 242) of subjects whilst rates of sub-threshold and risk scores were comparable with values 6.5% (n = 49) and 6.7% (n = 51), respectively.

Table 7.7: BDI period rates according to the operational score categories. Data presented in bold represent rates for the operational score category achieved on two occasions of screening. Data for subjects who completed the BDI on one occasion only are presented in the first column (1st screen only) and first row (2nd screen only).

Table 7.7: BDI period rates (1st and 2nd screen combined)

		BDI 2				
		Zero (n)	Sub-clinical (1-6) (n)	Sub-threshold (7-12) (n)	Risk (≥13) (n)	Total (n)
BDI 1		2	8	5	2	17
Zero (n)	4	***24** (3.2%)	13	0	0	41
Sub-clinical (1-6) (n)	29	50	***242** (31.8%)	35	7	363
Sub-threshold (7-12) (n)	20	8	115	***49** (6.5%)	30	222
Cut -off (≥13) (n)	11	2	17	35	***51** (6.7%)	116
Total (n)	64	86	395	124	90	759 (100%)

***678** (n) subjects underwent screening with the BDI at the 1st *and* 2nd episodes of screening. Period percentage rates were calculated according to the total number of subjects screened using the BDI (n = 759) across both episodes of screening.

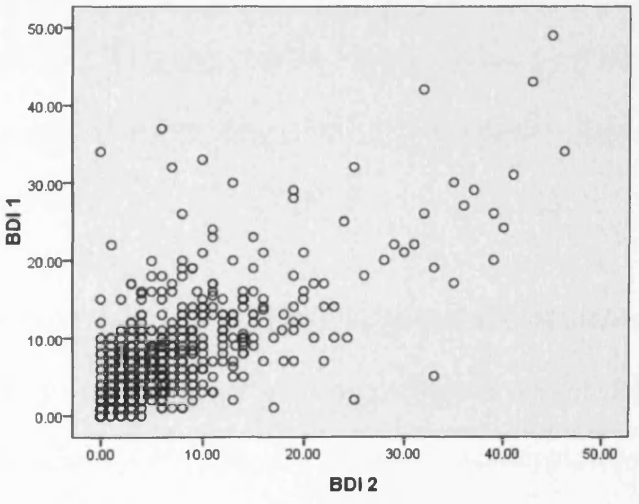
Similar to findings for the EPDS, the percentage of subjects who met the criteria for *BDI risk* at both screening episodes (6.7%) was less than half the rate at 1st screen (15.6%), approximately half the rate for the 2nd screen (13.0%) and almost three times lower than the period rate (20.4%).

A number of subjects progressed from the BDI criteria for risk at 1st screen to BDI sub-clinical (n = 17) and sub-threshold (n = 35) criteria for the 2nd. Conversely, others met BDI sub-clinical (n = 7) and sub-threshold (n = 30) criteria at 1st screen whilst meeting criteria for risk at the 2nd screen, suggesting a worsening of BDI depression symptoms over time. The period rate of BDI *zero scores*, assessed as those who were zero on both occasions of screening was 3.2% (n = 24). No subject who rated BDI zero depression at the 1st screen met threshold criteria at the 2nd; 2 subjects who rated threshold scores at the 1st screen rated zero at the 2nd screen.

Pearson Correlation demonstrated a strong positive relationship between the 1st and 2nd sets of BDI depression scores ($r = 0.70$, $n = 678$, $p < 0.01$).

Figure 7.10: Scatter plot demonstrating a weak linear relationship between BDI total scores for the 1st and 2nd screening episodes; scores are more tightly gathered at the lower score range, becoming more broadly distributed towards the higher score range.

Figures 7.10 - Scatter plot BDI (1st/2nd screen)



7.3.iii Summary: BDI depression - point and period rates

Findings for the BDI represent rates of discrete symptoms of depression according to the operational score categories. BDI point and period rates revealed that, similar to the EPDS, the majority of subjects typically rated *some* symptoms of depression across the total BDI score with the greatest percentage within the sub-clinical score category (score 1 - 6) (*symptoms experienced without impairment or distress*) followed by the sub-threshold (score 7 - 12) and risk score (≥13) categories. Point rates for the BDI criteria for *risk* (≥13) reflect findings for the reported prevalence of *postnatal depression* (10-15%) at the 1st (15.6%) and 2nd (13.0%) screen with the period rate for this category somewhat higher (20.4%). However, these rates were altogether lower than the prevalence reported by Stuart et al (1998) (23.3%) using a BDI cut-off score of ≥10 in postnatal women at 14 weeks postpartum.

Similar to the EPDS, BDI scores within the category for *risk of disorder* (≥13) at both episodes of screening (6.7%) suggest a sub-group of subjects with enduring symptoms of depression representing those at greatest risk or most likely to suffer a clinical disorder. This rate was less than half the point prevalence at 1st screen, approximately half that at 2nd screen and three times lower than the period rate although somewhat higher than the rate of clinical depression found in primary care samples (Maire et al 1997).

The statistically significant reduction in BDI total mean scores between the 1st and 2nd episodes of screening ($p = <0.001$) indicate that subjects overall experienced an improvement in the frequency/ severity of BDI depression symptoms in the first 16 weeks postpartum. Conversely, similar to the EPDS, at 2nd screen the BDI total SD value and the mean (SD) score value for the risk category (≥13) increased. These

outcomes suggest that some subjects improved to non-risk levels (≤12) whilst a number of those *at risk* at 1st screen experienced a worsening in symptoms over time. BDI zero scores more than doubled over time.

7.4. BAI anxiety - point and period rates

Rates of postpartum anxiety symptoms were investigated using the BAI (Beck 1988), which is a single construct measure of anxiety. As with the EPDS and BDI, point and period rates are reported for BAI sub-clinical (1-9 points), sub-threshold (10-18 points) and risk score (≥19) categories.

7.4.i BAI point rates

Point rates for symptoms of BAI anxiety revealed a similar distribution of total scores to the EPDS and the BDI for the 1st (n = 757) (range 1 - 40 points) and 2nd (n = 714) (range 1 - 47 points) screening intervals. The majority of subjects rated *some* symptoms of anxiety (1st = 86.4%; 2nd = 74.2%) with most doing so below the threshold for risk of disorder (≥19) (1st = 80.2%; 2nd = 68.9%). The rate of BAI sub-clinical (1-9), sub-threshold (10-18) and risk scores (≥19) was 65.3% (n = 494), 14.9% (n = 113) and 6.2% (n = 47) at 1st screen, respectively; rates at 2nd screen were 57.4% (n = 410), 11.5% (n = 82) and 5.3% (n = 38), respectively.

Figures 7.11 and 7.12: Bar charts for the frequency of BAI total scores according to operational score categories for the 1st and 2nd screening intervals.

Figure 7.11: Bar chart - BAI total scores/ operational categories 1st screen (n = 757)

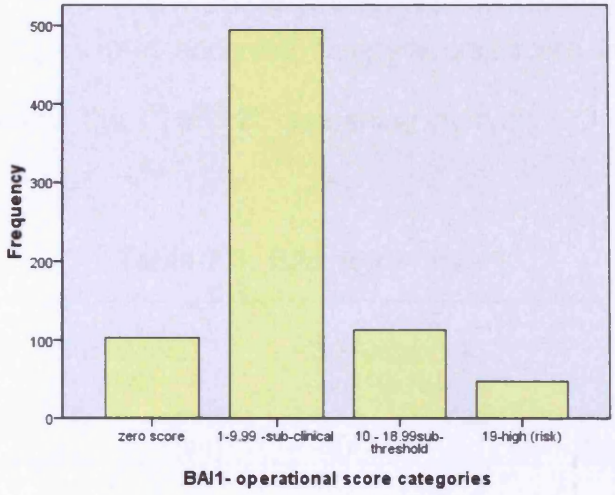
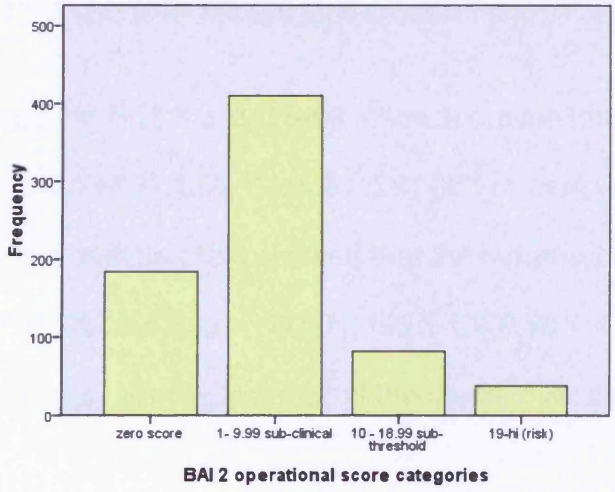


Figure 7.12 - Bar chart BAI total scores/ operational categories 2nd screen (n = 714)



In contrast to the EPDS and BDI the percentage of BAI scores for all operational categories fell over time. This outcome was reflected in an almost two fold increase in the point rate of BAI zero scores from 13.60% (n = 103) at 1st screen to and 25.8% (n = 184) at 2nd screen.

Table 7.8: BAI percentages rates according to operational score categories and BAI total mean (SD) scores for the 1st and 2nd screening intervals.

Table 7.8: BAI point rates

	zero (n)	Sub-clinical (1-9) (n)	mean (sd)	Sub-threshold (10-18) (n)	mean (sd)	Risk (≥19) (n)	mean (sd)	*Total mean (sd)
BAI 1 (n = 757)	103 (13.6%)	**494 (65.3%)**	3.9 (2.4)	**113 (14.9%)**	13.1 (2.5)	**47 (6.2%)**	26.3 (6.0)	6.1 (6.9)
BAI 2 (n = 714)	184 (25.8%)	**410 (57.4%)**	3.5 (2.2)	**82 (11.5%)**	13.4 (2.7)	**38 (5.3%)**	28.5 (8.5)	5.1 (7.3)

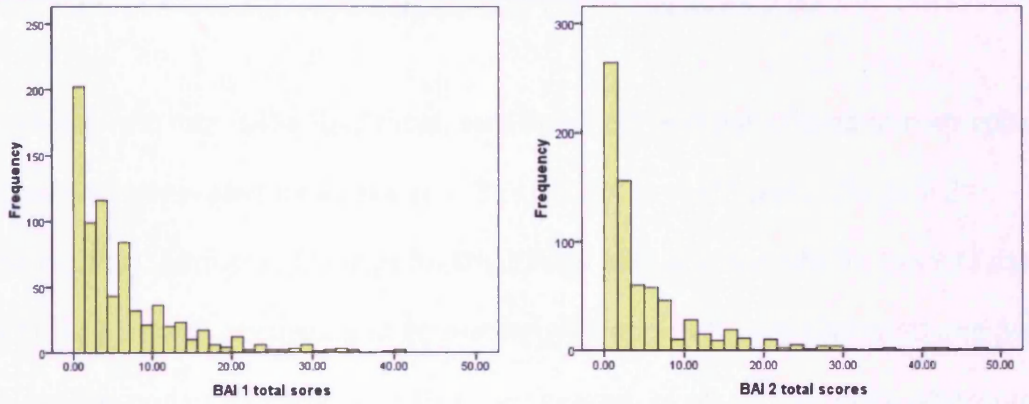
*Values indicate mean (SD) scores for the BAI total score -1st and 2nd screening intervals

Similar to the EPDS and the BDI, the BAI total mean score fell from 6.1 (6.9) (95% CI, 5.6 - 6.6) (1st screen) to 5.1 (7.3) (95% CI, 4.6 - 5.6) (2nd screen) whilst the total SD value increased. A paired sample t-test showed that the mean score reduction was statistically significant (t(703) = 4.6, p = <0.001) (95% CI, *0.58 – 1.44*) although total scores became more broadly distributed around the mean, over time.

Figures 7.13 and 7.14: Histograms for the BAI at 1st and 2nd screen, illustrating the non-normal distribution of data, which are positively skewed, demonstrating the greater concentration of total scores at the lower BAI score range.

Figures 7.13 and 7.14: Histograms – BAI total scores

1st screen **2nd screen**



Mean (SD) score values for the BAI sub-clinical and sub-threshold categories remained relatively unchanged between the 1st and 2nd screening intervals. In contrast, the mean (SD) score for BAI *risk* category (≥19) increased, reflecting the higher total SD value at 2nd screen. Outcomes for the BAI Mean (SD) score values are interpreted to suggest that, overall, postnatal subjects experienced an improvement in those symptoms of anxiety reflected by the BAI. However, the increased SD value at 2nd screen suggests that some subjects who met the criteria for risk (≥19) at 1st screen experienced a worsening in BAI anxiety symptoms over time.

7.4.ii BAI period rates

Period rates for the BAI operational score categories (n = 767) demonstrated a similar distribution of BAI total scores to those found at single time points; 90.2% (n = 692) of subjects rated *some* symptoms of anxiety across the total score range (0 - 63 points)

with 81.9% (n = 628) *below* the threshold for risk of disorder (≥19). Applying the hierarchy of operational score categories the BAI sub-clinical period rate was 64.8% (n = 497) with (n) 33 screened on the 1st occasion only and (n) 8 on the 2nd occasion only; the sub-threshold rate was 17.1% (n = 131) with (n) 10 screened at the 1st occasion only and (n) 1 on the 2nd occasion only. The BAI period rate for risk scores was 8.3% (n = 64) with (n) 2 screened at the 1st occasion only.

Subjects who met BAI sub-clinical, sub-threshold and risk criteria at *both* episodes of screening were rated by 41.9% (n = 321), 5.2% (n = 40) and 2.7% (n = 21), respectively. Similar to findings for the EPDS and BDI, the rate for the BAI risk criteria achieved on both occasions of screening was approximately half point and period rates. The period prevalence of BAI zero scores, assessed as those who were zero on both occasions of screening was 8.6% (n = 66).

Table 7.9: BAI period data according to the operational score categories. Data presented in bold represent rates for the same operational score category achieved the on two occasions of screening. Data for subjects who completed the BAI on one occasion only are presented in the first column (1st screen only) and first row (2nd screen only).

Table 7.9: BAI period rate (1st and 2nd screen combined)

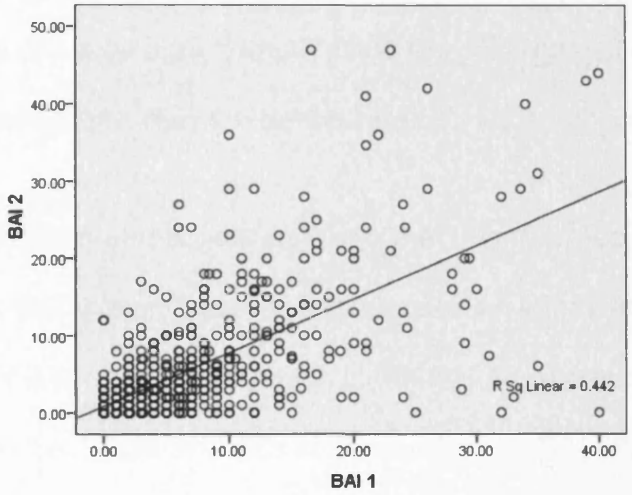
		BAI 2				
		Zero	Sub-clinical (1 - 9)	Sub-threshold (10 - 8)	Risk (≥19)	Total
		(n)	(n)	(n)	(n)	(n)
BAI 1	(n)	1	8	1	0	10
Zero	8	***66** (8. 6%)	27	2	0	103
Sub-clinical (1 - 6)	33	108	***321** (41.9%)	29	3	494
Sub-threshold (7 - 12)	10	6	43	***40** (5.2%)	14	113
Risk (≥13)	2	3	11	10	***21** (2.7%)	47
Total	53	184	410	82	38	767 (100%)

***(n) 704** subjects underwent screening with the BAI at the 1st *and* 2nd episodes of screening. Period percentage rates were calculated according to the total number of subjects screened using the BAI (n = 767) across both episodes of screening.

Pearson correlation demonstrated a strong, positive relationship between the 1st and 2nd sets of BAI scores ($r = 0.66$, $n = 704$, $p < 0.01$).

Figure 7.15: Scatter plot demonstrating a moderate linear relationship between BAI total scores for the 1st and 2nd screening episodes; scores are more tightly gathered at the lower score range becoming more broadly distributed towards the higher score range.

Figure 7.15: Scatter Plot - BAI (1st/2nd screen)



7.4.iii Summary: BAI anxiety - point and period rates

Findings for the BAI represent rates for discrete symptoms of anxiety according to the operational categories. Broadly, findings for BAI anxiety point and period rates were similar to those for the EPDS and BDI; the majority of subjects typically rated some symptoms of BAI anxiety with the greatest percentage falling within the sub-clinical score category (score 1 - 9) (*symptoms experienced without impairment or distress*) followed by the sub-threshold (score 10 -18) (*symptoms experienced with limited impairment or distress*) and *risk* (≥19) categories. BAI point rates for risk (≥19) at the 1st (6.2%) and 2nd (5.3%) screen were lower than the prevalence of BAI anxiety reported by Stuart et al (1998) (8.7%) with estimates based on a cut-off score of ≥10 at 14 weeks postpartum. The period prevalence (8.3%) was also lower than that

reported by Stuart et al (1998) (10.8%) with rates estimated between 14 and 30 postnatal weeks.

Similar to the EPDS and BDI, BAI scores within the category for *risk of disorder* (≥19) at both episodes of screening (2.7%) suggest a sub-group of subjects with enduring symptoms of anxiety, interpreted to represent those at greatest risk or suffering a clinical disorder. This rate was approximately half the point prevalence (1st and 2nd screen) and three times lower than the period rate.

The reduction in BAI total mean scores between the 1st and 2nd episodes of screening ($p = <0.001$) indicate that subjects overall experienced an improvement in the frequency/ severity of BAI anxiety symptoms in the first 16 weeks postpartum. Conversely, similar to the EPDS and BDI at 2nd screen, the BAI total SD value and the mean (SD) score value for the risk category (≥19) increased. These outcomes suggest that over time some subjects improved to non-risk levels (≤18) whilst a number experienced a worsening in BAI anxiety symptoms. The percentage of BAI zero scores almost doubled over time.

7.5 Co-existing symptoms of depression and anxiety (BDI/BAI)

In addition to assessing point and period rates of discrete symptoms *Research Question 1.1* was concerned with investigating the presence of co-existing symptoms of depression and anxiety postpartum. The EPDS (Cox et al 1987) was not included in co-existence analysis as it failed to perform as a discrete measure of depression with outcomes substantially influenced by anxiety items.

Similar to findings for BDI and BAI point and period rates the majority of subjects experienced some co-existing BDI/BAI symptoms of depression and anxiety with rates of 83.0% (n = 614) at 1st screen (n = 740) and 69.2% (n = 480) at 2nd screen (n = 694). Rates below the threshold score for risk according to both scales, represented by combinations of BDI and BAI sub-clinical and sub-threshold criteria were 65.7% (n = 486) at 1st screen and 55.2% (n = 383) at 2nd screen. Rates for the sub-clinical, sub-threshold and risk categories according to both the BDI and BAI were 36.9% (n = 273), 6.5% (n = 48), 4.3% (n = 32), respectively, at 1st screen; rates at 2nd screen were 36.9% (n = 256), 4.3% (n = 30) and 3.6% (n = 25) of postnatal subjects, respectively.

Tables 7.10 and 7.11: BDI/ BAI rates of co-existing symptoms of depression and anxiety according to the operational score categories.

