

Panic Disorder:

An exploration of attachment, heterogeneity and the regulation of anger.

Deborah Fooks

Supervisor: Marion Kostanski



~~DOER~~  
STA THESIS  
616.85223 FOO  
30001008610406  
Fooks, Deborah  
Panic disorder : an  
exploration of attachment,  
heterogeneity and the

Submitted in partial fulfilment of the requirements of the

Doctorate in Psychology (Clinical Psychology)

Department of Psychology

Victoria University

March 2005

## Declaration

“I declare that this report is an original piece of research, conducted by myself and does not incorporate any materials previously written by another person except where due reference is made in the text. I further declare that this study has adhered to the principles as established by the Faculty of Arts Human Research Ethics Committee of Victoria University.”

Deborah Fooks

25<sup>th</sup> March 2005

## Abstract

The impact of a panic attack on the individual can be physically and emotionally disabling. Panic disorder - the chronic fear associated with recurrent attacks, is estimated to have a lifetime prevalence of 3% within the adult population. To date, the treatment of panic disorder is restricted to focusing on removal of the manifest symptoms. However, research indicates that this focus is limited in its effectiveness. Recent studies have proposed that this may be due to the assumption in diagnosis, that panic disorder is a homogenous syndrome. Rather, they propose that panic disorder may arise out of a multiplicity of pathways incorporating biological, developmental and/or environmental factors.

This study investigated the proposition that panic disorder is not homogenous. It was proposed that a proportion of sufferers of panic disorder acquire this via a developmental pathway involving childhood separation anxiety and perceptions of aberrant parenting styles. These two variables, which arise out of insecure attachment in infancy, have been reported to be present in the histories of a large number of panic disorder patients. Furthermore, it was proposed that emotional issues, in particular anger suppression, which has been found to be a significant factor arising from insecure attachment and vulnerability in relationships, would also be implicated in the onset of panic attacks. Self-report surveys assessing panic symptomatology, and comprised further of the Separation Anxiety Symptom Inventory (Silove et al., 1993), Parental Bonding Instrument (Parker, 1979), Relationship Scales Questionnaire (Griffin & Bartholomew, 1994), and State-Trait Anger Expression Inventory (Spielberger, 1988), were completed by 242 undergraduate students aged between 17 and 55 ( $M = 23.3$ ). Of the participants who were classified as having self-reported panic disorder, 20% could be reliably identified as such, according to a combination of childhood separation anxiety and current anxieties about relationships. In addition, internalisation of anger was found

to significantly mediate the relationship between insecure attachment and panic frequency. These findings support the proposal that panic disorder is not necessarily homogenous. The outcomes of the study suggest that the assessment and treatment of panic disorder patients requires a more considered individual approach. Specifically, it is proposed that diagnostic assessments need to consider the specific developmental and environmental factors that may have contributed to the formation and maintenance of panic disorder for the individual. This assessment should then inform the recommendations for treatment in order to improve the prognosis for sufferers of panic disorder.

## Table of Contents

Title	i
Declaration	ii
Acknowledgements	iii
Abstract	iv
Table of Contents	vi
List of Tables and Figures	x
List of Appendices	xi
PANIC DISORDER: AN EXPLORATION OF ATTACHMENT, HETEROGENEITY, AND THE REGULATION OF ANGER	
	1
Chapter 1: Panic Disorder – The story so far	1
1.1 Overview	1
<i>Contribution to Knowledge</i>	7
<i>Significance of Outcomes</i>	8
1.2 The Evolution of Panic Disorder as A Diagnostic Construct	8
1.3 Epidemiology	11
1.3.1 <i>Prevalence and Incidence of Panic Disorder</i>	11
1.3.2 <i>Age of Onset and Gender differences</i>	13
1.3.3 <i>Comorbidity and Course</i>	14
1.3.4 <i>Summary</i>	17
1.4 Major Theoretical Perspectives	18
1.4.1 <i>Biological Perspectives</i>	18
1.4.1.1 Hyperventilation and Panic	19
1.4.1.2 CO2 Sensitivity and the “False Suffocation Alarm” Theory	24
1.4.1.3 Genetic Studies	28

1.4.1.4	Summary	30
1.4.2	<i>Psychological Perspectives</i>	31
1.4.2.1	Conditioning Theory	31
1.4.2.2	Sense of Control and Predictability	34
1.4.2.3	Catastrophic Misinterpretation of Bodily Sensations	37
1.4.2.4	Anxiety Sensitivity	40
1.4.2.5	Summary	48
1.5	Chapter Summary	50
Chapter 2:	Attachment Theory and Psychopathology	52
2.1	Attachment Theory: A summary	53
2.2	Determinants of Infant Attachment Status	59
2.3	Assessment of Attachment in Adolescence and Adulthood	64
2.4	Insecure Attachment and Psychopathology	71
2.5	Attachment and Emotion Regulation	79
2.6	Continuity of Attachment Status from the Cradle to the Grave	91
2.7	Summary	98
Chapter 3:	Panic Disorder and the Attachment Paradigm	100
3.1	Developmental Antecedents of Panic Disorder	100
3.1.1	<i>Behavioural Inhibition (temperament)</i>	101
3.1.2	<i>Perceived Parental Behaviours</i>	110
3.1.3	<i>Childhood Separation Anxiety Disorder</i>	115
3.2	Adult Personality and Interpersonal Functioning	122
3.3	Summary of PD research in light of attachment theory: Rationale for current study	127
3.3.1	<i>Aims</i>	132