Table 7.10: BDI/ BAI co-existing depression and anxiety (1st screen)

		BAI 1				
		zero (n)	Sub-clinical (1 - 9) (n)	Sub-threshold (10 - 8) (n)	Risk (≥19) (n)	Total (n)
BDI 1	Zero	***17** (2.3%)	24	0	0	41
	Sub-clinical (1-6)	72	***273** (36.9%)	14	3	362
	Sub-threshold (7-12)	12	151	***48** (6.5%)	10	221
	Risk (≥13)	1	33	50	***32** (4.3%)	116
	Total	102	481	112	45	**740** (100%)

The 1st row and 1st column show zero scores the BAI and BDI, respectively, combined with data for the operational score categories and therefore are not representative of co-existence

Table 7.11: BDI/ BAI co-existing depression and anxiety (2nd screen)

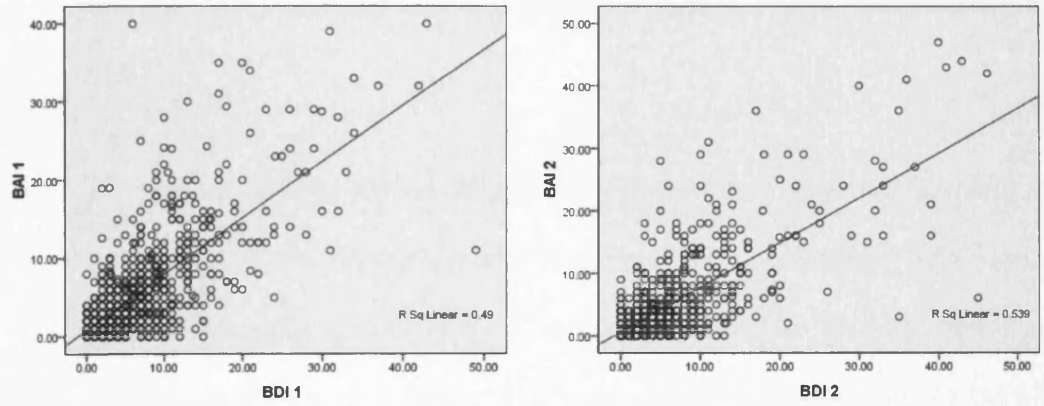
		BAI 2 Zero (n)	Sub-clinical (1-9) (n)	Sub-threshold (10-18) (n)	Risk (≥19) (n)	Total (n)
BDI 2	Zero	***51** (7.3%)	35	0	0	86
	Sub-clinical (1-6)	120	***256** (36.9%)	16	3	395
	Sub-threshold (7-12)	7	81	***30** (4.3%)	6	124
	Risk (≥13)	1	30	33	***25** (3.6%)	89
	Total	179	402	79	34	**694** (100%)

The 1st row and 1st column show zero scores the BAI and BDI, respectively, combined with data for the operational score categories and therefore are not representative of co-existence

Additional analysis of BDI/ BAI data showed that at the 1st screen (n) 13 subjects rated symptoms of anxiety within the risk category (≥19) whilst rating the BDI sub-clinical (score 1-6) (n = 3) and sub-threshold (score 7-12) (n = 10) categories. Conversely, (n) 83 subjects rated depression symptoms within the BDI risk category (≥13) whilst rating BAI sub-clinical (1-9) (n = 33) and sub-threshold (10-18) (n = 50) categories. At 2nd screen (n) 9 subjects met BAI criteria for risk whilst rating symptoms of depression within the BDI sub-clinical (n = 3) and sub-threshold (n = 6) categories. A total of (n) 63 subjects rated symptoms of depression within the BDI risk category whilst rating BAI sub-clinical (n = 30) and sub-threshold (n = 33) levels of anxiety.

Figures 7.16 and 7.17: Scatter plots demonstrating a moderate linear relationship between BDI total depression and BAI total anxiety scores. On both occasions of screening scores are tightly gathered at the lower score range although become more randomly distributed as total scores increase for one or both scales with this pattern less prominent at the 2nd screen.

Figure 7.16 and 7.17: Scatter plots - BDI/ BAI co-existence
1st screen (n = 740) 2nd screen (n = 694)



Pearson Correlation demonstrated a strong positive association between total scores for the BDI and BAI at 1st (r = 0.70, n = 740, p = <0.01) and 2nd screen (r = 0.73, n = 694, p = <0.01).

7.6 Comparison of geographical outcomes for the EPDS, BDI and BAI

The study sample is comprised of three geographical cohorts with distinct socio-demographic profiles (WAG 2005). A one way between groups analysis of variance (ANOVA) was computed to investigate the difference in mean score variance for EPDS, BDI and BAI total scores, between Cardiff and Vale (T1), Rhondda and Taff Ely (T2) and Merthyr and Cynon (T3), for the 1st and 2nd episodes of screening and between screenings.

Analysis confirmed that the difference in mean score variance for the EPDS between the 3 postnatal cohorts was statistically significant ($p<0.05$) at both occasions of screening:

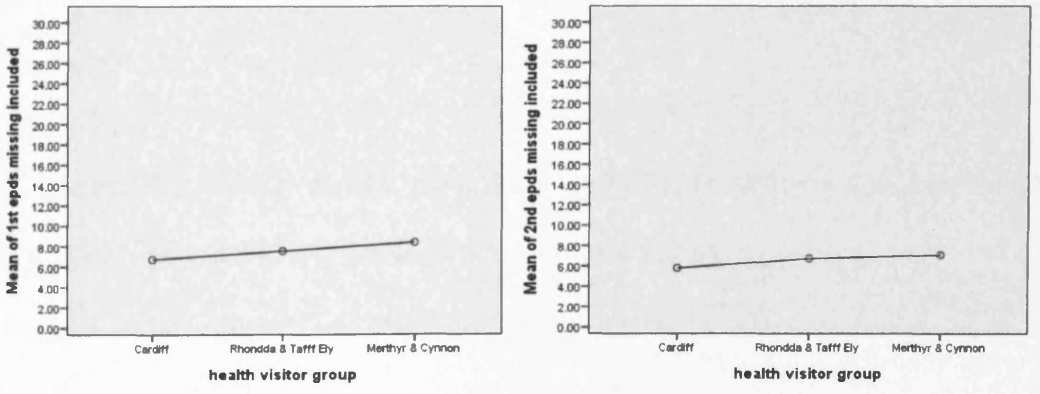
- 1st screen = **[F (2, 756) = 6.4, p = 0.01]**; the effect size **(0.02),** calculated using eta squared, indicated that the difference between the three postnatal cohorts was small. Post-hoc comparisons using Tukey HDS and Scheffe confirmed a statistically significant difference ($p< 0.05$) in mean score variance between T1 (M = 6.71 (4.56)) and T3 (M = 8.4 (5.4)) although neither of these differed significantly from T2 (M = 7.56 (4.91)).

- 2nd screen = **[F (2, 711) = 3.8, p = 0.02];** the effect size (**0.01**), using eta squared, indicated that the difference between the three cohorts was again small. Post hoc comparisons using the Tukey HDS and Scheffe tests failed to demonstrate a significant difference in EPDS mean score variance between T1 (M = 5.79 (4.9)), T2 (M = 6.72 (5.4) and T3 (M = 7.08 (5.75)) ($p>0.05$).

Figure 7.18 and 7.19 show means plots for EPDS total mean scores, for the three geographical cohorts.

Figures 7.18 and 7.19: EPDS Means plots for postnatal cohorts

1st screen **2nd screen**



In contrast to findings for the EPDS, ANOVA confirmed equal variance in BDI mean depression scores for the three cohorts (1st and 2nd screen);

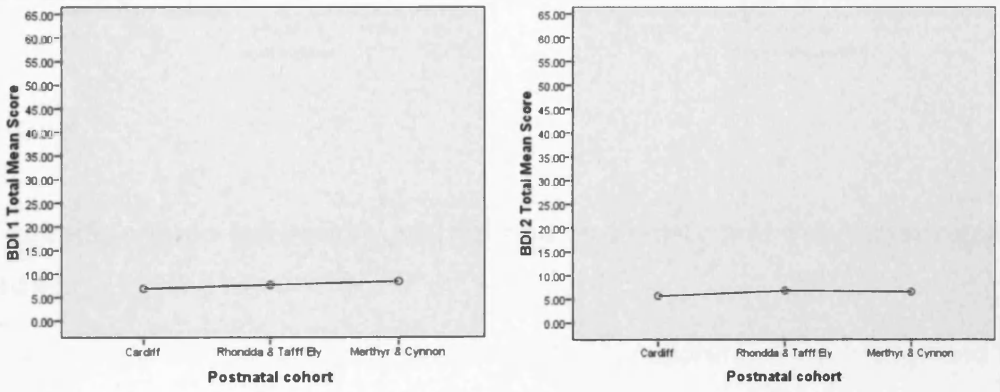
- 1st screen = **[F (2, 739) = 2.68, p= 0.69]** (p>0.05) (Trust 1 (n = 428, M = 6.85 (6.44); Trust 2 (n = 204, M = 7.56 (6.84); Trust 3 (n = 110, M = 8.38 (7.15)).

- 2nd screen = **[F(2, 692) = 1.72, p= 0.179]** (p>0.05) (Trust 1 (n = 387, M = 5.72 (6.85); Trust 2 (n = 204, M = 6.08 (7.65); Trust 3 (n = 104, M = 6.59 (7.63)).

Figures 7.20 and 7.21 show means plots for BDI total mean scores, for the three geographical cohorts.

Figures 7.20 and 7.21: BDI Means plots for postnatal cohorts

1st screen **2nd screen**



Similar to the BDI, ANOVA confirmed equal variance in BAI anxiety means scores for the three cohorts (1st and 2nd screen);

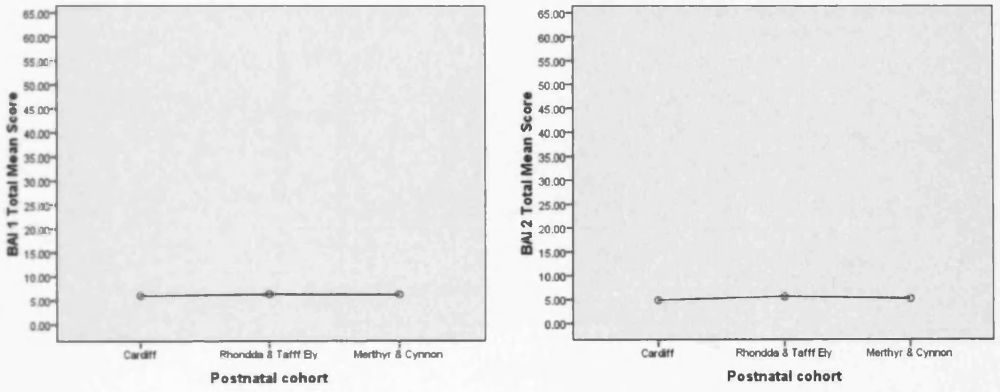
- 1st screen **[F(2, 754) = 0.145, p= 0.865]** (p>0.05) (Trust 1(n = 439, M = 6.0 (6.9); Trust 2 (n = 207, M = 6.3 (7.0); Trust 3 (n = 111, M = 6.2 (6.8)).

- 2nd screen **[F(2, 711) = 0.716, p = 0.489]** (p>0.05) (Trust 1 (n = 397, M = 4.8 (7.0); Trust 2 (n = 208, M = 5.6 (7.5); Trust 3 (n = 109, M = 5.2 (7.9)).

Figures 7.22 and 7.23 show means plots for BAI total mean scores, for the three geographical cohorts.

Figures 7.22 and 7.23: BAI Means plots *for postnatal cohorts*

1st screen ***2nd screen***



7.7 The relationship between maternal age and parity and the measurement outcomes

Two covariates, maternal age and parity (number of children), were recognised to potentially influence outcomes for the EPDS, BDI and BAI (1st and 2nd screen), as dependent variables; the three geographical health visitor/ subject cohorts (Trust 1, Trust 2 and Trust 3) represent the independent variables. Scatter plots were generated to check the relationship between these covariates and independent variables. Scatter plots are produced for the 1st screen only as comparable outcomes were replicated at the 2nd screen.

Figures 7.24 – 7.26: Scatter plots for the relationship between EPDS, BDI and BAI total scores (dependent variables) and maternal age (covariate).

Figures 7.24, 7.25 and 7.26: Scatter plots – maternal age/ EPDS, BDI and BAI (1st screen)

Figure 7.24

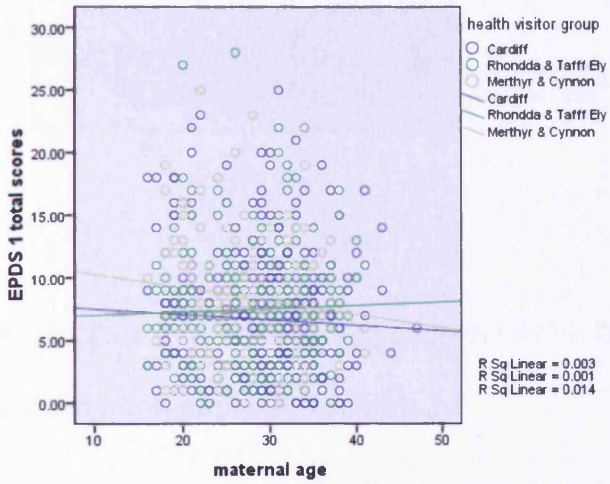


Figure 7.25

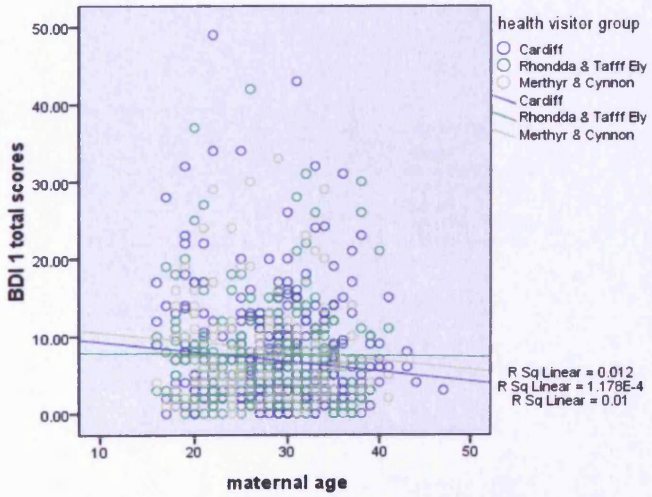
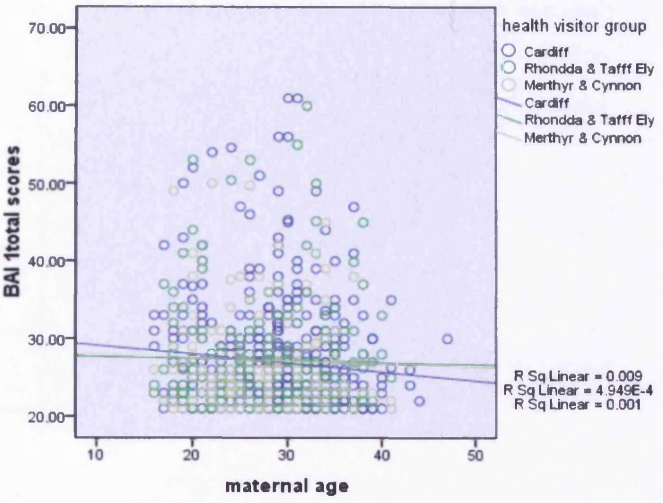


Figure 7.26



Figures 7.27 – 7.29: Scatter plots for the relationship between EPDS, BDI and BAI total scores (dependent variables) and parity (covariate)..

Figures 7.27, 7.28 and 7.29: Scatter plots –EPDS, BDI and BAI/ parity (1st screen)

Figure 7.27

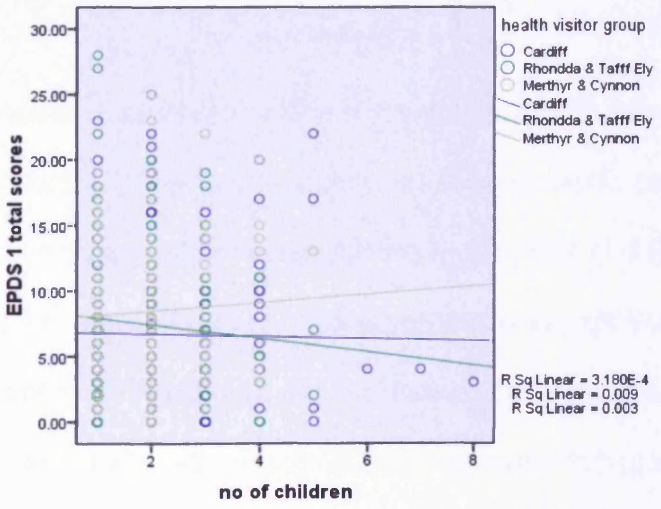


Figure 7.28

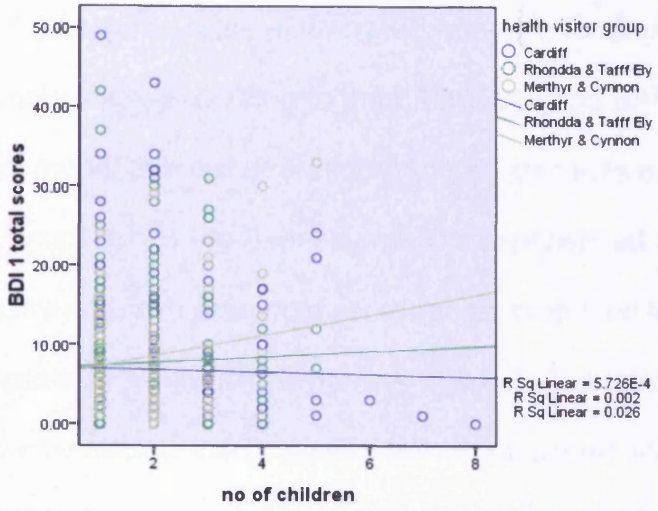
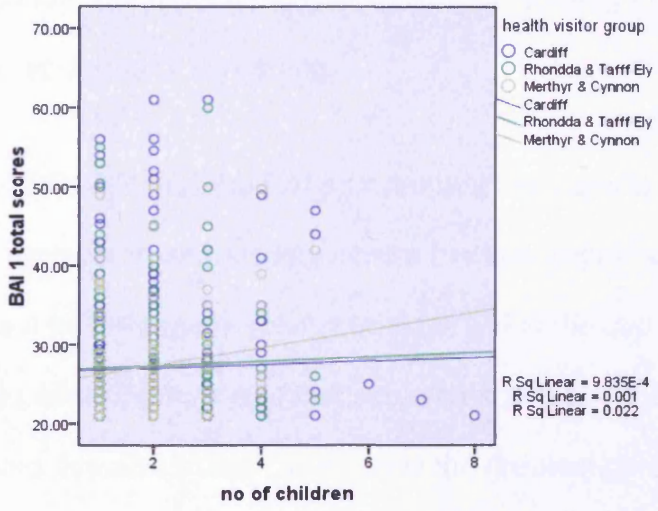


Figure 7.29



In all cases the scatter plots demonstrate a non-linear distribution of data with low R values. These outcomes indicate only a very weak relationship between covariates of maternal age and parity and total scores for the EPDS, BDI and BAI for the three cohorts (1st and 2nd screen); total score variance that was explained by maternal age and/ or parity for the three measures did not exceed 3%. These outcomes suggest that maternal age and parity did not significantly influence subjects' experience of symptoms of depression and anxiety. Consequently further exploration of any potential relationship between variables was inappropriate.