3.3.2	<i>Hypotheses</i>	134
3.4	Design	134
Chapter 4:	Method	136
4.1	Participants	136
4.2	Materials	136
4.2.1	<i>Assessment of panic symptomatology</i>	137
4.2.2	<i>Anxiety Sensitivity Index</i>	137
4.2.3	<i>Separation Anxiety Symptom Inventory</i>	138
4.2.4	<i>Parental Bonding Instrument</i>	139
4.2.5	<i>Relationship Scales Questionnaire</i>	140
4.2.6	<i>Anger Expression - State-trait Anger Expression Inventory</i>	142
4.3	Procedure	143
Chapter 5:	Results	144
5.1	Variable Distributions and Normative Data	147
5.1.1	<i>Self-reported Panic Disorder Classifications</i>	147
5.1.2	<i>Anxiety Sensitivity Index</i>	147
5.1.3	<i>Separation Anxiety Symptom Inventory</i>	150
5.1.4	<i>Parental Bonding Instrument</i>	151
5.1.5	<i>Relationship Scales Questionnaire</i>	153
5.1.6	<i>Anger-Expression scales of the State-Trait Anger Expression Inventory</i>	156
5.2	Relationships between the Anxiety Sensitivity Index and the independent variables	157
5.3	Hypothesis 1: Heterogeneity	160

5.4 Hypothesis 2: The relation between panic disorder, separation anxiety, parental bonding and attachment.	161
5.5 Hypothesis 3: Panic frequency, attachment variables, and emotion regulation as mediator.	164
Chapter 6: Discussion	167
6.1 Hypothesis 1: Heterogeneity	168
6.2 Hypothesis 2: Attachment and Panic Disorder	172
6.3 Hypothesis 3: Emotion Regulation and Panic Disorder	179
6.4 Implications	182
6.5 Limitations	185
6.6 Conclusions	186
References	189
Appendices	232

## List of Tables and Figures

### Table 1

*Means and standard deviations for the Anxiety Sensitivity Index (ASI) and Separation Anxiety Symptom Inventory (SASI) by panic disorder classification.....* 149

### Table 2

*Means and standard deviations for the subscales of the parental bonding instrument (PBI) by panic disorder classification, reported separately for mother and father..* 152

### Table 3

*Means and standard deviations for the factors of the Relationship Scales Questionnaire (RSQ), and Anger Expression scales of the State-Trait Anger Expression Inventory (STAXI) by panic disorder classification.....* 155

### Table 4

*Correlations between Anxiety Sensitivity Index scores and the independent variables for the total sample (N=242). .....* 159

### Table 5

*Regression analyses testing conditions for the mediation of attachment style and panic frequency by anger suppression (N=88).....* 165

*Figure 1. The distribution of total scores on the Separation Anxiety Symptom Inventory (SASI) for each of the self-reported panic classifications. ....* 161

*Figure 2. The classification hierarchy used to determine membership of the ‘panic’, subclinical panic (‘subpanic’) and ‘no panic’ groups. ....* 235

## List of Appendices

## Appendix A

Self-Report Survey .....	232
--------------------------	-----

## Appendix B

Faculty of Arts Human Research Ethics Committee Approval .....	233
--	-----

## Appendix C

Flow chart detailing the classification hierarchy used to classify participants according to panic status .....	234
---	-----

## Appendix D

Information Statement for Participants .....	236
--	-----

## Appendix E

Participant Consent Form .....	237
--------------------------------	-----

# PANIC DISORDER: AN EXPLORATION OF ATTACHMENT, HETEROGENEITY, AND THE REGULATION OF ANGER

## Chapter 1: Panic Disorder – The story so far

### *1.1 Overview*

Individuals with panic disorder experience crippling recurrent panic attacks that seem to come out of nowhere (American Psychiatric Association, 1994). The condition is only considered to be problematic however when and if the person develops persistent and troubling fears about having further attacks. Often such fears result in significant avoidance behaviours, as the individual attempts to avoid certain places and situations from which escape might be difficult or embarrassing if they were to have a panic attack. This latter condition is referred to as agoraphobia. Panic disorder with/ without agoraphobia affects between one and three percent of the population (e.g., Reed & Wittchen, 1998), and can have devastating effects on the quality of life of sufferers (Markowitz, 1989).

Panic disorder has been well researched since its delineation by Klein (1981), with a plethora of theories proposed to contribute to its etiology. Primarily these theories stem from what could be broadly classed as biological (including genetic studies), and cognitive-behavioural psychology perspectives. The most comprehensive and promising of the biological theories implicated the process of hyperventilation (e.g., Ley, 1987a, 1987b) or possible carbon-dioxide sensitivity (Gorman & Papp, 1990) as the physical mechanisms which triggered panic attacks. While biological theories have focused primarily on the mechanisms that trigger the original or individual panic attacks, psychological theories of panic disorder have

generally focused on the development of pathological anticipatory anxiety, referred to as the “fear of fear”, and the way that this construct contributes to recurrent attacks. The most comprehensive and promising psychological theories to have emerged have differentially implicated: a) the role of conditioning in generating recurrent panic attacks (e.g., Bouton, Mineka & Barlow, 2001), b) the desire for control and predictability and the way in which these variables can modulate anxiety (e.g., Zvolensky, Lejuez & Eifert, 2000), c) the catastrophic misinterpretation of bodily sensations which leads to spiralling anxiety and panic (e.g., Clark, 1986), and d) a dispositional sensitivity to respond fearfully to anxiety symptoms (known as anxiety sensitivity) which increases the likelihood of a panic attack (e.g., Reiss & McNally, 1985).

Both the biological and psychological approaches referred to above have proved invaluable in illuminating specific parts of the panic disorder phenomenon, and in contributing to the development of effective pharmacological and psychological treatments. None have been successful however in accounting for its full complexity. For example, while chronic hyperventilation appears to play a significant role in the genesis or maintenance of panic for a subset of sufferers, panic attacks have also been found to occur in the absence of hyperventilation (Gorman, Liebowitz, Fyer & Stein, 1989). Similar contradictions exist for all of the approaches offered to understand panic disorder etiology to date. As a result, many authors have concluded that panic disorder with/ without agoraphobia is probably a heterogeneous condition with a number of possible etiological pathways leading to it as an end-point condition (e.g., Goldstein & Chambless, 1978; Klein, 1993; McNally, 1994; Silove & Manicavasagar, 2001). Little research exists however which specifically tests this assumption.

Other findings which suggest that the current picture of panic disorder is limited come from treatment efficacy studies and observations of particular personality and interpersonal difficulties amongst panic disorder patients which appear to predate the onset of the disorder (Busch et al., 1991). Longitudinal studies of treatment programs, which have consisted primarily of pharmacological or cognitive-behavioural interventions or a combination of both, indicate that while many patients improve with treatment, the majority continue to be symptomatic and a significant minority (between 10 and 20%) experience no relief of their symptoms at all (e.g., Nagy, Krystal, Woods & Charney, 1989). These studies suggest that an underlying psychopathological process is not being adequately addressed in current treatment programs. Further, clinical observations amongst panic disorder populations of potentially problematic interpersonal traits such as unassertiveness, passivity and problems in tolerating and/or expressing feelings of anger, frustration and disapproval, (e.g., Chambless, Hunter & Jackson, 1982; Emmelkamp & Bouman, 1991) support the idea that the current biological and psychological theories of panic disorder are particularly limited in their lack of a more holistic perspective of personality and psychopathology. By contrast, attachment theory has proved itself to be invaluable in providing a framework that allows for the understanding of psychopathology and related interpersonal and emotional problems from a developmental perspective.

Attachment theory proposes that a child's relationship with its caregivers, whether secure or insecure, leads to and is reflected by the degree of confidence and trust with which the child, interacts with its environment, especially in uncertain or difficult circumstances (Ainsworth, Blehar, Waters, & Wall, 1978; Bowlby, 1969, 1973, 1980). Furthermore, attachment theory proposes that the security of the child's

relating style becomes internalised as representations of 'self' and 'other', and thereby governs and shapes later development. This framework has established itself in recent times as the dominant paradigm from which to research personality and interpersonal variables. Developmental and neurophysiological research has supported many of the suppositions of attachment theory, and has found that major internal processes such as cognitions/thinking and emotion regulation (which necessarily govern responses relevant to panic and anxiety), arise as a function of the early relationships in which they develop (e.g., Schore, 2001; Stern, 1985).

Conducting research into attachment theory, Ainsworth and colleagues found that inconsistent responsiveness and/or consistent unresponsiveness by a mother to her infant leads to an insecure attachment in the child that manifests either as excessive and unsettled clinginess to the mother or detached distance (Ainsworth et al., 1978). Both of these response styles are considered to be adaptive in the immediate environment as they serve the function of maintaining proximity and attachment to the mother under difficult circumstances, albeit in a manner that is likely to have maladaptive consequences if replicated in other environments and/or later relationships. Specifically, the excessively clingy and submissive behaviour exhibited in response to an unpredictable caregiver is characteristic of the insecure attachment style known as anxious-ambivalent attachment. The distant and detached behaviour typical of children with consistently rejecting caregivers is referred to as avoidant attachment.