7.8. Analyses summary: Research Question 1.1

Research Question 1.1 explores rates of discrete symptoms for depression and anxiety and their co-existence, according to the EPDS, BDI and BAI (1st and 2nd screen). A theoretical model of three operational score categories defined a *sub-clinical* score range, proposed to represent symptoms experienced w*ithout impairment or distress* and *a sub-threshold* score range, proposed to represent symptoms experienced with *limited impairment or distress*. The third score category indicating *risk of disorder* utilised existing validated cut-off scores for the EPDS (≥13), BDI (≥13) and BAI (≥19). Point rates were defined by data for single episodes of screening with the period rate defined by the highest score category achieved by subjects across both episodes of screening.

Point rates for the EPDS, BDI and the BAI showed that the majority of subjects rated some symptoms of depression and anxiety across the total score range. For all measures the greatest percentage of total scores fell within the *sub-clinical* score range followed by the s*ub-threshold and risk score* categories. Period rates for the BDI and BAI replicated this distribution. In contrast the greatest percentage of EPDS total scores for the period rate fell within the sub-threshold score category indicating that more subjects experienced elevated EPDS symptoms (frequency and/ or severity) compared with the BDI and BAI.

Total scores in the *risk* category were similar to the findings of others for the EPDS (for example see Cox 1987; Gavin 2005), BDI (e.g. Stuart et al 1998) and the BAI (e.g. Stuart et al 1998). The prevalence of *risk* scores at both episodes of screening (EPDS, BDI and BAI), indicating a sub-group of subjects with enduring symptoms and interpreted to suggest those at greatest risk of disorder, are two to three times lower

than point and period rates. Point rates for BDI *risk* scores at 1st (15.6%) and 2nd (12.9%) screen were higher than for the EPDS rates (1st = 12.6%; 2nd = 11.5%). This difference is interpreted to reflect the presence of multiple symptom domains in the structure of the BDI compared with the EPDS.

Although the percentage of total scale scores and total mean score values for the EPDS, BDI and BAI fell over time total SD values increased. These outcomes were reflected in increased mean (SD) score values for total *risk* scores at the 2nd screen suggesting that a number of subjects rated a worsening in symptom frequency/ severity, within the risk category, over time.

Outcomes for combinations of operational score categories for the BDI and BAI (1st and 2nd screen) showed that the majority of subjects experienced co-existing symptoms of depression and anxiety rather than discrete symptoms. Similar to point and period rates for discrete symptoms it was found that *most* subjects rated co-existing symptoms within the sub-clinical score range.

ANOVA confirmed equal variance in BDI and BAI mean scores for the three geographical cohorts ($p>0.05$) with the difference in EPDS mean scores between Trust 1 and Trust 3 statistically significant ($p<0.05$). Scatter plots demonstrated only a weak association between covariates of maternal age and parity and total scores for the EPDS, BDI and BAI suggesting that these factors are of little influence in outcomes for symptoms of depression and anxiety.

In conclusion these findings suggest that the majority of postnatal women experience co-existing symptoms of depression and anxiety, with most rated at a level proposed

to be without impairment and /or distress, which overall improved over time. Maternal age and parity were not shown to significantly influence rates of postpartum symptoms of depression and anxiety.

The relevance of these finding will be addressed in Chapter 9 (*Discussion).* The next section turns to *Research Question 1.2*, which examines the rate and direction of spontaneous change in postpartum symptoms of depression and anxiety, over time.

Research question 1.2: How do rates of postpartum symptoms of depression and anxiety change over time?

7.9 Measuring change in postpartum symptoms of depression and anxiety over time

The literature reviewed in Chapters 2 - 4 highlight important considerations regarding the nature of depression and anxiety symptoms in women after childbirth. For example, the most recent systematic review of postnatal depression (Gavin 2005) demonstrates that the rate of symptoms labelled as *postnatal depression* falls spontaneously after the third month postpartum (see Chapter 3, pp 30-32). Such findings invite further investigation into the rate and direction of spontaneous change in postpartum symptoms of depression and anxiety over time.

7.10. Change over time in postpartum symptoms of depression and anxiety

Change over time in percentage rates, total scores and *risk* scores, has been investigated according to the EPDS, BDI and BAI. Preliminary assessment found data for all measures were non-normally distributed confirming it was appropriate to apply non-parametric techniques; all statistical analyses were 2 tailed (See EPDS

histograms, figures 7.3 and 7.4; EPDS scatter plot, figure 7.5; BDI histograms, figures 7.8 and 7.9; BDI scatter plot, figure 7.10; BAI histograms, figures 7.13 and 7.14; BAI scatter plot, figure 7.15).

7.10.i Change over time in percentage rates

Change in the percentage of subjects rating symptoms of depression and anxiety according the operational score categories, between the 1st and 2nd episodes of screening, was investigated. According to the EPDS, BDI and BAI the percentage of sub-clinical and zero scores increased over time whilst the percentage of sub-threshold and risk scores fell. These outcomes support an overall reduction in the frequency and severity of postpartum symptoms of depression and anxiety.

Table 7.12 reports the difference in percentage rates for the zero and operational score categories for the EPDS, BDI and BAI, between the 1st and 2nd screen.

Table 7.12 Percentage change in rates for depression and anxiety

	EPDS	BDI	BAI
Zero	6.3%	6.8%	12.2%
Sub-clinical	4.7%	7.9%	-7.9%
Sub-threshold	-9.6%	-12.1%	-3.4%
Risk category	-1.4%	-2.6%	-0.9%

7.10.ii Change over time in total scores

Total scores for the EPDS, BDI and BAI were investigated to further assess spontaneous change over time in the percentage rates of postpartum symptoms of depression and anxiety.

The non-parametric Wilcoxon Signed Ranks Test was computed; outcomes showed that ranked EPDS total scores (n = 706) at 2nd screen, compared with the 1st, were rated as *lower* by 57.2% (n = 404), *higher* by 30.9% (n = 218) and unchanged by 11.9% (n =84). The rate and direction of change was found to be similar for ranked BDI total depression scores (n = 678), rated as *lower* by 57.4% (n = 389), higher by 29.6% (n = 201) and *unchanged* by 13.0 % (n = 88). Findings for the change in rates of BAI anxiety were found to replicate those of the EPDS and BDI. Ranked BAI total scores (n = 704) were rated as *lower* by 52.7% (n = 371) at 2nd screen, *higher* by 28.5% (n = 201) and unchanged by 18.8 % (n = 132). The reduction in total scores for the EPDS, BDI and BAI was statistically significant (p = <0.01) suggesting that overall postnatal subjects experienced an improvement in self-rated symptoms of depression and anxiety.

7.10.iii Assessing variance in change scores

The difference in *total change scores* between the three geographical cohorts for EPDS, BDI, and BAI was investigated using one way between groups analysis of variance (ANOVA). Outcomes confirmed equal variance in the mean *change* scores (p = > 0.05) for all measures,

- EPDS; T1 (n= 393, M = -0.88, SD = 4.11), T2 (n = 205, M = -0.84, SD = -1.25) and T3 (n = 108, M= -1.25, SD = 4.39) **[F (2, 698) = 0.36, p= 0.698]**,

- BDI; T1 (n = 375, M = -1.07, SD = 5.37), T2 (n = 200, M = -0.88, SD = -5.66) and T3 (n = 103, M= -1.79, SD = 4.98) **[F (2, 675) = 1.02, p= 0.362]** and

- BAI; T1 (n = 391, M = -1.06, SD = 5.97), T2 (n = 205, M = -0.701, SD = -5.04) and T3 (n = 108, M= -1.05, SD = 6.67) **[F (2, 696) = 0.273, p= 0.76].**

Figures 7.30, 7.31 and 7.32: Means plots for EPDS, BDI and BAI demonstrating mean values for the total *change scores (*2<1) for the three postnatal cohorts.

Figure 7.30: Means plot - EPDS Mean *change* score

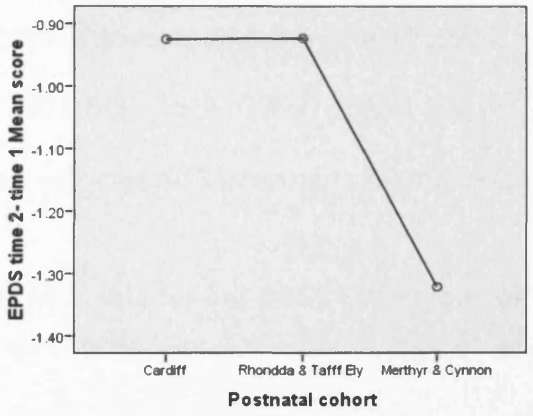


Figure 7.31: Means plot - BDI mean *change* score

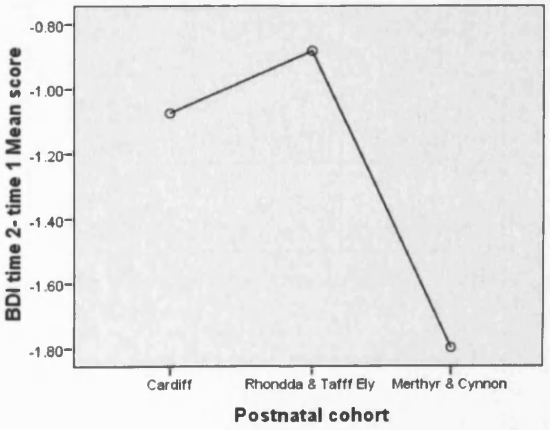
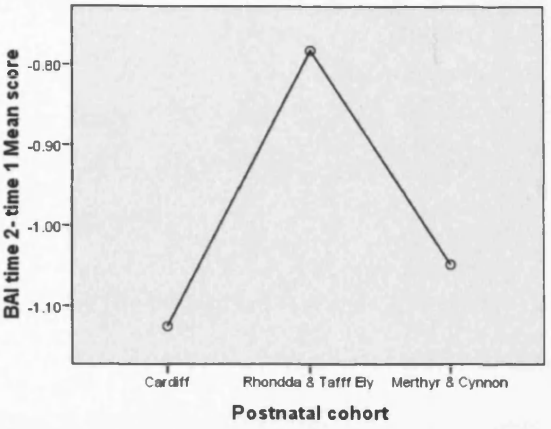


Figure 7.32: Means plot - BAI mean *change* score



7.10.iv Change over time in risk scores

The rate of change in total *risk* scores, between the 1st and 2nd episode of screening for the EPDS (≥13) (n = 706), BDI (≥13) (n = 678) and BAI (≥19) (n = 704), was investigated using the non-parametric McNemar's Matched Observations Test.

Tables 7.13 – 7.15 report 2X2 data for the rate of change over time in total *risk* scores for the EPDS BDI and BAI.

Table 7.13 Change in EPDS *risk* scores (≥13)

	EPDS 2 ≤12	EPDS 2 ≥13
EPDS1 ≤12	576 (81.6%)	38 (5.4%)
EPDS1 ≥13	50 (7.1%)	42 (5.9%)

n = 706 *McNemar's non-parametric test (p = 0.241)

Table 7.14 Change in BDI risk scores (≥13)

	BDI 2 ≤12	BDI 2 ≥13
BDI1 ≤12	536 (79.1%)	37 (5.5%)
BDI1 ≥13	54 (7.9%)	51 (7.5%)

n = 678 *McNemar's non-parametric test (p = 0.093)

Table 7.15 Change BAI in risk scores (≥19)

	BAI 2 ≤18	BAI 2 ≥19
BAI1 ≤18	642 (91.2%)	17 (2.4%)
BAI1 ≥19	24 3.4%	21 3.0%

n = 704 *McNemar's non-parametric test (p = 0.349)

Outcomes showed that the EPDS criteria for *risk (≥13)* was met by 7.1% (n = 50) of postnatal subjects at the 1st screen only and 5.4% (n = 38) at the 2nd screen only; 5.9% (n = 42) met the EPDS *risk* criteria for both occasions of screening. Similarly, the BDI *risk* criteria (≥19) was rated by 7.9% (n = 54) at 1st screen only and 5.5% (n = 37) at 2nd screen only; 7.5% (n = 51) of subjects met the BDI *risk* criteria for both occasions of screening. Outcomes for the BAI risk criteria (≥19) was met by 3.4% (n = 24) at 1st screen only and 2.4% (n = 17) at 2nd screen only; 3.0% (n = 21) met the risk criteria at both episodes of screening.

The null hypothesis was retained with the change over time in total *risk* scores failing to meet statistical significant for the EPDS (p = 0.343), BDI (p = 0.093) and BAI (p = 0.349). These outcomes suggest that the prevalence of postpartum symptoms of

depression and anxiety at the level for risk of disorder was relatively stable over time. However, prevalence rates for the three measures were not derived from a consistent group of subjects but were composed of sub-groups in which symptoms (1) remained unchanged at the level of *risk*, (2) improved from risk to non-risk and (3) deteriorated from non-risk to risk levels.

7.11 Analyses summary: Research Question 1.2

Research Question 1.2 investigated change in rates of postpartum symptoms of depression and anxiety according to the EPDS, BDI and BAI. The statistically significant reduction in total scores for all measures between the 1st and 2nd episodes of screening ($p = < 0.01$) suggests that subjects experienced an improvement in symptoms of depression and anxiety, over time; no difference was found in these outcomes between the three geographical groups. In contrast, the rate of change in total scores meeting the *risk* criteria for the EPDS (≥13), BDI (≥13) and BAI (≥19) failed to meet statistical significance ($p = > 0.05$) with the prevalence relatively stable over time. The estimated prevalence of risk scores, according to the three measures, did not reflect a consistent sub-group of subjects.

7.12 Chapter summary: Rates and change over time in postpartum symptoms of depression and anxiety

Cross sectional data were investigated for self rated symptoms of depression and anxiety in a postnatal sample at two time points, measured according to the EPDS (Cox et al 1987), BDI (Beck et al 1960) and BAI (Beck et al 1988). Outcomes for the EPDS are interpreted to represent rates of co-existing symptoms of anxiety and

depression. Outcomes for the BDI and BAI have been reported to reflect their single construct status as measures of depression and anxiety, respectively.

Findings showed that the majority of subjects experienced some symptoms of depression and anxiety according to the EPDS (≥13), BDI (≥13) and BAI (≥19) with most rated below the threshold for risk of disorder. For all three measures the statistically significant reduction occurred in total mean scores ($p = <0.01$) although total standard deviation values increased. This outcome suggested that subjects overall rated an improvement in symptoms of depression and anxiety between the 1st and 2nd episodes of screening. ANOVA confirmed equal variance in change scores.

In contrast to the reduction in total mean scores over time, mean (SD) values for the *risk* score categories, according to the EPDS, BDI and BAI, increased. This outcome was reflected in a reduction in total risk scores, which failed to meet statistical significance ($p = >0.05$). Furthermore, some subjects were found to experience a worsening of symptoms at the 2nd screen. These women either progressed from non-risk to risk categories overtime or rated an increase in existing risk scores at 2nd screen, for depression and/or anxiety. Conversely, these outcomes also suggest that subjects can experience transient symptoms at the level for risk of clinical disorder before values spontaneously fall to non-risk levels.

7.13 Conclusion

It is not the absence of postpartum symptoms of depression and anxiety that defines non-pathological maternal mood. Most likely it is the presence of a range of co-existing and elevated symptoms mainly experienced below the threshold for risk. Furthermore, the transient nature of symptoms of depression and anxiety typically

associated with the postpartum period, even when meeting the threshold for risk of disorder, distinguishes their potentially *normative* function from the enduring symptoms of psychopathology. Considering this potential, estimating the prevalence of depression in the postpartum period based upon findings for single time points, particularly within the first three months after childbirth, is unreliable. Importantly, the rate of enduring, heightened symptoms at the level for risk, more indicative of actual psychopathology, is shown to be substantially lower than reported rates of *postnatal depression*.

Outcomes for rates of symptoms of anxiety and depression according to the EPDS, BDI and BAI are interpreted to support that (1) the majority of postnatal women commonly experience *some* symptoms with most rated at a level proposed to be without impairment or distressed; (2) the frequency and severity of symptoms diminish over the first 16 weeks postpartum; (3) elevated and transient symptoms of depression and anxiety associated with the postnatal period and proposed to be non-pathological, can meet the criteria for risk of disorder, and (4) subjects who met the criteria for *risk* on two occasions of screening provide a more reliable measure of psychopathology, with the prevalence rate for this group substantially less than estimates for postnatal depression.

Chapter 8 to follow reports outcomes for the second objective of this study, to examine the relationship between rates of anxiety, blame and panic, vis-à-vis other EPDS symptoms.

CHAPTER 8

COMPOSITION OF THE EPDS TOTAL DEPRESSION SCORE

8.1 Synopsis

This chapter reports analysis outcomes for the second objective of this study, to examine the relationship between rates of anxiety, blame and panic, vis-à-vis other EPDS symptoms.

Two research questions are considered;
Research question 2.1: What is the prevalence of anxiety, blame and panic compared with other symptoms according to the EPDS?
Research question 2.2: What contribution do rates of anxiety and guilt make to the overall depression score according to the EPDS?

The EPDS (Cox et al 1987), BDI (Beck 1961) and BAI (Beck 1988) used in this study have been validated as discrete measures of depression (EPDS and BDI) and anxiety (BAI). However, unlike its counterparts the EPDS, at face value, cannot be considered as a *single* construct measure because the scale includes items for depression *and* anxiety. Findings from factor analysis undertaken in this and other studies (Brouwers et al 2001; Ross et al 2003) demonstrate that this combination of EPDS items/ symptoms are of influence in the performance of the scale, transforming the total scale into sub-scales. Findings for the EPDS postpartum sub-scale, which is consistently comprised of EPDS depression item 3 (*blame*) and EPDS anxiety items 4 (*anxious*) and 5 (*panic*), assumes particular importance in light of the specific role played by these features of depression and anxiety in early parental preoccupations

and harm avoidant behaviours (Leckman et al 1999; Feldman et al 1999;) and their proposed utility (Nesse 2000; Keller and Nesse 2005; Gilbert 2006). Furthermore, anecdotal evidence from health visiting practice suggests that ratings for these EPDS items might exert a disproportionate influence upon the composition of the total scale score, compared with the remaining items.

These issues introduce some uncertainty around what the EPDS tells us about the mental domain of the maternal experience postpartum and raise a number of questions about how outcomes for the sub-scales and total scale scores might be interpreted in postnatal women. Constructively, a more detailed inspection of how EPDS items and sub-scales contribute to the EPDS total depression score is seen to provide an opportunity to examine how those *symptoms* of depression and anxiety reflected by the scale may be organised in postnatal women.

The reader is reminded that outcomes for research question 2.2 are presented and interpreted according to findings for rotated PCA found in this study, and reflect the composition of the EPDS sub-scales (see Chapter 6, pg. 114-138). Also, the EPDS sub-scale previously interpreted by Brouwers et al (2001) and Ross et al (2003) to represent *anxiety* is defined here as the *postpartum sub-scale*. This reinterpretation of this sub-scale is discussed fully in Chapter 9.

Research question 2.1: What is the prevalence of anxiety, blame and panic compared with other symptoms according to the EPDS?

8.2 Frequency of subject ratings for individual EPDS items

In general the focus upon prevalence rates and total mean (SD) scores for the EPDS provides the basis for investigating postnatal depression. However, this analysis contributes little to our understanding of the affective content of the mental domain of postnatal women. In this section the frequency of depression and anxiety symptoms reflected by the 10 EPDS items was explored as a means of demonstrating how these may be organised to suggest a characteristic and potentially non-pathological pattern of postpartum mood. This was investigated by examining the percentage of subjects who endorsed individual EPDS items with a score of 1 (least severe), 2 (moderately severe) or 3 points (most severe) (1st and 2nd screen).