Anxious ambivalent attachment in children is also associated with a hypervigilant and hyperreactive response style to the mother. For example, such children show frequent and intense displays of angry protest and anxiety towards the mother in response to separation (Ainsworth, et al., 1978). Furthermore, adolescents

characterised as having an anxious-ambivalent attachment style demonstrate higher levels of hostility, anxiety and depression than their secure or avoidant counterparts (Cooper, Shaver & Collins, 1998). Avoidant attachment is generally associated with minimal emotional displays, and possibly with occasional aggressive outbursts (Ainsworth et al., 1978). It is likely that the child's experiences of anger, rejection and fear are suppressed in order to avoid further rejection by the caregiver and/or to avoid feeling overwhelmed by these feelings in the absence of a caregiver who can safely contain these potentially dysregulating emotions (Main & Weston, 1982). Anger is thought to be a particularly problematic emotion for the insecurely attached individual, due to fears that the expression of anger might lead to actual abandonment (Bowlby, 1973; Main & Weston, 1982). Finally, psychopathology is almost always associated with an insecure attachment style (e.g., Dozier, Chase, & Albus, 1999). As such, one would expect individuals with panic disorder to exhibit insecure attachment styles, and as a consequence to demonstrate difficulties with anger and anxiety.

Research into the impact of attachment variables, such as perceptions of parenting behaviour and childhood separation anxiety, on the development of panic disorder has produced significant and interesting, but inconsistent, results. There appears to be a definite relationship between panic disorder and these earlier developmental variables, but not a specific one. For example, individuals with panic disorder with/ without agoraphobia typically characterised their parents as uncaring or rejecting, with reports of overprotectiveness being present but less consistent (Manicavasagar, Silove, Wagner, & Hadzi-Pavlovic, 1999). In addition, a sizeable proportion of panic disorder and/or agoraphobia patients (between 20 and 50% of samples) retrospectively reported having had separation anxiety disorder in childhood (e.g., Aronson & Logue, 1987). However, perceptions of aberrant parenting and

childhood separation anxiety disorder are not reported by all panic disorder sufferers, and furthermore are often reported amongst sufferers of other conditions such as those with specific phobias, generalised anxiety disorder and depression (McNally, 1994). As a consequence, some authors such as McNally have suggested that adverse parenting experiences and childhood separation anxiety disorder are probably general risk factors for later mental health problems, rather than specific to panic disorder. However it is possible that a clearer link between panic disorder and a proposed etiological pathway characterised by nonoptimal parenting, childhood separation anxiety disorder, and current attachment difficulties, is being obscured in the literature by problems associated with high comorbidity rates amongst panic disorder sufferers, but primarily by the assumption of homogeneity in the panic disorder population. That is, the developmental pathway outlined above will be highly relevant in the genesis and maintenance of panic disorder for a distinct subset of sufferers.

This study aims to contribute to previous research into the possible developmental and attachment-related precursors of and/or associations with panic disorder in a number of ways. Firstly, this research aims to investigate the assumption of homogeneity in the panic disorder population, which is seen as a significant limitation that is inherent in much of the past research in the field. In so doing, this study also aims to establish the influence of attachment-related variables in the development of panic disorder. The specific attachment-related variables referred to are the retrospective perceptions of parental behaviours from childhood, possible experiences with childhood separation anxiety, and attachment style in current relationships. In particular, we expect to find that these attachment-related variables will characterise one pathway in the development of panic disorder for a definable subset of panic sufferers only, and not the panic disorder population as a whole.

Finally, this study aims to extend the field of panic disorder research by investigating a possible relationship between the experience of panic attacks and any indications of emotion regulation irregularities. Very little, if any, systematic research has been conducted into this latter idea, despite the existence of important observational and anecdotal evidence of problems in this arena for many panic disorder sufferers. Specifically this study will examine whether a relationship exists between attachment style, emotion regulation with respect to anger expression in particular, and the experience of panic attacks. These aims will be addressed within the framework of a quantitative study using self-report questionnaires to explore the relationships between the above-mentioned variables. A convenience sample of undergraduate university students will be used.

### *Contribution to Knowledge*

This research hopes to clarify the contributions that developmental variables relevant to attachment theory play in the etiological pathway to panic disorder. Panic disorder has been researched extensively over recent decades, however much debate still exists as to how panic disorder evolves. One possible reason for this confusion is that panic disorder is an end-point condition arrived at via a number of different possible pathways. If an investigator were looking to establish a particular theoretical model for panic disorder as the dominant one, any evidence that such a model is indeed relevant for panic disorder development, albeit only for a subset of patients, would be obscured by the fact that it would not be characteristic of the entire population if the assumption of homogeneity continued to hold sway. Such a situation would seem to invalidate much important research into the condition. This study aims to address this important limitation, and in so doing, it hopes to establish

whether further consideration of attachment issues (such as interpersonal and emotion regulation functioning) is warranted in the search for an etiological pathway for panic disorder.