At the 1st screen 85.9% (n = 653) rated item 3 (*blame*); 83.9% (n = 423), item 6 (*things getting on top of me*); 75.5% (n = 573), item 4 (*anxious*); 65.5% (n = 495), item 8 (*sad or miserable*) and 53.4% (n = 405) rated item 5 (*panic*). With the exception of EPDS item 9 (*unhappy/ crying*), which was rated by 50.7% (n = 385), the remaining items were rated by less than one third of postnatal subjects; 27.4% (n = 208) rated item 7 (*unhappy/ can't sleep);* 28.2% (n = 214) rated item 1 (*laugh*); 25.9% (n =192) rated item 2 *(enjoy)*; 4.5% (n = 34) rated item 10 (*suicide/ self-harm).*

Figures 8.1 - 8.10: Bar charts showing the percentage of subjects who rated individual EPDS items with a score of 1, 2 or 3 points, at the 1st screen.

Fig. 8.1 - EPDS item 1

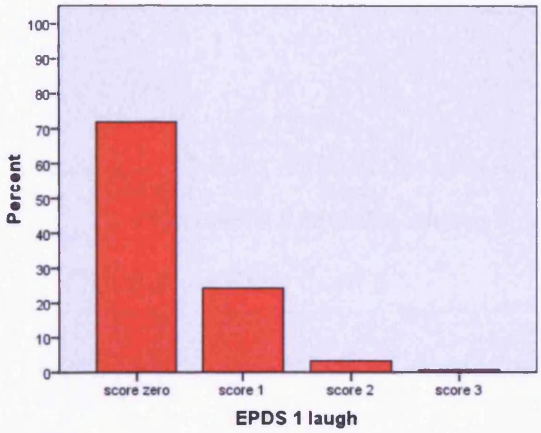


Fig. 8.2 - EPDS item 2

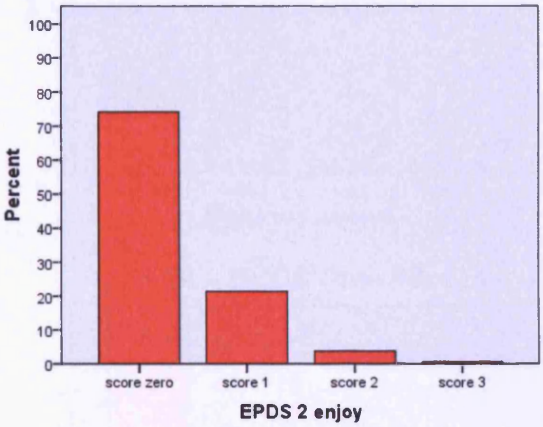


Fig. 8.3 - EPDS item 3

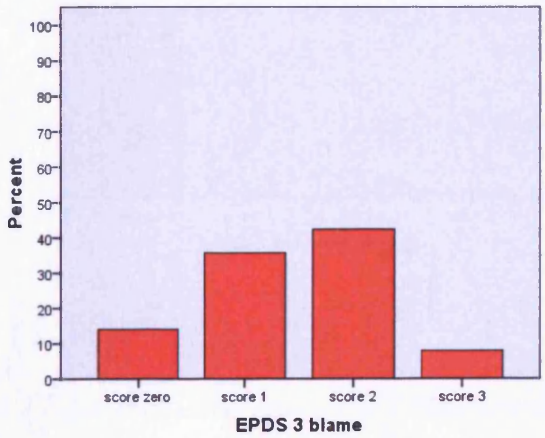


Fig. 8.4 - EPDS item 4

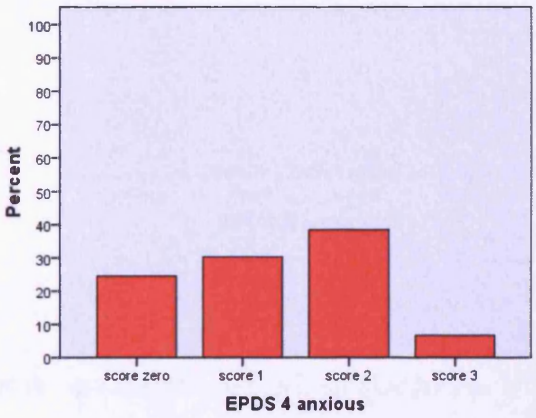


Fig. 8.5 - EPDS item 5

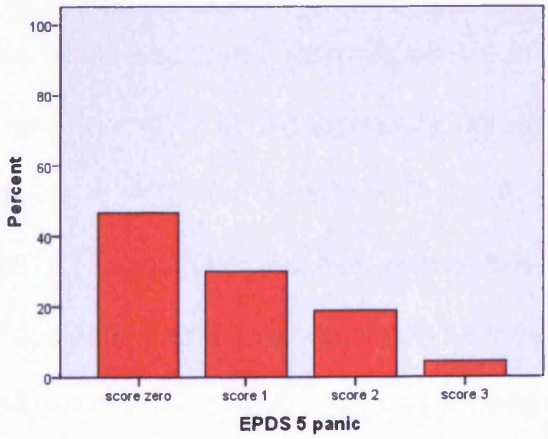


Fig. 8.6 - EPDS item 6

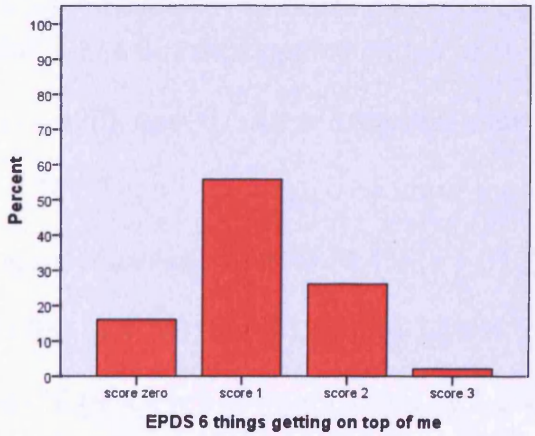


Fig. 8.7 - EPDS item 7

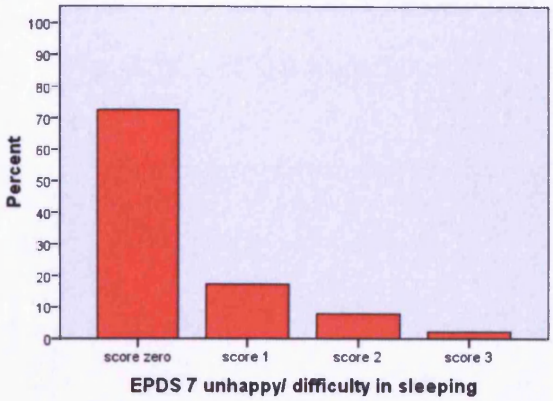


Fig. 8.8 - EPDS item 8

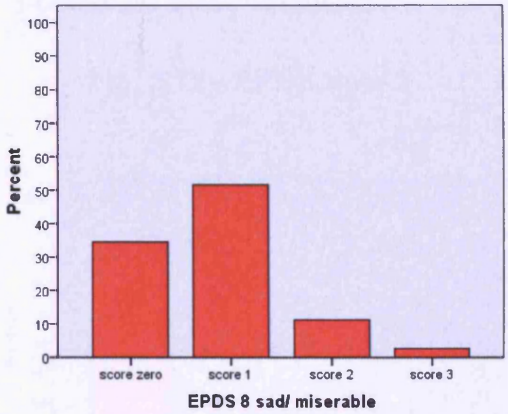


Fig. 8.9 – EPDS item 9

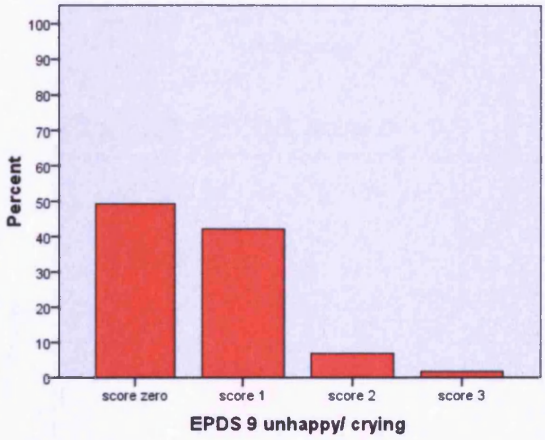
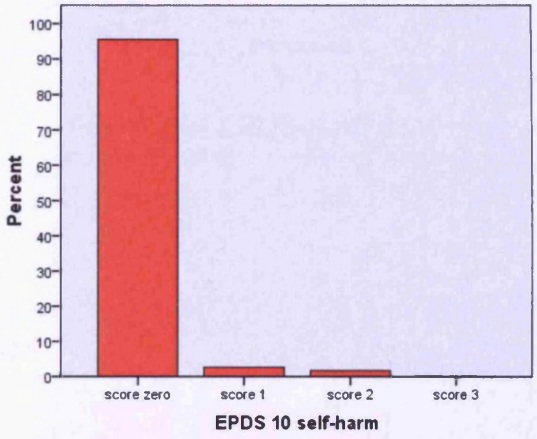


Fig. 8.10 – EPDS item 10



At 2nd screen a distribution of EPDS symptoms was demonstrated, similar to the 1st, according to the percentage of subject who rated individual items (score 1-3); 79.9% (n = 571) rated item 3 (*blame*); 68.2% (n = 487), item 6 (*things getting on top of me*); 67.8% (n = 485), item 4 (*anxious*); 59.95% (n = 428), item 8 (*sad or miserable*) and 48% (n = 343), item 5 (*panic*). The remaining EPDS items were rated by fewer than half of the postnatal subjects with item 9 (*unhappy/ crying*) rated by 43.4% (n = 310) of subjects, item 7 (*unhappy/ can't sleep),* 29.2% (n = 209), item 1 (laugh), 21.6% (n = 154), item 2 *(enjoy)*, 20.7% (n =148) and item 10 (*suicide/ self-harm)*, rated by 6.2% (n = 44).

Figures 8.11 - 8.20: Bar charts showing the percentage of subjects who rated individual EPDS items with a score of 1, 2 or 3 points, at the 2nd screen.

Fig. 8.11 - EPDS item 1

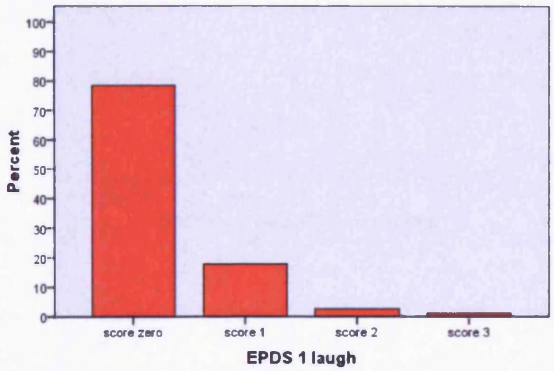


Fig. 8.12 - EPDS item 2

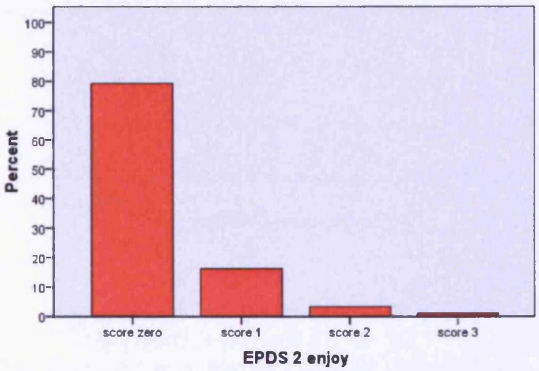


Fig. 8.13 - EPDS item 3

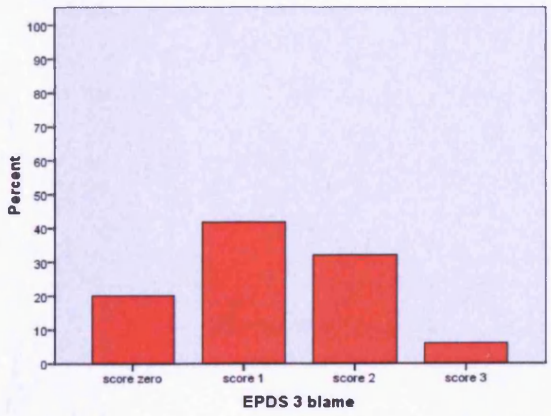


Fig. 8.14 - EPDS item 4

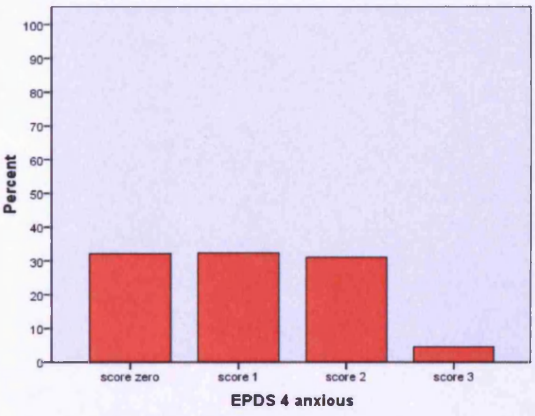


Fig. 8.15 - EPDS item 5

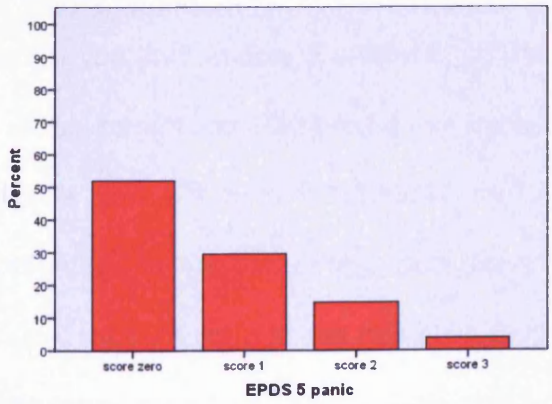


Fig. 8.16 - EPDS item 6

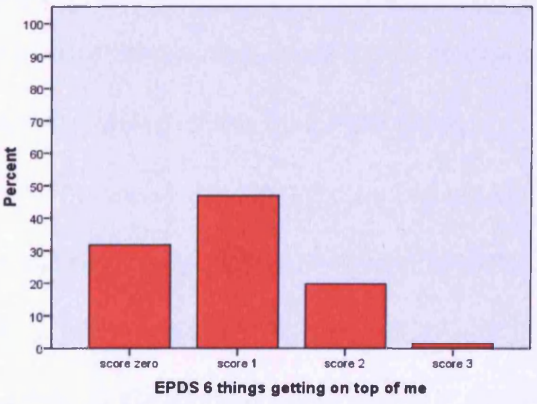


Fig. 8.17 - EPDS item 7

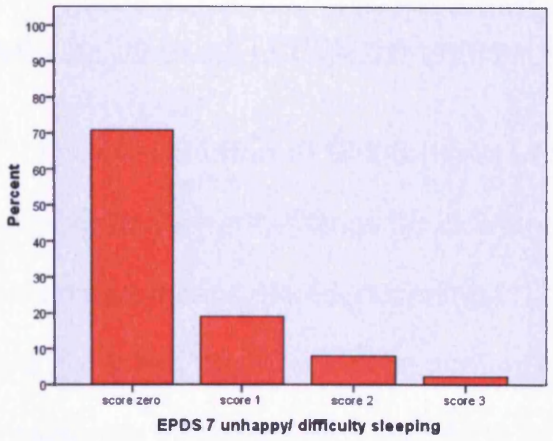


Fig. 8.18 - EPDS item 8

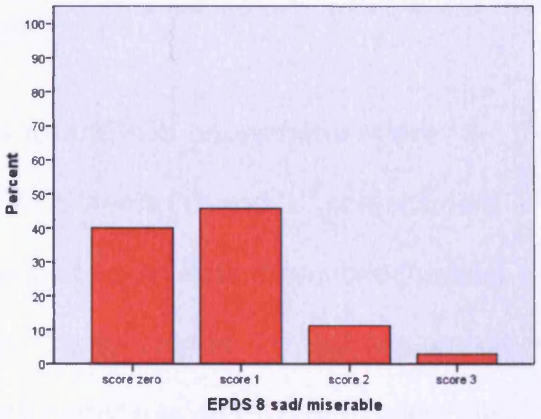


Fig. 8.19 - EPDS item 9

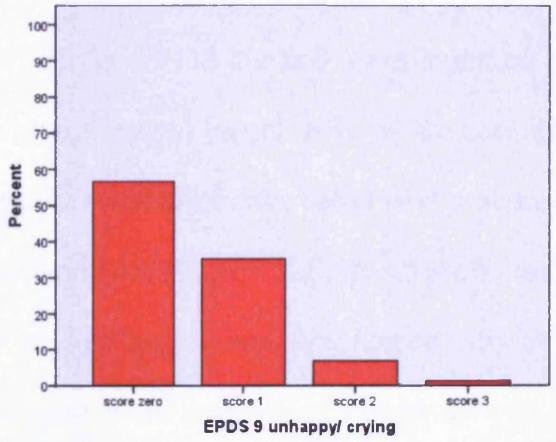
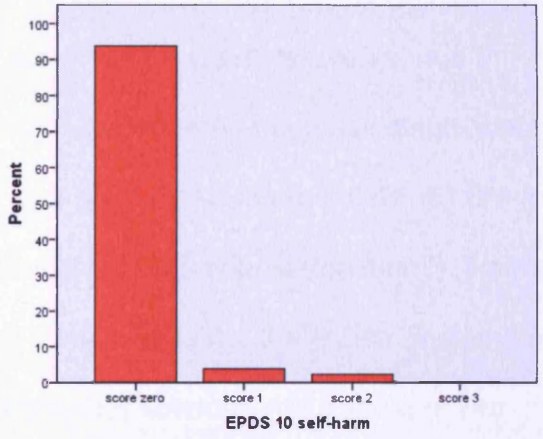


Fig. 8.20 - EPDS item 10



The percentage rates for individual EPDS items, when measured over time, suggest that they are organised to reflect a consistent hierarchy of postpartum symptoms of depression and anxiety. Furthermore, the three postpartum sub-scale items (*blame, anxious, panic*) were within the five most frequently rated of the 10 EPDS items. Notably, item 3, *blame*, a subjective symptom of depression was rated by the greatest percentage of women on both occasions of screening. This raises the possibility that *blame*, similar to subjective anxiety and panic, is a characteristic feature of postpartum mood and non-pathological in the first instance. The implications of this outcome are discussed fully in chapter 9.

Research question 2.2: What contribution do rates of anxiety, blame and panic make to the overall EPDS depression score?

8.3 The contribution of EPDS items to the total EPDS depression score

Frequency and severity ratings for individual EPDS items (1st and 2nd screen) were explored as a means of understanding (1) how these outcomes influenced the total EPDS score and (2) the symptom content of postpartum mood. Throughout, outcomes are presented and interpreted according to findings for rotated PCA and reflect the composition of the EPDS sub-scales in this study (see Chapter 6, pg. 114-138).

At 1st screen EPDS items 6 (*things getting on top of me*), 8 (*sad/ miserable*) and 9 (*unhappy/ crying*) (score 1-3), which contributed to the five-item depression sub-scale, were the *most* frequently rated with a score of one point by 55.7% (n = 423), 51.6% (n = 390) and 42.2% (n = 320) of subjects, respectively. EPDS depression item 3 (*blame*) and anxiety item 4 (*anxious)* (score 1-3), which contribute to the three item postpartum sub-scale, were rated *most frequently with the greatest severity* with a score of two points, by 42.4% (n = 322) and 38.5% (n = 292), respectively.