### *Significance of Outcomes*

Establishing whether or not the panic disorder population is indeed heterogeneous, has important implications for the assessment and treatment of this condition. Similarly, establishing the influence of personality and interpersonal variables on the onset and maintenance of panic disorder is critical, as these variables will have a significant impact on patient responses to treatment and on prognosis. That is, if a treatment plan were devised that failed to address an underlying vulnerability to panic, which might be, for example, difficulty expressing anger or disappointment with a significant attachment figure, then a successful treatment outcome for panic disorder would be unlikely in the long-term. As such, if the hypotheses of this study are supported, this research will ultimately contribute to ensuring treatment resources for panic disorder are used more effectively by facilitating more accurate and comprehensive psychological assessments in the first instance. In addition, a fuller picture of the possible risk factors for the development of panic disorder will enable prevention programs to be designed and targeted much more effectively than is currently possible.

### *1.2 The Evolution of Panic Disorder as A Diagnostic Construct*

Panic disorder was first conceptualised as separate from anxiety in the early 1960s as a result of the observation that panic disorder patients experienced an apparently paradoxical reaction to the then new drug imipramine (Klein, 1981). It

was observed that patients diagnosed as schizophrenic (but with symptoms that would earn them a diagnosis of panic disorder with agoraphobia today) and treated with imipramine experienced an alleviation of their spontaneous panic attacks, but no therapeutic effect on their experience of chronic anxiety. This observation seemed to be contradictory of the existing assumption that panic attacks were simply a more severe form of anxiety. As such, Klein argued that panic attacks must characterise a disorder that was distinct from chronic anxiety and not on a continuum with it, albeit strongly associated with chronic anticipatory anxiety. However it was not until 1980, in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (*DSM-III*: American Psychiatric Association, 1980), that panic disorder was formally recognised as a distinct construct, and differentiated from generalized anxiety disorder. The diagnosis of panic disorder evolved over subsequent editions of the *DSM* with respect to the definition of a panic attack (e.g. requirements for frequency and number of symptoms), and the centrality of anticipatory anxiety over panic attacks as the defining feature of the disorder. For example, *DSM-III* panic disorder was diagnosable if a person had at least three attacks over a three-week period, with no requirement for the person to exhibit a fear of subsequent attacks in order to qualify for the diagnosis. In contrast, the *DSM-IV* requires “repeated, unexpected attacks” (with no frequency criterion specified), but also persistent anxiety about further attacks.

In the fourth and most recent edition of the *DSM* (American Psychiatric Association, 1994), agoraphobia has been inextricably linked with panic disorder, so that a clinician can choose to diagnose a patient with “Panic Disorder With Agoraphobia”, or “Panic Disorder Without Agoraphobia”. The *DSM-IV* describes agoraphobia as the fear of having a panic attack in a place from which escape might

be difficult and/or help might be difficult to access. The fear typically leads to pervasive avoidance of a variety of situations including, being alone outside the home, being in crowded supermarkets or using public transport. Some individuals continue to expose themselves to the feared situations, but do this with considerable anxiety and dread. As with panic disorder, the concept of agoraphobia has undergone significant transformations across *DSM* editions. Originally identified as a discrete disorder in its own right, agoraphobia was long viewed as a phobia of public places in which patients had a morbid fear of coming to sudden harm (e.g., Benedikt, 1870, as cited in Barlow, 1988; Deutsch, 1929). By the end of the century agoraphobia was seen as a disorder not diagnosable in itself, but rather a complication and direct consequence of panic disorder (e.g., American Psychiatric Association, 1994; Barlow, 1986). This theoretical shift occurred as a consequence of evidence that a history of panic attacks usually preceded the development of agoraphobia (e.g., Uhde et al., 1985). Some debate still exists however as to the relation between panic and agoraphobia (refer to McNally, 1994, pp. 173-175 for a more detailed presentation of this debate). The view outlined above has been generally characterized as the “American” perspective, and disputed by proponents of the “European” view who hold that agoraphobia need not be preceded by panic attacks nor motivated by anticipation of them, but constitutes a unique diagnosis in its own right (Marks, 1987). The debate about the relationship between panic and agoraphobia suggests the need for caution in reviewing and evaluating the vast body of existing panic disorder literature. Given that the diagnostic criteria for panic disorder and agoraphobia have changed subtly but significantly over the years, and also that the disorders are diagnosed differently in various parts of the world, grouping a number of separate studies together for the purposes of a review might disguise important differences in

the populations of each study. These differences could then potentially confound any generalizations or conclusions drawn about the panic disorder population as a whole. For example, Silove and Manicavasagar (2001) have noted that inconsistencies emerging between past and present research investigating the relationships between agoraphobia, childhood separation anxiety and parental behaviours may well be compounded by the fact that agoraphobia is no longer defined in a way that equates the disorder with an adult form of separation anxiety.