Table 8.1 (pg.192) reports the percentage contributed by individual EPDS items (score 1-3 points) to total subject scores combined, at 1st screen; mean (SD) and median scores are also reported. Data are presented to illustrate the composition of the EPDS sub-scales established in this study. Item 7 does not contribute to any sub-scale as it failed to discriminate between principle components.

Table 8.1: EPDS item - percentage of combined subject total scores (5464) (1st screen)

		Item 1 *Laugh as usual*	**Item 2** *Enjoy*	**Item 6** *Things getting on top*	**Item 8** *Sad/ Miserable*	**Item 9** *Unhappy/ crying*
EPDS depression sub-scale		(n) 759	(n) 759	(n) 759	(n) 759	(n) 759
	Mean (SD)	0.33 (0.57)	0.31 (0.57)	1.14 (0.69)	0.82 (0.73)	0.61 (0.69)
	Median	0	0	1	1	1
	% of total score	4.6%	4.3%	15.9%	11.3%	8.5%

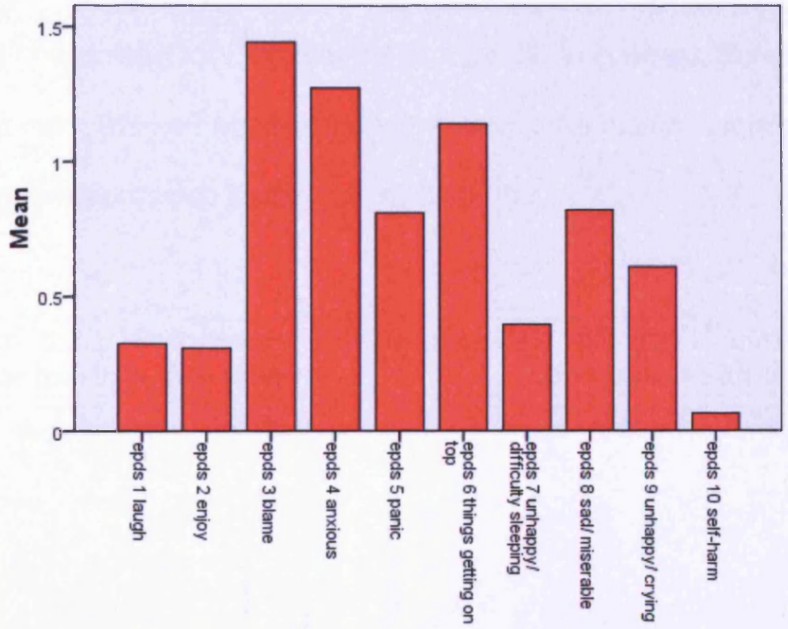
		Item 3 *Blame* (n) 759	**Item 4** Anxious (n) 759	**Item 5** *Panicky* (n) 759
EPDS anxiety sub-scale				
	Mean (SD)	1.44 (0.83)	1.27 (0.91)	0.81 (0.84)
	Median	2	1	1
	% of total score	20.0%	17.7%	11.3%

EPDS Item 7 unhappy/ can't sleep (n) 759	Mean (SD)	0.04 (0.73)
	Median	0
	% of total score	4.7%

EPDS Item 10 *suicide/ self –harm* (n) 759	Mean (SD)	0.06 (0.32)
	Median	0
	% of total score	0.9%

Figure 8.21: Bar chart shows the mean scores (score 1-3 points) for individual EPDS items at 1st screen, illustrating the higher mean score values for EPDS item 3, *blame* and item 4, *anxious*. (The bar chart illustrating complimentary data for the 2nd screen can be found on page 198).

Figure 8.21: Bar chart – EPDS mean score/ severity ratings (1st screen)

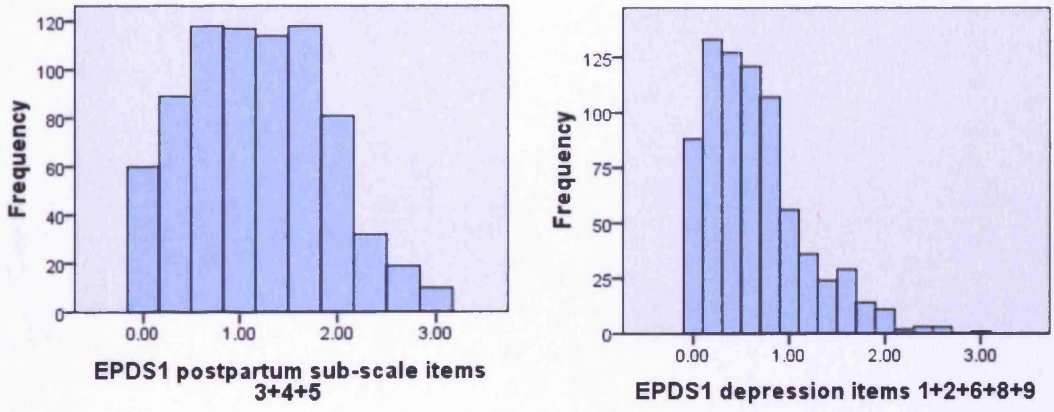


The EPDS 3 item *postpartum sub-scale* was found to contribute 49% to the total EPDS score (24.4% of the total variance) compared with the 5 item depression sub-scale which contributed 44.6% to the total EPDS score (27.9% of the total variance). The mean score for the postpartum sub-scale (M = 1.2 (0.71)) (95% CI's 1.12 -1.22) was higher than that for the depression sub-scale (M = 0.64, (0.52)) (95% CI = 0.60 - 0.68). A paired samples t-test confirmed the mean score difference between the sub-scales (mean = 0.53 (0.6)) was statistically significant (t (754) = 25.8, p<.001) (95% CI = 0.49 - 0.57). These outcomes suggest that, overall, the combination of depression and anxiety symptoms reflected in the

postpartum sub-scale exert greater influence in postpartum mood compared with pure depression.

Figure 8.22: Histograms showing the frequency of scores (1-3 points) for the EPDS postpartum and depression sub-scales (1st screen), with the item/ sub-scale composition demonstrating the outcomes for rotated PCA established in this study. The postpartum sub-scale data demonstrates a slight positive skew only and reflect the collective higher frequency/ severity ratings (1-2 points) for EPDS items 3, 4 and 5. In contrast, the depression sub-scale demonstrates a more marked positive skew, reflecting the higher frequency/ lower severity ratings (1 point) for depression items 1, 2, 6, 8 and 9.

Figure 8.22: Histograms - EPDS sub-scale ratings (1st screen)
postpartum sub-scale **depression sub-scale**



Outcomes for EPDS data at 2nd screen demonstrated some inconsistencies in ratings for individual EPDS items over time. EPDS item 3 *(blame)*, item 6 (*sad/ miserable*) and item 8 (*getting on top of me*) were most frequently endorsed with a score of one point (score 1-3) by 46.9% (n = 335), 45.8% (n = 327) and 41.8% (n = 299) of subjects, respectively. Similar to the 1st screen EPDS *postpartum sub-scale* items 3 (*blame*) and 4 (*anxious*) were rated at 2nd screen with a score of two points (score 1-3) by 32% (n =

229) and 31% (n = 222) of subjects, respectively, again representing the highest frequency/ severity ratings of the ten EPDS items.

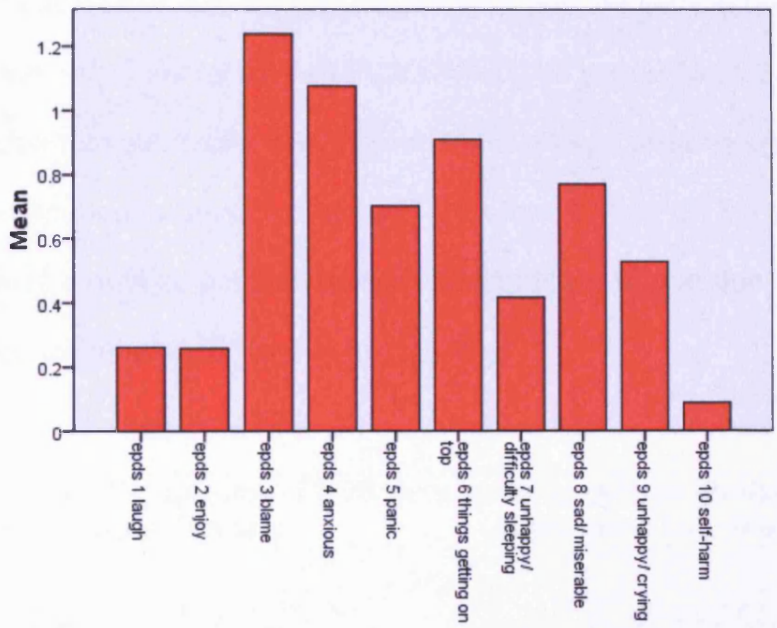
Table 8.2 (see pg. 197) reports the percentage contributed by each EPDS item (frequency/ severity) to total subject scores combined at 2nd screen; mean (SD) and median scores for items are also reported. Data are presented to illustrate the composition of EPDS sub-scales found in this study. Items 6, 8 and 9 do not contribute to any sub-scale; item 6 failed to discriminate between components whilst items 8 and 9 loaded on the anxiety sub-scale although did not influence its overall interpretation.

Table 8.2: EPDS items - percentage of combined total scores (4473) (2nd screen)

EPDS depression sub-scale		**Item 1** *Laugh as usual* (n) 714	**Item 2** *Enjoy* (n) 714	**Item 7** *Unhappy/ can't sleep* (n) 714
	Mean (SD)	0.26 (0.57)	0.26 (0.57)	0.42 (0.73)
	Median	0	0	0
	% of total score	4.2%	4.2%	6.7%
EPDS anxiety sub-scale		**Item 3** *Blame* (n) 714	**Item 4** Anxious (n) 714	**Item 5** *Panicky* (n) 714
	Mean (SD)	1.24 (0.84)	1.08 (0.89)	0.70 (0.86)
	Median	1	1	0
	% of total score	19.8%	17.2%	11.2%
EPDS items excluded from sub-scales		**Item 6** *Things getting on top* (n) 714	**Item 8** *Sad/ miserable* (n) 714	**Item 9** *Unhappy/ crying* (n) 714
	Mean (SD)	0.91 (0.76)	0.77 (0.76)	0.53 (0.68)
	Median	1	1	0
	% of total score	14.5%	12.3%	8.4%
EPDS Item 10 *Suicide/self-harm* (n) 714	Mean (SD)	0.09 (0.36)		
	Median	0		
	% of total score	1.4%		

Figure 8.23: Bar chart shows the mean scores (score 1-3 points) for individual EPDS items at 2nd screen, illustrating the higher mean score values for EPDS item 3, blame and 4, *anxious*.

Figure 8.23: Bar chart – EPDS mean score/ severity ratings (2nd screen)



At 2nd screen the EPDS *postpartum sub-scale* contributed 48.2% to the total EPDS score (33.1% of the total variance). On this occasion only three EPDS items comprised the *depression sub-scale*, contributing only 15.1% to the total EPDS score (27.1% of the total variance). EPDS items 6, 8 and 9, although excluded from the EPDS depression sub-scale, contributed 35.2% to the total EPDS score.

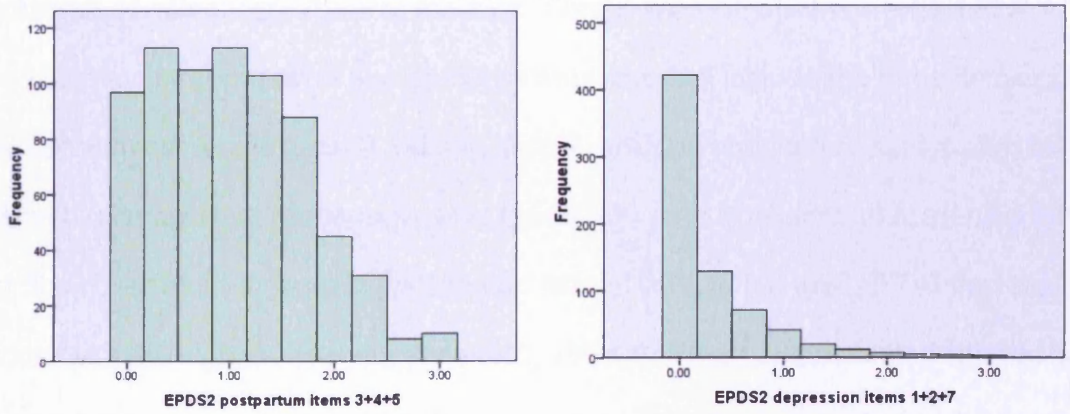
Similar to the 1st screen, the mean score for the postpartum sub-scale (Mean = 1.0 (0.72)) (95% CI = 0.95 - 1.1) at 2nd screen was higher than that for the depression sub-scale (Mean = 0.3 (0.53)) (95% CI = 0.27 - 0.35). A paired sample t-test confirmed the mean score difference between the sub-scales (Mean = 0.7 (0.58) was statistically significant (t (712) = 31.9,

p<0.001) (95% CI = 0.65 - 0.74). These outcomes again suggest that, overall, the combination of depression and anxiety symptoms reflected in the postpartum sub-scale exert greater influence in postpartum mood compared with pure depression.

Figure 8.24: Histograms showing the distribution of scores (1-3 points) for the EPDS postpartum and depression sub-scales (2nd screen), with the item/ sub-scale composition demonstrating the outcomes for rotated PCA established in this study. Similar to the 1st screen, the postpartum sub-scale data demonstrates a slight positive skew only, reflecting the collective higher frequency/ severity ratings (1-2 points). In contrast the depression sub-scale data demonstrates a marked positive skew, reflecting fewer depression items (1, 2 and 7) with higher frequency/ lower severity ratings (0-1 points).

Figures 8.24: Histograms - EPDS item severity ratings (2ndscreen)

postpartum sub-scale **depression sub-scale**



A paired sample test confirmed that the mean score difference for the postpartum sub-scale between at the 1st (M = 1.2 (0.71)) and 2nd screen (M = 1.0 (0.72)) was statistically significant (t (703) = 7.01, p<0.001) (95% CI = 0.13-0.22). Similarly, the difference in the mean scores for the depression sub-scale between the 1st (M = 0.65

(.52)) and 2nd screen (M = 0.31 (0.53) was also statistically significant (t (701) = 17.2, p = <0.001) (95% CI = 0.30-0.27).

8.4 Chapter summary: Composition of the EPDS total depression score

Percentage rates and frequency/ severity ratings for individual EPDS items were examined as a means of understanding the composition of the EPDS and the influence of EPDS depression and anxiety symptoms in characteristic postpartum mood (1st and 2nd screen). Data were reported according to outcomes for rotated PCA found in this study (see Chapter 6, pg. 114-138), reflecting the composition of the postpartum and depression sub-scales for both occasions of screening.

Findings for the percentage of ratings for individual EPDS items demonstrated a hierarchy of depression and anxiety symptoms that was consistent across the two episodes of screening. The five most frequently rated EPDS items reflected a combination of depression *and* anxiety symptoms and include the three items of the postpartum sub-scale (item 3, blame; item 4, anxious and item 5, panic), with blame rated by the greatest percentage of subjects. On both occasions of screening ratings for the postpartum sub-scale contributed almost 50% to the total EPDS depression score with the highest frequency/ severity ratings of the 10-item scale achieved by items 3 and 4. The mean (SD) values for the postpartum sub-scale was higher than the depression sub-scale on the two occasions of screening and over time with the difference meeting statistical significance (p= <0.001).

8.5 Conclusion

Outcomes highlight a number of contentions around the validity of the EPDS as a measure of *postnatal depression* as well as the content of non-pathological postpartum mood. In view of the fact that the EPDS total depression score is significantly influenced by ratings for items that represent discrete symptoms of depression *and* anxiety it cannot be considered as a single construct measure of depression. As such, EPDS outcomes should not be compared with other discrete measures of depression such as the BDI. Furthermore, specific symptoms of depression and anxiety measured by the EPDS are consistently organised to suggest a unified and stable mood state that may be characteristic of postpartum mood and therefore non-pathological in origin.

Findings for items 3 (*self-blame)*, 4 (*anxiety*) and 5 (*panic*), both individually and combined as the postpartum sub-scale, suggest that postpartum women are significantly affected by their experience of these symptoms. The significance of these features of depression and anxiety are reflected in findings for early parental preoccupations and harm avoidant behaviours described by Leckman et al (1999) with this position supported by hypotheses for their evolutionary purpose and utility (discussed in Chapter 4). Therefore it is likely that these symptoms are phenotypical features of childbearing. It must be considered that rates of EPDS postnatal depression, both in research and clinical practice, are augmented by *normative* features of depression and anxiety.

Chapter 9 to follow discusses the study limitations and strengths as well as the implication of study findings in relation to the wider evidence for depression and anxiety in relation to postpartum mood.

CHAPTER 9

DISCUSSION

9.1 Introduction

Postnatal depression, a clinical construct, represents the principal model of postnatal mental health. Until this study there has been no investigation into the rate and severity of symptoms of depression and anxiety as features of non-pathological and potentially *normative* postpartum mood. Conceptually, the study therefore represents a first line of investigation with exploratory data that is essentially descriptive. One disadvantage of this primary position is the relative lack of any existing literature upon which to draw regarding the question in hand. In response the current review has examined the wider evidence and hypotheses for depression and anxiety, beyond that for perinatal mental health. In combination these theoretical areas have served to highlight important discrepancies which exist between the literature for postnatal depression and that for concomitant depression and anxiety, and maternal attachment.

These discrepancies yield two broad areas of argument which support this examination into non-pathological, postpartum symptoms of depression and anxiety. The first is that, overall, the evidence does not support that childbearing incurs an increased risk for depression, compared with other times of life, bringing the validity of *postnatal depression* into question. The second is that specific features of depression and anxiety underpin normal maternal pre-occupations and bonding behaviours (see Leckman et al 1999), suggesting that they are conserved features of maternal-infant attachment.

The value of this study, which was founded in health visiting practice, lies is its conceptual orientation and reinterpretation of the quantitative findings for postpartum symptoms of depression and anxiety. As such the study challenges widespread beliefs, about the innate risk of depression associated with childbearing, and the pathological interpretation of the mental domain of maternal attachment. Equally the study raises important questions about the nature of normative postpartum mood. The combined outcomes propose a model for symptoms of depression and anxiety as normative features of postpartum mood that has not been previously considered. The contribution of this study, both in terms of research and practice, therefore needs to be measured against the realtive lack of a non-pathological, normative model of postpartum mood and what might be viewed as the pernicious influence of the postnatal depression industry upon the wellbeing of mothers and infants.

The following sections examine the limitations and strengths of the study, interpret the results in light of the widest evidence, present the study conclusion and identify implications for future research and practice.

9.2 Study limitations and strengths

A number of study limitations and strengths are recognised: Two broad areas of bias, notably *selection* and *information* bias (Vandenbroucke et al 2007) are considered to have potentially influenced the sampling error, that is, the difference between the study and population statistics (De Vaus 2002, pg 70 & 229). Firstly, the decision taken early on in the study to exclude ethnic minority groups for ethical reasons is seen to significantly increase the risk of selection bias, with outcomes only reflecting Caucasian, predominanlty English speaking postnatal women. This influence reduces

the representativeness of the sample and imposes the need for caution in applying study outcomes to the postnatal population as a whole.

Secondly, the risk of selection bias (Vandenbroucke et al 2007) increased both with the use of a pragmatic sample of convenience and a failure to achieve the estimated, optimum sample size (1,275 subjects). The latter was not anticipated at the outset of the study and was due to the participation of only fifty percent of available health visitors rather than high numbers of postnatal refusals. Even so, the postnatal cohort was large (n = 767) with participants recruited from the caseloads of generic health visitors, indicating that they were not dissimilar from the postnatal population as a whole, thus increasing confidence in the likely representativeness of study outcomes. In addition, rates of depression and anxiety symptoms at the level for risk of disorder found in this study were not dissimilar to widely reported rates for postnatal depression and perinatal anxiety (for example Gavin et al 2005; Stuart et al 1998) supporting that there was little difference between this and other samples. Consequently, it is unlikely that the subjects, who were recruited from a representative range of socio-economic divisions, differed in any significant way from the postnatal population in general. Significantly, this study ranks amongst the largest of prospective studies undertaken in the field of postpartum mood, although comparison can only be made with those studies that have investigated rates of *postnatal depression* and pathological anxiety.

An increase in the risk of *response* bias, a form of selection bias (Vandenbroucke et al 2007), is acknowledged in view of those eligible postnatal women who refused to participate. These were not observably different to postnatal participants although it was not possible to determine whether any significant difference existed between the

two groups regarding rates of clinical depression and anxiety. Even so it must be considered that eligible postnatal women who refused might represent a sub-group of high or low risk women, with their non-participation potentially influencing rates of depression and anxiety symptoms in either direction. Similarly, neither data for clinical history were gathered, nor were clinical interviews undertaken. As a result it was not possible to assess individual risk or compare the clinical status of subjects with quantitative outcomes. In turn, the use of self-rating questionnaires alone is recognised to potentially over estimate rates.

An important strength of this study, not widespread in studies investigating postnatal depression, is that prospective data were gathered from the same postnatal cohort during two screening intervals, spanning the third postpartum month. This facilitated investigation into change over time in (i) scale performance, (ii) the affective content of postpartum mood and (iii) the rate and severity of depression and anxiety symptoms. In particular, no other study could be found to have reported scale performance on two occasions in a postnatal cohort. Because of the transient nature of heightened postpartum symptoms of depression and anxiety a closer inspection of how the measures performed over time was considered necessary. This analysis provided a valuable insight over time, into the performance of the EPDS and the affective content of postpartum mood.

The omission of social data such as educational attainment, marriage and employment status represents a significant study limitation and increases the risk of *information bias* (Vandenbroucke et al 2007). Without such data it has not been possible to fully demonstrate the representativeness of the sample. However, the high

level of subject and health visitor participation, as already described, provides considerable support for the representativeness of the postnatal sample.

In summary, the study limitations, broadly categorised as selection and information bias (Vandenbroucke et al 2007), impose the need for caution in terms of estimating the generalisability of study outcomes. However, the large sample size with little variation in the key variables such as postnatal status, maternal age range and number of children reflected the female childbearing population, and adds weight to the representativeness of the findings.

9.3 Interpretation of results

The principal aim of this exploratory study was to investigate a characteristic distribution of *symptoms* of depression and anxiety in postpartum women (n = 767), recruited from the caseloads of generic health visitors (n = 124). In efforts to address the research questions three discrete areas of literature were identified that were considered pertinent to the mental domain of postpartum women.

These were

- Theme One: Mothers and risk; from physical morbidity to mental health.
- Theme two: Investigation into depression and anxiety; how it relates to women and postpartum mood.
- Theme Three: Symptoms of depression and anxiety as normative features of postpartum mood.

These theoretical areas have not previously been linked in relation to postpartum mood and their association introduced a dimension to this work that was not anticipated at the outset. This unexpected dimension is manifest in the synthesis of

the literature review areas and serves not only to strengthen the rationale for this study but also forcefully undermines any argument for the existence of a discrete disorder labelled *postnatal depression.* This synthesis is therefore considered as a study outcome in its own right; interpretation of the study results appropriately commences with the literature review synthesis and is followed by a discussion of the outcomes for anxiety and depression found in this study.

9.3.i Synthesis of the literature review

There is a scientific fervour for postnatal depression which escalates perceptions of risk around childbearing both in the lay and scientific communities. However, scrutiny of the relevant literature indicates that the phenomenon of postnatal depression is characterised by contradictions. On one hand it is reported that no difference exists between depression in postpartum and non-childbearing groups (e.g. Kumar et al 1982; Cooper et al 1988, O'Hara 1990; Whiffen 1992; Whiffen and Gotlib 1993; Augusta 1991; Eberhard-Gran 2002); clinical depression detected in postpartum women is therefore a matter of recurrence, pre-existing or first onset and is mediated by pre-existing individual risk. On the other hand, factors such as widespread screening and the reporting of postnatal depression as a discrete disorder are proposed to substantiate its status as a clinical diagnosis. Between these two perspectives lie a number of latent considerations that arise from the wider evidence for depression and anxiety. These are addressed here and provide the critical lens through which to consider the veracity of postnatal depression versus the content of *normative* postpartum mood.

Reporting the prevalence of depression and anxiety as single disorders, particularly based on cross sectional data, inevitably excludes important findings. Such exclusions restrict understanding of the nature of the inextricable relationship between these mood states and of the sex linked risk. In contrast, it might be said that concomitance studies, which have investigated temporal sequence and co-existence in depression and anxiety, potentially offer more reliable and meaningful data. In any case an argument exists for a fundamental need to accurately identify incidence versus prevalence in depression and anxiety in order to associate life events with first onset and recurrence in these disorders (Lapouse 1967: Murphy et al 1988; Kalidini and McGuffin 2002).

One implication of using cross sectional data in relation to perinatal mental health is that decisions about first and episode onset in depression and anxiety, based upon diagnosis, help seeking behaviour and self-report, are unreliable. Furthermore, a firm distinction between incidence and prevalence becomes essential when attributing risk of psychiatric disorder to life events such as childbirth (see Lapouse 1967; Kalidini and McGuffin 2002). Consequently, reports of the *incidence* of postnatal depression (Cooper et al 1988; Hannah et al 1992; Cox et al 1993; Yamashita et al 2000; Gavin et al 2005) require cautious consideration. *Ipso facto, existing, very long term epidemiological studies do not support that the postnatal period is associated with increased risk for pathological depression or anxiety.*

Broadly, the relevance of *incidence* is more than merely the onset of pathological depression and/ or anxiety. Whilst the accurate detection of the first episode of disorder represents the margin between mental health and illness, it also provides a fundamental marker of the relationship between these two mood states. Findings for

the temporal sequence in which anxiety typically precedes depression in first and episode onset, adds clarity to the notion of sex-linked risk. Notwithstanding findings for increased rates of depression in those born after World War II, with a two fold increase in women (Cross National Collaborative Group 1992; Murphy 2000), the female/ male risk for anxiety (2-3:1) and depression (2:1) assumes a different nuance taking into account that men and women are reported to suffer similar risk for depression following *any* anxiety disorder (Murphy et al 1988; Breslau et al 1995).

It is significant then, in addressing the question of postpartum mood that the higher rate of depression onset in women emerges, in the main, as a result of the female propensity to anxiety. More precisely, anxiety rather than female sex has been shown to incur the greatest risk for depression onset (Breslau et al 1995; Parker et al 2001). To qualify this further, the absolute rarity of depression in the absence of a history of pathological anxiety, whether or not this was diagnosed, tells us that anxiety is a significant diathesis factor in depression (Murphy et al 1988, 2000, 2004; Breslau et al 1995; Parker et al 2001; Kovacs 1990). Findings for the greater frequency of co-existing symptoms of depression and anxiety compared with single disorders, particularly below the threshold for disorder, must therefore come as no surprise (Zinbard et al 1994; Stein et al 1995; Wittchen and Essau 1993; Von Korf et al 1987; Angst et al 1997; Brown et al 1996; Roy-Byrne et al 1994; Zung et al 1990; Helmchem and Linden 2000).

The evolutionary rationale for prevalent features of depression and anxiety make the female primary propensity to anxiety an intriguing basis for exploring non-pathological postpartum mood. However, *symptoms* of these mood states are seen to be equal to disorder with their normative potential overlooked. Furthermore, the hierarchical

dominance of depression over anxiety (Green 1998), which is suggested to have induced a general lack of scientific interest in anxiety compared with depression, has served to inhibit enquiry into perinatal anxiety (Pigott 1999; Ross 2006). One possible reason for this lack of interest into anxiety disorders in the perinatal period is that there is no anxiety equivalent to postnatal depression. Another reason might be that the aetiology of anxiety compared with depression, is considered less clear (Kendler 1992, 1996, 1999). Regarding depression, it is recognised that traumatic experiences in early childhood, particularly emotional deprivation and maltreatment, increase individual risk in adulthood (Heim et al 2000; Heim and Nemeroff 2001; Gunnar et al 2002; Gunnar and Donzella 2002; Tarulloa and Gunnar 2006). However, it is reasonable to propose that these formative, adverse influences can be interpreted to represent protracted experiences of anxiety that influence H-PA reactivity and lead to clinical anxiety and depression later in life.

Exploration of recent postnatal depression literature suggests that it is an area of investigation that is in transition. There is increasing attention to the prominence of anxiety as a specific feature of postnatal depression compared with depression at other times of life. Anxiety detected in postnatal women is considered only from a pathological perspective and is seen as the *key* to postnatal depression as a differential diagnosis to general depression (Ross et al 2003; Matthey 2003). This interpretation reflects a worrying theoretical trajectory: Given evidence that suggests subjective anxiety is a *normative* and essential feature of maternal attachment (Feldman 1999; Leckman 1999), investigation into maternal anxiety from the perspective of pathology is likely to yield high rates with increased severity compared with the non-childbearing population. In addition, Ross (2003) correctly reports that

anxiety acts as a risk factor for depression noting only that *the relationship between depression and anxiety in women* explains the higher rate of female anxiety. This represents an example of how the perception of risk associated with women and childbearing is escalated by a failure to clarify that a history of pathological anxiety incurs a similar risk for depression in both men and women.

Returning specifically to the concept of postnatal depression, scientific interest is proposed here to be motivated by three factors; first, is the challenge provided by the ambiguity of the postnatal depression phenomenon. Second, are the consistent findings to support that women exhibit heightened features of depression (and anxiety) in the first three months postpartum, following which rates of postnatal depression are reported to diminish (Gavin 2005; O'Hara 1984; Pitt 1985; Margison 1987; Muzik 2000; Matthey 2003; Ross et al 2003). Third, is the tacit and widespread belief that symptoms of depression and anxiety in the puerperium are abnormal because childbearing in affluent societies is viewed as a singularly life enhancing experience;

'For many couples the event of childbearing is a joyous and memorable occasion. However, for some women, the postpartum experience may not always be positive or pleasant, as a significant number will develop postpartum depression and/ or anxiety' (Stuart et al 1998).

The concept of postnatal depression seems to have developed in response to the detection of transient symptoms of depression and anxiety in the first three months after childbirth. These are measured against diagnostic criteria for disorder (DSM-1V APA 1994; ICD-10 WHO 1994) without any regard for the biologically unique and evolutionary sensitive postpartum period that is more than a life enhancing event.

9.3.ii Performance of the measurement scales in the postnatal sample

Quantitative data for symptoms of depression and anxiety were gathered using the 10 - item EPDS (Cox et al 1987), 21 - item BDI (Beck et a 1961) and 21 - item BAI (Beck et al 1987) between 4 and 8 (1st screen) and 12 and 16 (2nd screen) postnatal weeks, completed by 98.8% and 92.9 % of participants, respectively. High internal reliability was confirmed for the three measures. Outcomes for rotated PCA showed that, similar to their validation status in psychiatric samples, the BDI (Beck et al 1961) and BAI (Beck et al 1988) consistently performed as discrete measures of depression and anxiety, respectively, in the postpartum cohort.

In contrast to the single construct status of the BDI and BAI the EPDS, at face value, includes two anxiety items, challenging its validated status as a discrete measure of postnatal depression (Cox 1987). Moreover, the EPDS two and three component models investigated in this study (1st and 2nd screen) confirmed the presence of two sub-scales measuring discrete depression and combined anxiety and depression, replicating the findings of Brouwers et al (2001) and Ross et al (2003). These two EPDS sub-scales have previously been defined to represent pathological depression and pathological anxiety (Brouwers et al 2001; Ross et al 2003).

In particular, Ross et al (2003) argues that the presence of the EPDS *anxiety sub-scale* provides *'...important evidence for postpartum depression as a specific diagnostic construct distinct from major depression'*; Brouwers et al (2001) draws attention to ratings for EPDS item 5, *'panic...for no good reason'* as possibly revealing low self esteem associated with depression. Findings for the anxiety items/ sub-scale in women at 24 weeks gestation (Brouwers et al 2001) and 6 weeks postpartum (Ross et al 2003) have been further interpreted to confirm that the internal structure of

the sub-scale is constant across the perinatal period (Ross et al 2003). The implication here is that the relationship between anxiety and perinatal psychopathology is more strongly affirmed because this sub-scale is constant.

In this study the *anxiety sub-scale* (Brouwers et al 2001; Ross et al 2003) has been redefined as the *postpartum sub-scale*. This revision was necessary because the sub-scale is not a discrete measure of anxiety as it is comprised of one depression item (item 3-*blame*) and two anxiety items (item 4-*anxious*; item 5-*panic*). EPDS item 3-*blame* (self reproach/ guilt) is a classified symptom of depression rather than anxiety (APA 1994; WHO 1994; Gilbert 1997) with this fact neglected in previous studies (Brouwers et al 2001; Ross et al 2003). However, the distinction highlights how scale items/ component loadings are open to subjective interpretation rather than representing a measure of any concrete construct beyond the content of the total scale. Some understanding of this potential for variation in how item/components are interpreted is important because inappropriately interpreting the sub-scale content can be misleading not least with regards to the proposed role of anxiety as a diagnostic characteristic of postnatal depression.

In this study the consistent relationship between items 3, 4 and 5 over time was also considered to provide good support for the stability of the EPDS *postpartum sub-scale*. This stability was further emphasised by the changing structure of the depression sub-scale, with fewer item loading on the sub-scale at the 2nd screen (items 1-*laugh*, 2-*enjoy* and 7-*unhappy/ difficulty sleeping*) compared with the 1st screen (items 1- *laugh*, 2-*enjoy*; 6-*things getting on top of me*, 8-*sad/ miserable* and 9, *unhappy/ crying*). Items 8 and 9 loaded on the postpartum sub-scale at 2nd screen although their lower loadings, compared with items 3, 4 and 5, did not overall

influence the interpretation of the postpartum sub-scale. At the 2nd screen item 6 failed to discriminate between the postpartum subscale and the third EPDS component. Consequently, item 6 did not contribute to the interpretation of any sub-scales on this occasion of screening.

The change over time in EPDS, BDI and BAI component/ item loadings demonstrates that scale performance was not entirely consistent in the postnatal cohort. This finding was viewed as more significant for the EPDS because of its mixed depression/ anxiety status, which influenced the interpretation of outcomes in a way that was not possible for the BDI and BAI as single construct measures. Although the change in scale performance does not provide new information about the content of postpartum mood over time, beyond that which was measured on the first occasion, it is never the less a response to some change in the collective mood state of the sample. In keeping with the contention of Bergin and Lambert (1978, p 172) a critical distinction must be made between the underlying construct of a scale and the limits of the information that can be provided about what is being measured. This point is perfectly illustrated by the percentage of women who rated items 6 (1st screen = 83.9%; 2nd screen = 68.2%) and 8 (1st screen = 65.5%; 2nd screen = 60%), which were ranked in the top five most frequently rated on both occasions of screening, indicating that these symptoms were significant in postpartum mood. However, technically they failed to influence the overall interpretation of EPDS component/ sub-scales at the 2nd screen.

No data was available with which to properly compare these findings as neither Brouwers et al (2001) or Ross et al (2003) reported item/ component loadings for the depression sub-scale at different times in the perinatal period. PCA outcomes for the

EPDS established in this study are interpreted to suggest that the stable relationship between a subjective symptom of depression (*blame)* and a subjective symptom of anxiety (*anxious)* might be meaningful in non-pathological and potentially *normative* postpartum mood. However, comparable data for non-childbearing groups could not be found and therefore it is uncertain whether findings for the EPDS sub-scales represent an artefact of the total scale or reflects some relationship between subjective symptoms of depression and anxiety that is specific to perinatal women.

Comparison of PCA outcomes for the BDI between the postnatal cohort in this study and the psychiatric sample investigated by Steer et al (1987) highlight some differences that may reflect the specific nature of postpartum mood, compared with clinical depression. In particular, BDI item 10 (crying) failed to load adequately on any component according to Steer et al (1987), compared with the high loading (0.52) achieved at both occasions of screening in this postnatal cohort. This outcome may reflect crying as a feature that is typically associated with early postpartum mood, whilst indicating that it is a lesser feature of clinical depression. From an evolutionary perspective crying is proposed as a defence mechanism as it signals the need for help (Lewis et al 1934) and increases in response to a lack of social support (Keller and Nesse 2005). Crying is also shown to solicit empathy and comforting behaviours from others (Labott et al 1991; Cornelius and Labott 1997) whilst serving to strengthen social bonds (Frijda 1986). In the postpartum context crying is therefore likely to confer adaptive benefits aimed at promoting the survival of the vulnerable mother/ infant dyad.

In hindsight, use of the BAI was probably not the most appropriate scale to measure anxiety in the postpartum sample. Cox (1996) suggests that BAI items are heavily weighted towards panic symptoms rather than measuring more general dimensions of anxiety and this was the view held in this study. In turn, the consistent contribution of EPDS item 5, *panic,* to the postpartum sub-scale is relevant as it is has been shown to be a prevalent feature of normative primary maternal preoccupations (Leckman 1999). As such it does not signify a disorder, whereas in the context of the EPDS item 5 is intended as a measure of pathology.

9.3.iii Symptom rates, co-existence and change over time

The opportunity to explore postpartum symptoms of depression and anxiety with a normative bias was enhanced by two features of this study; screening the same postpartum cohort on two occasions and the application of operational score categories. Screening the same cohort on more than one occasion is not a frequent feature of studies investigating postnatal depression although it is recognised to be necessary (Stuart 1998). In turn the application of operational categories facilitated interpretation of total scores across the score range of the measures used.

Analysis for *Research Question 1.1 and 1.2* examined point and period rates for postpartum symptoms of depression and anxiety, co-existence and change over time. As the topic under investigation is the rate and severity of symptoms below as well above the cut-off for risk, with the interpretation non-pathological, there is little to be gained from emphasising rates for postnatal depression and anxiety. Suffice to say that the percentage rates and total mean (SD) score values for the EPDS, BDI and BAI are comparable with findings from other studies (Cox et al 1987; Stuart et al 1998; Gavin 2005) suggesting that outcomes below the criteria for risk found in this

study might also be comparable. However, no study was found to have examined total scores for these measures by categorising rates and symptom severity across the total score range, therefore there are no reports to confirm this.

Findings for *Research Question 1.1* showed total scores for the EPDS, BDI and BAI at single time points confirm that the majority of subjects rated some symptoms of depression and anxiety across the total score range, with the greatest percentage in the sub-clinical score categories. Period rates for the BDI and BAI were found to demonstrate a similar distribution of total scores. In contrast, the EPDS period rate was dominated by the sub-threshold score category (42.2%) and was influenced by the consistent higher ratings for EPDS items 3 (*blame*) and 4 (*anxious*).