The current accepted diagnostic criteria for panic disorder, according to the *DSM-IV* requires that a person experience “recurrent unexpected panic attacks”, along with persistent concern about having additional attacks, or worry about the implications of an attack (e.g., having a heart attack); or a significant change in behaviour as a consequence of the attacks (e.g., avoidance behaviour). In addition, the panic attacks must not be the physiological consequence of a medical condition or medication, and must not be better accounted for by another mental disorder. A panic attack is defined as a discrete period of fear or discomfort in which at least four of a list of 13 symptoms develop abruptly and reach a peak within 10 minutes. The possible symptoms include heart palpitations, trembling, shortness of breath, chest pain, feelings of dizziness or derealisation, sensations of numbness or tingling, and fears of losing control, dying or going crazy.

### *1.3 Epidemiology*

#### *1.3.1 Prevalence and Incidence of Panic Disorder*

Worldwide epidemiological studies have demonstrated fairly consistent prevalence rates for panic disorder across such diverse countries as the United States,

Germany, Lebanon, Korea and New Zealand (Weissman, Canino, Greenwald & Joyce, 1995). The lifetime prevalence rates for *DSM-III-R* and/or *DSM-IV* diagnosed panic disorder vary between about one and three percent, depending on different samples and different methodologies used for diagnosing panic disorder in the population (e.g., Eaton, Dryman, & Weissman, 1991; Katerndahl & Realini, 1993; Reed & Wittchen, 1998). Approximately one third to one half of these panic disorder sufferers also have agoraphobia (e.g., Eaton, Kessler, Wittchen & Magee, 1994; Eaton, Dryman & Weissman, 1991; Markowitz, Weissman, Ouellette, Lish & Klerman, 1989). Aronson and Logue (1987) found relatively few predictors for the development of phobic complications from panic disorder. Contrary to their expectations, they found that neither the frequency of panic attacks reported by their panic disordered patients, nor the duration of the illness was differentially associated with the development of agoraphobia. The risk factors for agoraphobia that emerged from their study included female sex, interpersonal sensitivity (as measured by the Symptom Check List or SCL-90), and a history of childhood difficulties with separation anxiety disorder, overanxious disorder or childhood depression.

Despite the relatively small percentage of people who qualify for a *DSM* diagnosis of panic disorder, somewhat more report experience with panic attacks. Based on structured interviews, Katerndahl and Realini (1993) reported that 5.6% of their large community sample of Texan residents ( $N= 1306$ ) had experienced panic attacks. Similarly, Reed and Wittchen (1998) found that 4.3% of their community sample of 3021 young people in Munich, aged 14 to 24 years, had experienced one or more *DSM-IV* panic attacks in their lifetime. A further 13.1% of the community sample had experienced panic symptoms which did not however meet the criteria for a panic attack. Finally, as is to be expected, the reported prevalence rates for panic

attacks based on self-report methodologies are considerably higher than for techniques using structured interviews. Predominantly, self-reported studies of panic disorder have used university student samples. In three studies conducted by Norton and Colleagues, up to 36% of university students reported having experienced one or more panic attacks in the previous year, according to *DSM-III* or *DSM-III-R* criteria (Cox, Endler, Norton, & Swinson, 1991; Norton, Dorward, & Cox, 1986; Norton, Harrison, Hauch, & Rhodes, 1985).

### *1.3.2 Age of Onset and Gender differences*

The age at onset of panic disorder typically occurs in late adolescence/ early adulthood. Some evidence also exists to suggest that an additional and smaller group of people develop panic disorder in their mid-30s (American Psychiatric Association, 1994; Dick, Bland & Newman, 1994; Eaton, Kessler, Wittchen & Magee, 1994). By contrast, prepubertal onset is rare, but does appear to exist (e.g., Alessi & Magen, 1988).

A considerable difference exists in the gender distribution of panic disorder, with women consistently diagnosed with the disorder between two and three times more frequently than men (e.g., Dick et al., 1994; Eaton et al., 1991; Katerndahl & Realini, 1993; King, Ollendick, Mattis, Yang & Tonge, 1996; Reed & Wittchen, 1998). Further, the ratio of women to men diagnosed with agoraphobia has been reported to be even higher. For example in a review of 12 studies, Clum and Knowles (1991) found that women constituted 59% of the pure panic disorder cases and 89% of the cases of panic disorder with agoraphobia. Furthermore, women tend to demonstrate an earlier onset of both panic attacks and panic disorder than men (Reed & Wittchen, 1998). Although a number of possible reasons for the observed sex

differences in panic disorder incidence have been proposed, as yet there are no conclusive answers. Genetic linkage studies examine the familial distribution of DNA markers in the genome (that have known hereditary properties), to determine the pattern of segregation of panic disorder diagnoses within a family. In one such study, Fyer and Weissman (1999) found some indication of a possible sex-linked hereditary component for panic disorder, however this study has not been replicated, and, as yet, the evidence for sex-linkage is not compelling. Meanwhile Chambless and Mason (1986) proposed that the social conditioning of women and men in stereotypical gender-role behaviours might contribute to higher rates of agoraphobia in women. They suggested that it might be considered more socially acceptable for women than men to use avoidance as a legitimate method for coping with fearful situations. Finally, findings that women and men report differences in panic attack symptomatology have led some authors to suggest that the pathophysiology of panic disorder might differ for men and women (e.g., Kim, Min, & Yu, 2004; Sheikh, Leskin, & Klein, 2002).