Some findings for anxiety and depression established in this study are suggested to be artefacts of the measures used in relation to the postnatal cohort and are relevant in their interpretation. For example, high BAI total scores might well have been anticipated in response to the relatively high ratings for EPDS item 4 (*anxious*). The fact that this was not borne out is likely to reflect EPDS item 4 as a measure of subjective anxiety rather than somatic anxiety. Although the BAI has been validated as measure of *subjective* and *somatic* anxiety (Beck et al 1988) there is some justification for considering that it is weighted towards the neurophysiological and cognitive dimensions of anxiety more closely associated with panic (Cox et al 1996), which is not commensurate with the maternal anxiety that is expressed in early parental preoccupations and behaviours (Leckman et al 1999). Even so, the consistent contribution of EPDS item 5 (*panic*) to the postpartum subscale, with lower ratings compared with items 3 (*guilt*) and 4 (*anxious*), reflect parental reports of guilt also associated with primary preoccupations (Leckman et al 1999).

The decision to investigate co-existing, postpartum symptoms of depression was based on their reported frequency at the diagnostic threshold for disorder (O'Hara et al 1990; Cooper et al 1988; Hannah et al 1992; Stuart 1998; Yamashita et al 2000; Brouwers et al 2001: Ross et al 2003; Misri et al 2000; Gavin 2005). Previous studies that have investigated depression and anxiety in the same postpartum subjects provide limited insight into co-existence because they tend to report rates as discrete symptoms at the threshold for disorder rather than rates of co-existence (Williams and Koran 1997; Misri et al 2000; Wenzell et al 2001). Estimates for co-existing symptoms of depression and anxiety in the postpartum sample suggest that the picture is similar to that for the non-child bearing population, with rates below the level for risk of disorder the rule rather than the exception (Zinbard et al 1994; Stein et al 1995; Wittchen and Essau 1993; Von Korf et al 1987; Angst et al 1997; Brown et al 1996; Roy-Byrne et al 1994; Zung et al 1990; Maire 1997; Helmchem and Linden 2000; Rapport 2001).

Throughout this study outcomes for the EPDS and BDI have not been reported to represent comparable measures of depression in view of the fact that the former reliably measures anxiety *and* depression. In short, the overload of EPDS data can be argued to provide widespread evidence for co-existing symptoms of depression and anxiety rather than rates of *postnatal depression*. In response, only combined outcomes for the BDI and BAI, according to the operational score categories, were examined to assess rates of co-existing symptoms of depression and anxiety. Similar to point and period rates for the BDI and BAI the majority of subjects rated co-existing BDI/ BAI symptoms with the greatest percentage in the sub-clinical score category

(1st screen = 36.9%; 2nd screen = 36.9%), followed by the sub-threshold and risk categories. No meaningful relationship was found between maternal age and number of children upon rates of symptoms of depression and anxiety at the level for risk of disorder, suggesting that these factors exert little influence in the development of clinical disorder postpartum.

Thus, overall, it is clear that characteristic postpartum mood is not represented by the complete absence of symptoms (i.e. normal = zero). More precisely it is typical for postnatal women *without* enduring, clinical depression and anxiety to experience some features of both.

Research Question 1.2 investigated change in EPDS, BDI and BAI scores over time as a means of understanding how the frequency/ severity of postpartum symptoms of depression and anxiety changed. Outcomes demonstrated a statistically significant reduction in rates of depression and anxiety symptoms between the 1st and 2nd episode of screening ($p = <0.01$). These findings reflect reports of a peak in rates of *postnatal depression* during the first three months postpartum which fall thereafter until the 6th month (Gavin 2005). This spontaneous remission in features of anxiety and depression can be interpreted to suggest an increase/ decrease pattern which invites an adaptive interpretation that is associated with maternal-infant attachment.

In contrast to change over time in total scale scores (EPDS, BDI and BAI), total scores within the categories for *risk of disorder* failed to demonstrate a statistically significant change ($p = >0.05$), with higher mean (SD) risk scores at 2nd screen. Closer inspection of data for the change in risk scores for all three measures revealed that similar proportions of subjects (a) improved to non-risk levels (b) deteriorated to

the level for risk, or (c) remained unchanged. Importantly, the sub-group of subjects with enduring symptoms at the level for risk (c, above), are most likely to represent those with psychopathology, with estimates of prevalence comparable to those for clinical depression in the general population (for example, Maire et al 1997). These outcomes suggest a striking difference to the widespread reporting of an overall reduction in prevalence rates for postnatal depression after the third month post partum (Cox et al 1987; Stuart et al 1998; Gavin et al 2005) and warrant some consideration.

The discrepancy between outcomes for this study and others (for example Cox et al 1987; Stuart et al 1998; Gavin et al 2005) highlight the technical influence of research methodologies upon estimates for postnatal depression and how these are interpreted. Therefore, one reason for this difference in prevalence is likely to be that the reported reduction in *postnatal depression rates* are based on reductions in total mean (SD) scores and the percentage of risk scores, over time (see Gavin et al 2005), i.e. it is purely statistical. Outcomes for this study support that the *'at risk' groups*, according to measures used are not homogenous, over time. Consequently, these findings challenge the veracity of postnatal depression rates and consequently it's existence as a discrete disorder. Further, outcomes for the risk group add weight to the inadequacy of assessing clinical depression and anxiety at single time points and suggest that this practice is unsafe when used to substantiate an increased risk of psychopathology for childbearing women.

9.3.iv EPDS depression and anxiety and the influence of the postpartum sub-scale

Investigation into the performance of the EPDS and how individual items are rated provide a valuable insight into postpartum mood. Outcomes for *Research Questions 2.1ands 2.2* showed that postnatal subjects consistently rated items 3, *blame; item* 6, *things getting on top of me;* item 4, *anxious;* item 8, *sad or miserable* and item 5, *panic* (score 1-3 points) in a hierarchy of EPDS depression and anxiety symptoms that was stable over time. Notably, the EPDS postpartum sub-scale (*blame; anxious; panic*) was included amongst these five most frequently rated items of the10-item scale.

Despite the emphasis placed upon EPDS item 3 (*anxious*) by Ross et al (2003), as justification for postnatal depression as a discrete disorder, it is significant that EPDS item 3, *blame*, a subjective symptom of depression (APA1994; WHO 1994), was rated by the greatest percentage of postnatal women at the first (85.9%) and second (79.9%) screening intervals. Two possibilities for these substantial rates must be considered; either they support that maternal *blame* acts as a marker of pathology, in which case it would not be unreasonable to expect much higher rates of clinical depression in postnatal women, or it is a feature of characteristic and potentially normative postpartum mood, similar to subjective anxiety.

On both occasions of screening depression item 3 (*blame*) and anxiety item 4 (*anxious*) were rated by the greatest percentage of subjects with a score of 2 points, representing the highest frequency/ severity level, compared with the remaining EPDS items. Ross et al (2003) found that the anxiety sub-scale score *changed in parallel* with the diminishing EPDS score between 6 and 16 weeks postpartum '*rather than showing a unique pattern of change*'. A similar pattern of change over time was

observed in mean score values for both the postpartum sub-scale and depression sub-scale in this study, with the difference statistically significant. However, it was found that the frequency/ severity ratings for the postpartum sub-scale remained elevated and relatively stable, compared with the depression sub-scale, accounting for almost 50% of the EPDS total score on both occasions of screening. These outcomes were somewhat higher than the 38% at 6 postpartum weeks reported by Ross et al (2003) who did not report equivalent data at 16 postnatal weeks.

Overall, findings for the EPDS demonstrate that the postpartum sub-scale in its entirety and items item 3 (*blame*) and *item 4* (anxiety) in particular, exert a disproportionate influence in the postnatal period, compared with other EPDS symptoms. No studies could be found to have considered *symptoms* of depression and anxiety as non-pathological features of postpartum mood and maternal attachment. However, findings for the postpartum sub-scale are replicate Leckman et al's (1999) model of EPPB and AITHAB with high rates of parental anxiety, guilt (*blame*) and panic. From an adaptive perspective the specific function of blame, subjective anxiety, and panic, as features of depression and anxiety, arguably underpin EPPB and AITHAB, and provoke in parents harm avoidance (Keller and Nesse 2005), hyper vigilance and physiological arousal that promotes maternal responsiveness (Marks 1987). Therefore, in the context of childbearing, the primary purpose of the *symptoms* that comprise the EPDS postpartum sub-scale, acting in concert, is almost certainly normative.

Because of the deleterious impact of maternal psychiatric disorder on infant development and health one challenge to this assertion arises in the need to clarify the distinction between psychiatric versus normative maternal mood states

postpartum. This distinction is best understood through the influence of maternal preoccupation, which broadly is defined as a state of narrowed attention, with recurrent intrusive thoughts that are difficult to control or dismiss (Stein 2009). The repertoire of EPPB and AITHAB exhibit maternal preoccupations that are infant focused and therefore *normative and biologically protective* (Leckman et al 1999). EPPB promote healthy synchrony in the mother - infant relationship that is essential for the regulation of infant neurobiological systems, leading to optimum social and cognitive development in offspring (Feldman 2007).

In contrast, maternal preoccupations that are characteristic in a range of psychiatric disorders are implicated in the transgenerational transmission of mother related risk (Stein 2009). These psychiatric preoccupations are associated with narrowed internal or self-focused attention (Ingram 1990; Mor 2000) that distracts attention of away from the infant, reducing maternal responsiveness and sensitivity to infant cues and disrupts healthy synchrony in the mother-infant dyad (Stein 2009).

Diagram 9.1 (pg. 238) shows a model that incorporates features of depression (D) and anxiety (A) postpartum, based on findings for the EPDS postpartum sub-scale and how these might be organised in non-pathological and normative maternal mood. N*ormative* maternal anxiety (1) and *blame* (2) emerge in an attentional bias that directs the mother's cognition and behaviours towards the infant. Panic is likely to promote maternal responsiveness through action taken, such as *checking behaviour (Leckman et al 1999;* Feldman1999), to relieve the emotional discomfort associated heightened physiological arousal.

Model of Postpartum Mood

(Normative symptoms of anxiety (1 and 3) and depression (2, 4 and 5))

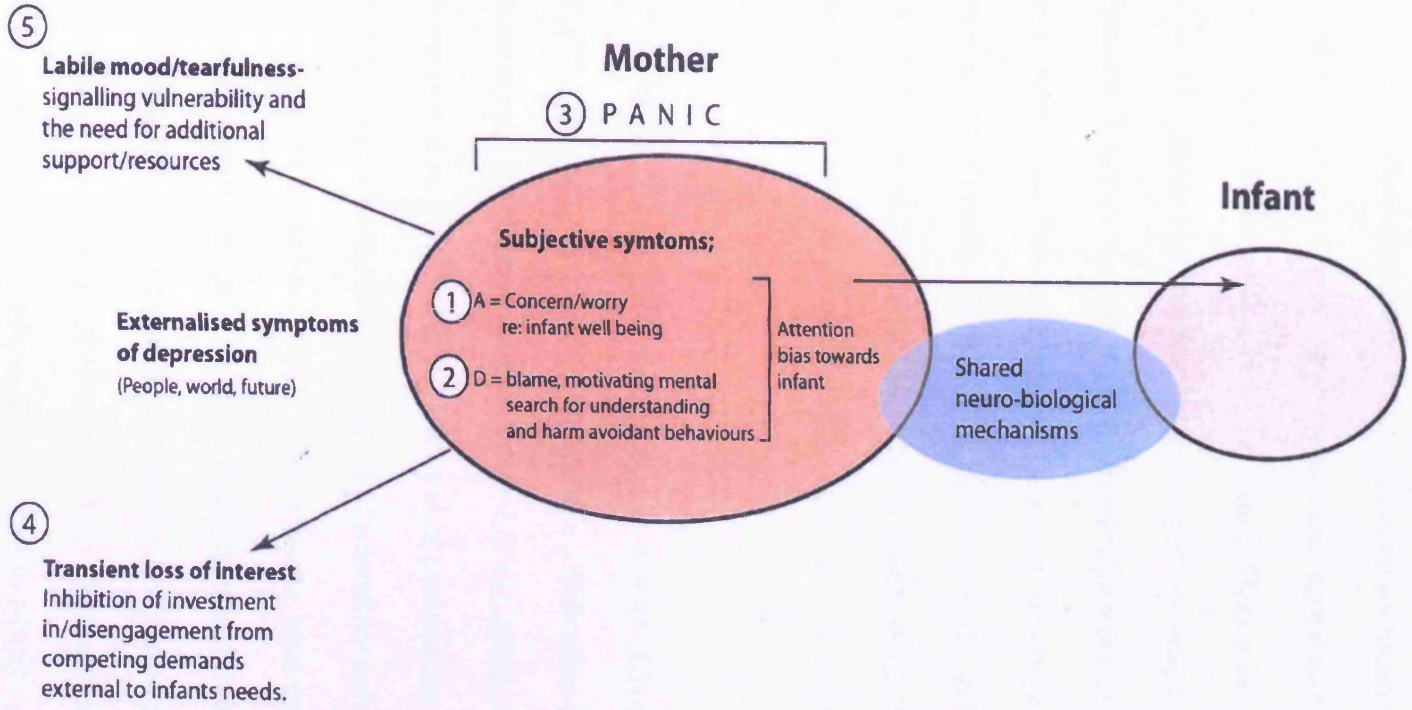


Diagram 9.1

Based on the impact of preoccupation on maternal cognition and attentional processes (Stein 2009), this rationale is extended to suggest how other symptoms of depression, specifically labile mood and loss of interest in mothers, might also be functional: *Normative* maternal preoccupation that is infant focused is likely to inhibit or disengage maternal attention and investment away from external demands that might otherwise compete with the needs of the infant. Thus a transient loss of interest in the external world is potentially normative in postpartum women rather than a symptom of disorder. Equally, tearing (4) in the postnatal period might best be explained by the association between elevated levels of serum prolactin postpartum and the proposed utility of crying (Eugster and Horsten 2001). In the context of childbearing, rather than a symptom of disorder, the purpose of crying is likely to signal maternal need for additional support and resources (Eugster and Horsten 2001).

In conclusion, findings for the heightened and stable relationship between EPDS *blame*, *anxiety* and *panic* are proposed to offer some justification for considering that their primary purpose is (1) adaptive (Nesse 2000; Gilbert 2006) and therefore, (2) principally normative and non-pathological features of postpartum mood that are aimed at promoting maternal-infant attachment and ultimately survival (Winnicott 1956; Leckman et al 1999; Feldman 1999). These specific features of anxiety and depression can be considered as phenotypical expressions of childbearing and suggest that the mental domain of postnatal women is not rooted in clinical concepts of depression. *In light of Leckman et al's model (1999) for early parental preoccupations and behaviours (EPPB) outcomes for the EPDS found in this study raise important questions about what total scale scores for depression and anxiety*

truly represent. For example, it is apparent that the EPDS measures normative symptoms of anxiety and depression in relation to EPPB (Leckman et al 1999). However, because the scale is a measure of postnatal depression these normative symptoms are interpreted in the context of pathology and have possibly served to augment rates of EPDS postnatal depression.

9.4 Conclusion

Outcomes for this study cannot be interpreted to confirm a normative pattern of postpartum mood. However, this thesis proposes that there are two competing concepts at play regarding the mental domain of maternal postpartum experience that have so far not been differentiated. One is pathological and secondary to individual risk; the presence of clinical depression, typically chronic and recurring, detected in the postnatal period, the origins of which are historical. The other is normative and transient; a phenotypical phenomenon so far labelled *postnatal depression* that is composed of specific dimensions of depression and anxiety, which may or may not reach the threshold for risk of disorder.

The phenomenon of postnatal depression has arguably developed in response to the detection of heightened features of depression and anxiety in the first three months after childbirth. It is also possible that a perception of heightened risk for maternal mood disorder postpartum has arisen because evidence for infant risk from maternal psychiatric disorder has been investigated largely in relation depression. Rather than signifying any causal relationship between childbearing and depression this position reflects the prevalence of depression in general. The postnatal experience of self-blame, anxiety and panic, features of depression and anxiety that comprise the EPDS postpartum sub-scale and are associated with primary maternal preoccupations, have

been interpreted according to diagnostic criteria for disorder (DSM-1V APA 1994; ICD-10 WHO 1994) without any regard for the neurobiologically sensitive and evolutionary critical postpartum period. The DSM-1V (APA 1994) comments on the need to consider the context of symptoms before arriving at a diagnosis although this directive is poorly applied to postpartum women.

Evidence from long term psychiatric epidemiologic studies and psychological investigation suggest three important factors relevant to the mental domain of postpartum maternal experience. The first factor is that childbearing is not shown to be an event associated with an increased risk of depression onset in women. The second factor is the primary female propensity to anxiety rather than depression is likely to underpin primary maternal preoccupations in response to childbearing. The third factor is that a better understanding of anxiety is likely to confer an important advantage from which to consider individual risk for the onset of depression at any time of life.

Despite the proposal for discrete pathological and normative dimensions of postpartum maternal mental health, the two are inextricably linked. The key is *attachment.* Consequently, one fundamental factor in reducing individual risk for pathological anxiety and depression at any time of life must be an understanding of the content and purpose of *normative* postpartum mood as the basis for supporting maternal-infant attachment. This is necessary in order to minimise the deleterious impact of (1) of findings that are interpreted to suggest the higher risk of psychopathology in postpartum women; (2) the application of inappropriate parenting regimes for mothers and infants and (3) the long term sequelae of pathological

anxiety and depression in those who experience relative trauma in infancy and early childhood.

9.5 Implication for future research

As a matter of urgency future research must be directed at efforts to develop a cohesive model of *normative maternal attachment* that considers a primary, non-pathological role for postpartum features of depression and anxiety.

A unified scientific approach is required that involves epidemiological, psychological and neurobiological investigation with greater use of control groups for comparison. Large studies are needed that include a more representative mix of racial and ethnic groups, utilising consistent definitions, methodologies and diagnostic criteria. Further, exploration of the frequency and severity of depression and anxiety symptoms below the threshold for risk of disorder is required to confirm their distribution in a characteristic pattern of postpartum mood. Quantitative and qualitative investigation is needed to explore the mental domain of normative maternal attachment and the relationship between specific features of depression and anxiety in the postpartum period. In conjunction, extensive investigation using functional MRI is required to evidence changes in maternal neurobiology in response to childbearing and infant stimuli. In all areas strict perinatal time frames must be applied to demonstrate any association between features of maternal mood and early patterns of attachment in the mother/ infant dyad. With this in mind outcomes for the antenatal period, first three months postpartum and later in the first year after childbirth, cannot be compared.

9.6 Implications for practice

Findings from this study point to the existence of specific features of depression and anxiety in the postpartum period, which so far are typically interpreted according to pathological mood states. These features might be described as characterizing a period of primary maternal preoccupation that is consistent with evolutionary theory, about the role of low-level and/ or transient symptoms of this nature. Service providers need to ensure that those professionals involved with childbearing women and infants receive appropriate education and training in relation to *normative postpartum mood* as the primary model of maternal mental health. Promoting *normative* postpartum mood will serve to facilitate maternal-infant attachment and in turn support optimum maternal and infant mental health, and help reduce the possibility of clinical disorder for those with exisitng risk. Working in such a way will introduce a psychoeducative approach that provides a long overdue counterbalance to the emphasis on pathology associated with childbearing.

Knowledge of how specific features of depression and anxiety facilitate normative maternal attachment will better equip professionals to identify the margin between health and pathology. Use of the Whooley questions (NICE 2007) in practice will contribute some assessment of individual risk. However, the detection of disrupted mother-infant synchrony, arising from maternal psychiatric disorder, personality disorder and attachment style is a matter of observation. Therefore meaningful assessment of maternal risk must be made through surveillance in the context of professional support in the early postpartum period. Prompt detection of low maternal sensitivity and responsiveness is necessary to minimise the transmission of mother related risk to the infant. Professionals must utilised relevant knowledge regarding

features of normative postpartum mood to inform therapeutic interventions aimed at postpartum women suffering with enduring, clinical anxiety and depression. This is because the impact of these disorders upon women and infants, superimposed upon the unique mental domain of the postpartum experience, cannot be fully compared with episodes at other times of life. Finally, in the field of perinatal psychiatry assessment of risk in mothers with existing mental illness, treatment progress and parenting capacity must incorporate a cohesive model of normative maternal attachment.

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Appendix I

Research protocol

Version 3 **July 2002**

Towards a normative model of postnatal mood; symptoms of depression and anxiety among women after childbirth

Remember:

Screening takes place on two occasions,

- 4 – 8 postnatal weeks; 1st screen
- 12 – 16 postnatal weeks; 2nd screen

Exclusions:

- Women with a current, major mental health diagnosis, for example, schizophrenia
- Women whose infant is premature or in intensive care
- In the event of stillbirth or sudden infant death
- Females under the age of 16years

Primary birth visit:
Inform all women with information about this confidential study exploring symptoms of depression and anxiety in the early postnatal period

Before 1st screening interval

- Ask every woman if they will agree to participate in the study. This should be done until you have recruited your target number of subjects.
- Do not select women to take part.
- Provide all women who agree to participate or are undecided with the study *information sheet.*
- Consenting women together with the recruiting health visitor will sign the two copies of the consent pro-forma. A witness is not required. A signed copy of the consent will be retained by the women with the remaining copy held in the formal health visiting records.

- On this occasion schedule a designated appointment to complete the measurement scales. Explain how to complete these, i.e. *'Read each question carefully, consider each response and tick one that best reflects how you think/ feel'*. Address any queries and/or concerns the woman may have. Emphasize the advantage of privacy. Reassure women that they will remain anonymous beyond the recruiting health visitor.

First screening visit

- Check that the woman is happy to continue and that she understands how to complete the measurement scales. Record the subject numeric specifier in the designated screening booklet. Explanations for completing the scales should not be overly detailed and should be sufficient to clarify. Provide women with an opportunity to discuss any issues that might arise from completing the scales and respond to individual needs.

Before second screening

- Schedule a designated appointment for the second screening, between 12 -16 postnatal weeks.

Second screening visit

- Conduct as for the first. Inform all women that they might be asked to agree to participate in an interview between 6 and 7 postnatal months.

Appendix II

Patient information sheet

Version 3 July 2002

Towards a normative model of postnatal mood; symptoms of depression and anxiety among women after childbirth

You are invited to take part in a research study. Before you decide it is important to understand why the research is being done and what is involved. Please take time to read the following information carefully.

Take time to decide whether or not you wish to take part.

What is the purpose of this study?
This study intends to explore how women think and feel in the early postnatal period. It aims to promote understanding of how maternal mood might help women to adjust to motherhood. The results of this study will assist in the improvement of care and support for postnatal women.

Why have I been chosen?
Only women in the first 16 weeks after child birth are included in this study, which is why you are being asked to participate. Approximately 1,300 women will be studied.

Do I have to take part?
It is up to you to decide whether or not to take part. You may refuse to take part without giving a reason. A refusal will not affect the standard of care you receive now or in the future. If you decide to take part you will be given this information sheet to keep and asked to sign a consent form. If you decide to take part you are still free to take part at any time and without giving a reason.
This will not affect the standard of care now or in the future.

What will happen to me if I take part?
You will be asked to complete a small number of questionnaires on two occasions. The first will take place between 4-8 weeks after the birth of your baby and the second, 12-16 weeks. On each occasion it should take approximately 15-20 minutes to complete all questionnaires. The questionnaires will be provided by your health visitor and will be completed in your own home at a date and time that is convenient to you. (This does not represent an extra visit).

Completed questionnaires will be placed in an envelope before being returned to me.

Your health visitor will be happy to discuss how you think and feel as a result of completing the questionnaires as well as any personal concerns you might have. Should you decide you would like additional help and support your health visitor will be able to assist you.

Confidentiality
All information collected in this study is appropriately confidential. It will not be identified by your name and address and therefore this information will not be required on the questionnaires. Only your age, the number of postnatal weeks and number of children will be recorded.

What will happen to the results of the research study?
The results of this study will be used to improve services for women in the postnatal period. The results will be published and you will be able to obtain a copy (see contact details below).

YOU WILL NOT BE IDENTIFIED IN ANY REPORT OR PUBLICATION

Who is organising and funding the research?
Monies from the Cardiff and Vale NHS Trust Research and Development Small Grant Scheme financially support this research. The study has been organized by Orion Owen who works as a health visitor in Cardiff. The work is supervised by Professor Steve Rollnick at the Department of general Practice, College of Medicine Cardiff University.

Who has reviewed this study?
Bro Taf Local ethics Committee has reviewed this study.

Contact for further information
Ms. Orion Owen
Address .....
Telephone .....
Email .....

THANK YOU FOR READING THIS

Appendix III

Patient consent pro-forma

Version 3 **July 2003**

Towards a normative model of postnatal mood; symptoms of depression and anxiety among women after childbirth

1	Have you read and understood the patient information sheet? Please keep a copy.	Please circle one **Yes / no**
2	Have you had an opportunity to discuss this study and ask questions?	**Yes / no**
3	Have you had satisfactory answers to all your questions?	**Yes / no**
4	Have you received enough information about this study?	**Yes / no**
5	Has the health visitor give you an explanation about the study? Name of health visitor.....	**Yes / no**
6	Did you understand that you are free to withdraw from the study? • At any time • Without giving a reason • Without effecting your future medical care • That the details of your participation up to the time of withdrawal will be stored anonymously on file and maybe used in the final analysis of data	**Yes / no**
7	Have you had sufficient time to come to your decision?	**Yes / no**
8	Do you agree to take part in this study?	**Yes / no**

Patient signature .....

Date.....

Name (block letters).....

Health visitors signature .....

Date.....

Name (block letters).....

Appendix IV

Subject demographics pro-forma

Towards a normative model of postnatal mood; symptoms of depression and anxiety among women after childbirth

Thank you for taking part in this study

Please remember The study is anonymous. The name and address of participants is not required. Please record the following information	
Maternal age	
No. of children	
Postnatal weeks	1st screen
	2nd screen
Results for the EPDS may be used from the research materials where the scale is applied in routine practice and the woman has agreed; a photocopy may be made if necessary. Otherwise please do not examine completed measurement scales. Please include any relevant, additional information.	

Appendix V

Edinburgh Postnatal Depression Scale

Name: .....

Address:.....

Baby's Age:.....

As you have recently had a baby, we would like to know how you are feeling. Please UNDERLINE the answer which comes closest to how you have felt IN THE PAST 7 DAYS, not just how you feel today.

I have been able to laugh and see the funny side of things.
- As much as I always could
- Not quite so much now
- Definitely not
- Not at all

I have looked forward with enjoyment to things.
- As much as I ever did
- Rather less than I used to
- Definitely less than I used to
- Hardley at all

I have felt scared or panicky for no very good reason. *
- Yes, quite a lot
- Yes, sometimes
- No, not much
- No, not at all

Things have been getting on top of me. *
- Yes, most of the time I haven't been able to cope at all
- Yes, sometimes I haven't been coping as well as usual
- No, most of the time I have coped quite well
- No, I have been coping as well as ever

I have been so unhappy that I have had difficulty sleeping. *
- Yes, most of the time
- Yes, sometimes
- Not very often
- No, not at all

I have blamed myself unnecessarily when things went wrong. *
- Yes, most of the time
- Yes, some of the time
- Not very often
- No, never

I have been anxious or worried for no good reason.
- No, not at all
- Hardly ever
- Yes, sometimes
- Yes, very often

I have felt sad or miserable. *
- Yes, most of the time
- Yes, quite often
- Not very often
- No, not at all

I have been so unhappy that I have been crying. *
- Yes, most of the time
- Yes, quite often
- Only occasionally
- No, never

The thought of harming myself has occured to me *
- Yes, quite often
- Sometimes
- Hardly ever
- Never

J.L. Cox, J.M. Holden, R. Sagovsky (1987)

Beck Depression Inventory

BDI-II

Date:

Name: _____ Marital Status: _____ Age: _____ Sex: _____

Occupation: _____ Education: _____

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past two weeks, including today**. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness
- 0 I do not feel sad.
- 1 I feel sad much of the time.
- 2 I am sad all the time.
- 3 I am so sad or unhappy that I can't stand it.

2. Pessimism
- 0 I am not discouraged about my future.
- 1 I feel more discouraged about my future than I used to be.
- 2 I do not expect things to work out for me.
- 3 I feel my future is hopeless and will only get worse.

3. Past Failure
- 0 I do not feel like a failure.
- 1 I have failed more than I should have.
- 2 As I look back, I see a lot of failures.
- 3 I feel I am a total failure as a person.

4. Loss of Pleasure
- 0 I get as much pleasure as I ever did from the things I enjoy.
- 1 I don't enjoy things as much as I used to.
- 2 I get very little pleasure from the things I used to enjoy.
- 3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings
- 0 I don't feel particularly guilty.
- 1 I feel guilty over many things I have done or should have done.
- 2 I feel quite guilty most of the time.
- 3 I feel guilty all of the time.

6. Punishment Feelings
- 0 I don't feel I am being punished.
- 1 I feel I may be punished.
- 2 I expect to be punished.
- 3 I feel I am being punished.

7. Self-Dislike
- 0 I feel the same about myself as ever.
- 1 I have lost confidence in myself.
- 2 I am disappointed in myself.
- 3 I dislike myself.

8. Self-Criticalness
- 0 I don't criticize or blame myself more than usual.
- 1 I am more critical of myself than I used to be.
- 2 I criticize myself for all of my faults.
- 3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes
- 0 I don't have any thoughts of killing myself.
- 1 I have thoughts of killing myself, but I would not carry them out.
- 2 I would like to kill myself.
- 3 I would kill myself if I had the chance.

10. Crying
- 0 I don't cry anymore than I used to.
- 1 I cry more than I used to.
- 2 I cry over every little thing.
- 3 I feel like crying, but I can't.

_____ Subtotal Page 1

Continued on Back

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11. Agitation

0 I am no more restless or wound up than usual.

1 I feel more restless or wound up than usual.

2 I am so restless or agitated that it's hard to stay still.

3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.

1 I am less interested in other people or things than before.

2 I have lost most of my interest in other people or things.

3 It's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.

1 I find it more difficult to make decisions than usual.

2 I have much greater difficulty in making decisions than I used to.

3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.

1 I don't consider myself as worthwhile and useful as I used to.

2 I feel more worthless as compared to other people.

3 I feel utterly worthless.

15. Loss of Energy

0 I have as much energy as ever.

1 I have less energy than I used to have.

2 I don't have enough energy to do very much.

3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.

1a I sleep somewhat more than usual.

1b I sleep somewhat less than usual.

2a I sleep a lot more than usual.

2b I sleep a lot less than usual.

3a I sleep most of the day.

3b I wake up 1–2 hours early and can't get back to sleep.

17. Irritability

0 I am no more irritable than usual.

1 I am more irritable than usual.

2 I am much more irritable than usual.

3 I am irritable all the time.

18. Changes in Appetite

0 I have not experienced any change in my appetite.

1a My appetite is somewhat less than usual.

1b My appetite is somewhat greater than usual.

2a My appetite is much less than before.

2b My appetite is much greater than usual.

3a I have no appetite at all.

3b I crave food all the time.

19. Concentration Difficulty

0 I can concentrate as well as ever.

1 I can't concentrate as well as usual.

2 It's hard to keep my mind on anything for very long.

3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.

1 I get more tired or fatigued more easily than usual.

2 I am too tired or fatigued to do a lot of the things I used to do.

3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.

1 I am less interested in sex than I used to be.

2 I am much less interested in sex now.

3 I have lost interest in sex completely.

23 24 25 26 27 28 29 30 A B C D E

_____ Subtotal Page 2

_____ Subtotal Page 1

_____ Total Score

Appendix VII

Beck Anxiety Inventory

NAME _____ DATE _____

Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by each symptom during the PAST WEEK, INCLUDING TODAY, by placing an X in the corresponding space in the column next to each symptom.

	NOT AT ALL	MILDLY It did not bother me much.	MODERATELY It was very unpleasant, but I could stand it.	SEVERELY I could barely stand it.
1. Numbness or tingling.				
2. Feeling hot.				
3. Wobbliness in legs.				
4. Unable to relax.				
5. Fear of the worst happening.				
6. Dizzy or lightheaded.				
7. Heart pounding or racing.				
8. Unsteady.				
9. Terrified.				
10. Nervous.				
11. Feelings of choking.				
12. Hands trembling.				
13. Shaky.				
14. Fear of losing control.				
15. Difficulty breathing.				
16. Fear of dying.				
17. Scared.				
18. Indigestion or discomfort in abdomen.				
19. Faint.				
20. Face flushed.				
21. Sweating (not due to heat).				

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Appendix VIII

Subject log

Towards a normative model of postnatal mood; symptoms of depression and anxiety among women after childbirth

Dear Health Visitor

Use this log as a memory aid. The numeric specifier corresponds to the numeric code recorded in the top right hand corner of each screening booklet. At the first screen enter the name of each subject together with the relevant numeric specifier. This record is for your information only. For example;

No.	Name
C1(1)	Mary Jones

Many Thanks

Orion Owen

Appendix IX

Study news letter

Towards a normative model of postnatal mood; symptoms of depression and anxiety among women after childbirth

NEWS LETTER APRIL 2003

Dear Colleagues

Things seem to be running fairly smoothly. THANK YOU!
There have been some initial teething problems in Health Visitors being married up to their research packs but little else to disrupt the collection of data. I have already spoken to a number of colleagues about their experiences in recruiting and screening women. The general feedback is favorable, indicating that the process is relatively straightforward and not particularly time consuming. Initially, many Health Visitors (including myself) felt apprehensive about the possible workload implications. With one or two exceptions, most Health Visitors happily report that women are able to complete the screening procedure in a relatively short period of time.

I have recently submitted an interim report to the Research and Development (R&D) unit (Cardiff and Vale NHS Trust). I am sending copies of this to the R&D and ethics committees of the North Glamorgan and Royal Glamorgan Trusts, to advise them of our progress to date.
For your interest, here are few typical queries:

Q. What happens when clients do not want their names recorded on the research material?

A. The names of clients **should not be included** on the research materials. Only the recruiting Health Visitor requires this information entered on the subject identifier log, to coordinate data from the first and second screening episodes.

Q. Does a past history of depression exclude a woman from the research?

A. Only a current and serious mental illness such as a psychotic depression or schizophrenia will exclude a woman from the research.

Q. How much information should I provide about the screening questions/ statements when women are completing the self-rating scales?

A. As some of you have found this can be difficult to judge. It is important that postnatal women are sufficiently informed by the recruiting health visitor to facilitate informed consent and to complete the measurement tools with the minimum of confusion. At the same time, it is essential that the information provided does not influence the response of subjects. This is best achieved by avoiding *feelings talk* when providing relevant information. Simply, the basic rules are, (1) tick the first response that (2) best reflects how a woman feels and/or thinks rather than '*why*'. It would be appropriate to discuss the '*why's*' on completion of the measurement tools if desired by the subject

---

I hope this information is useful. If you have any other queries, concerns or comments please continue to contact me

Tel: .....

Email.....

Thank You

Orion Owen

Appendix X

Head Count Pro-forma

Towards a normative model of postnatal mood; symptoms of depression and anxiety among women after childbirth

Dear Health Visitor

Once again thank you for your recruiting and screening women!

Just to recap, the total number of postnatal women required for this study is estimated at 1275, with approximately 250 of these being recruited from the Rhondda and Taff Ely. In the main, each Heath Visitor in Rhondda and Taff Ely is required to recruit is 6 postnatal women with a few recruiting 7.

Your Research and Development Committee require an interim report on the progress of the research to date. This needs to include an approximate completion date for screening in Rhondda and Taff Ely. I would be grateful if you could provide the following information;

Date:	
H.V name and base	Number
No. of screening booklets allocated	
Subjects recruited	
Participants with 1st phase completed	
Participants with 2nd phase completed	

Please complete and return to _____ at _____

Thank You

Orion Owen

Appendix XI

EPDS; varimax rotated PCA (Ross et al 2003)

EPDS Items		Components		
		1 Depression	2 Anxiety	3
EPDS 1	laugh	0.77		
EPDS 2	enjoy	0.69		0.52
EPDS 3	blame		0.78	
EPDS 4	anxious		0.65	
EPDS 5	panicky		0.77	
EPDS 6	things getting on top	0.51	0.52	
EPDS 7	so unhappy/difficulty sleeping	0.52	0.51	
EPDS 8	sad or miserable	0.70		
EPDS 9	so unhappy/ crying	0.70		
EPDS 10	thoughts of harming myself			0.90
Total variance		27.0%	25.4%	14.7%

Appendix XII

BDI; varimax rotated PCA (Steer et al 1987)

BDI	Components		
	1	2	3
BDI 1 *sadness*	65		
BDI 2 pessimism	61	66	
BDI 3 *sense of being a failure*			
BDI 4 dissatisfaction	68		
BDI 5 guilt		74	
BDI 6 expectation of *being punished*		61	
BDI 7 *self-dislike*		65	
BDI 8 *self accusation*		59	
BDI 9 *suicidal ideas*	51		
BDI 10 *crying*			
BDI 11 *irritability*	47		
BDI 12 *social withdrawal*	56		
BDI 13 *indecisiveness*	61		
BDI 14 *body image*		51	
BDI 15 *work difficulty*	73		
BDI 16 *insomnia*			54
BDI 17 *fatigability*	63		
BDI 18 loss of *appetite*			66
BDI 19 *weight loss*			67
BDI 20 *somatic preoccupations*			60
BDI 21 *loss of libido*	48		
Total variance	29.9%	8.1%	6%

Appendix XIII

BAI; promax rotated PCA (Beck 1988)

BAI Items	M	SD	r	Factor loading	
				1	2
1 numbness or tingling	.68	.80	.30	.24	
2 feeling hot	.86	.87	.63	.65	
3 wobbliness in the legs	.61	.83	.54	.44	
4 unable to relax	1.89	.78	.61		. 60
5 fear of the worst happening	1.74	1.03	.59		. 87
6 dizzy or light headed	1.00	.95	.63	.62	
7 heart pounding or racing	1.18	.98	.55	.42	
8 unsteady	.96	.99	.71	.65	
9 terrified	1.15	1.14	.63		. 68
10 nervous	1.89	.84	.60		. 61
11 feelings of choking	.39	.80	.46		.32
12 hands trembling	.77	.85	.55	.71	
13 shaky	1.01	.94	.67	.82	
14 fear of losing control	1.54	1.07	.64		.75
15 difficulty in breathing	.87	1.05	.53		. 41
16 fear of dying	.90	1.11	.50		.41
17 scared	1.66	.97	.68	.76	
18 indigestion/ discomfort in abdomen	1.10	.98	.42		.29
19 faint	.68	.91	.67	.67	
20 face flushed	.69	.85	.59	.67	
21 sweating (not due to heat)	.80	.97	.60	.68	