### *1.3.3 Comorbidity and Course*

In general, the existence of comorbidity amongst panic disorder patients for other *DSM* diagnosable conditions appears to be the rule, rather than the exception. Depression and generalized anxiety disorder co-occur with panic disorder most often, and Axis II conditions are also prevalent (e.g., Dick et al., 1994).

Generalized anxiety disorder has been diagnosed in as many as 70% of panic disorder patients (Aronson & Logue, 1987). Approximately one third of panic disorder patients have been concurrently diagnosed with a depressive disorder (Aronson & Logue, 1987; Servant & Parquet, 2000), and up to 90% of panic patients

in clinical populations (Aronson & Logue, 1987; Lesser, 1990; Noyes et al., 1990) have experienced a major depressive episode at some point in their lives. Between one and two thirds of these cases represented a primary depressive condition, rather than a reaction to difficulties associated with panic and avoidance behaviours (Aronson & Logue, 1987; Lesser et al., 1988).

Brooks, Baltazar, and Munjack (1989) noted that between 40 and 65% of panic disorder patients identified in the literature also qualified for a personality disorder diagnosis. Amongst these panic patients, avoidant, dependent and histrionic personality disorders predominated (e.g., Chambless, Renneberg, Goldstein, & Gracely, 1992). Dependent personality disorder was especially common amongst those patients diagnosed with agoraphobia (Reich, Noyes, & Troughton, 1987). Indeed, Noyes et al. (1990) found that the severity of avoidance behaviour (which essentially constitutes agoraphobia) demonstrated by panic disorder patients correlated strongly with comorbid depression and personality disorder. Furthermore in a review of the literature, Mennin and Heimberg (2000) found that the presence of personality psychopathology exerted a detrimental effect on treatment outcome for panic disorder patients. Indeed, individuals with comorbid panic and personality disorders were twice as likely as those panic patients without a personality disorder to have a history of depression, a history of childhood anxiety disorder, and a chronic, unremitting course of illness. Noyes et al. found that, in addition to those risk factors mentioned, extensive agoraphobia also predicted a severe chronic course and ongoing social maladjustment.

On the whole, even with treatment, panic disorder seems to present a relatively chronic picture, with panic attacks generally fluctuating in frequency and severity over time (Aronson & Logue, 1987; Breier, Charney & Heninger, 1986; Noyes et al.,

1990). Noyes and colleagues followed up 89 patients, three years after they received pharmacological treatment. The majority were still symptomatic but reported relatively little distress or social maladjustment. As mentioned above, personality disorder was the strongest predictor of social maladjustment. Aronson and Logue reported on the pre-treatment histories of 46 patients with panic disorder or agoraphobia according to *DSM-III* criteria. While these authors also found that the patients in their study reported variations in panic attack frequency over time, they also noted that the patients' phobic and dependent behaviours tended not to fluctuate. Once established, these behaviours appeared to be relatively resistant to extinction.

Longitudinal studies of treatment programs (e.g., Aronson & Logue, 1987; Nagy et al., 1989; Nagy et al., 1993; Noyes et al., 1990; Pollack & Marzol, 2000) which have consisted primarily of pharmacological or cognitive-behavioural interventions or a combination of both, have found that a majority of patients, between 80 and 90%, were significantly improved at follow-up, although only a minority were actually symptom-free (between 10 and 20%). In addition, up to 20% of patients were the same as or worse than they were prior to treatment. This picture of treatment outcome is complicated however by two important factors. Firstly the majority of the patients in these studies were continuing to receive treatment (primarily pharmacological) at the time of follow-up. Moreover, the attrition rates from treatment programs for panic disorder were quite high. For example, Aronson and Logue reported that 41% of patients dropped out of their six-month treatment program by the third session. They found that unemployment, high interpersonal sensitivity as assessed by the SCL-90 and a secondary depressive condition were associated with premature attrition. Each of these factors is likely to have the effect of inflating the true figures for a successful response to panic disorder treatment. In

that event, the numbers of patients who did not respond significantly to the current preferred treatment paradigm of medication and/or cognitive-behavioural training might be substantially higher than reported here.

#### *1.3.4 Summary*

Panic disorder is a complex clinical phenomenon whose definition has evolved quite considerably over the years. It affects more women than men, and generally develops in early adulthood, though a smaller proportion of cases emerge in middle adulthood. The comorbidity rates for additional axis I disorders, especially depression and generalized anxiety disorder, and/or personality psychopathology are high. As such, any given sample of panic disorder patients is likely to demonstrate quite a mix and range of pathologies. For example, individuals will almost certainly vary along dimensions assessing the current intensity of panic and anticipatory anxiety, but may also differ in their profile of panic attack symptomatology, on dimensions of agoraphobic avoidance, and in the type and severity of symptoms reflecting other mood or anxiety disorders.

Panic disorder has a relatively chronic, albeit fluctuating, course. Although treatment programs generally lead to more positive outcomes for a majority of panic disorder patients, a large proportion remain symptomatic or unchanged in their condition following treatment. Studies that have evaluated the available treatment programs for panic disorder have informed theoretical advances with regard to panic disorder etiology.

## 1.4 Major Theoretical Perspectives

Since the definition of panic disorder as a distinct clinical disorder, extensive research has been conducted into attempting to understand the genesis of this condition. Primarily this research has focused on explication of the physiological and psychological mechanisms that may trigger episodes of panic, and subsequently turn the experience of a panic attack into the disabling fear of further attacks that is central to the classification of the disorder. Given the complexity of this research, each of the main biological and psychological theories on panic disorder aetiology will be considered separately.

### 1.4.1 Biological Perspectives

Although a range of possible physiological mechanisms for generating the onset of panic attacks have been investigated over recent decades (e.g., carbon dioxide sensitivity), there does not seem to be any one distinct mechanism that can be identified as underlying all *DSM*-definable panic attacks. Similar difficulties have arisen with respect to the search for a direct *genetic* pathway to the development of panic disorder. Nevertheless, empirical support has indicated that some of the physiological mechanisms that have been identified in the literature as important to panic disorder pathogenesis may be differentially relevant to specific subsets of patients with the condition. As such, McNally (1994) argued that the experience of a panic attack in one person may reflect dysregulation in one physiological or neurobiological system, whereas a phenomenologically similar attack in another person may arise from dysfunction in another system.

To date, the most effective biological explanations of panic disorder etiology have arisen from the 'Biological Challenge Paradigm'. 'Biological challenge' refers

to the technique of generating the experience of a panic attack in the laboratory by inducing hyperventilation or introducing specific chemical agents to the body (e.g., caffeine or carbon dioxide). When introduced to the human body, these techniques consistently produce panic attacks in a substantially larger proportion of panic patients than healthy controls (McNally, 1994). In support of the assumption that the panic attacks provoked by biological challenge are reasonable approximations of naturally occurring panic attacks experienced by those with panic disorder, medications effective in the treatment of spontaneous panic attacks (such as imipramine) have been demonstrated to be successful at preventing laboratory-induced panic attacks (Klein, 1981). Thus, biological challenge studies have permitted investigation of the physiological and biochemical changes that accompany acute panic. Furthermore, understanding the mechanism by which a particular panicogenic agent might generate an episode of panic, has allowed inferences to be made regarding the possible underlying pathogenesis of panic disorder. The most popular and comprehensively debated theories evolving from the biological challenge paradigm refer to hyperventilation and sensitivity to carbon dioxide (CO<sub>2</sub>), and the false suffocation alarm as the origins for panic.

#### *1.4.1.1 Hyperventilation and Panic*

Hyperventilation has been viewed as both a cause and a consequence of panic disorder. Ley (1987a, 1987b) has been a strong advocate of the proposition that hyperventilation actually causes panic. Ley proposed that, although many people acutely hyperventilate in response to a significant stressor, some individuals may chronically overbreathe, leading them to be especially vulnerable to experiencing intense symptoms as a consequence of acute hyperventilation.

Hyperventilation occurs when persons breathe more quickly or more deeply than required to meet their bodies metabolic needs for oxygen. The result of overbreathing or hyperventilation is that more CO<sub>2</sub> is exhaled than is produced by cellular metabolism. As a consequence their body enters a physiological state known as hypocapnia, in which there is a reduction in the partial pressure of arterial carbon dioxide.

The acute reduction of CO<sub>2</sub> partial pressure, as occurs when an attack of hyperventilation begins intensely and peaks in a short space of time, has been shown to manifest in a series of physiological effects. These range from sensations of respiratory distress, increased heart rate, tingling, dizziness, and feelings of being detached from reality (the latter results from reduced blood flow to the brain) (Gorman & Papp, 1990), such sensations being analogous to those of a panic attack (Magarian, 1982).

Chronic hyperventilation occurs when a person breathes slightly more quickly or more deeply than their body requires over an extended period of time. As a result of this behaviour, the partial pressure of CO<sub>2</sub> in the blood drops relatively slowly. The gradual reduction in arterial partial pressure allows the body time to adjust and it begins to initiate compensatory mechanisms that re-establish equilibrium for most of the systems disturbed by this process. As a consequence of the body's compensatory actions, it is able to operate relatively normally despite the reduced partial pressure of CO<sub>2</sub> in the blood. Furthermore the body's actions ensure that the physical sensations and symptoms of acute hyperventilation are prevented. However, despite being able to operate relatively normally, the body has no compensatory mechanisms which will automatically restore normal levels of arterial carbon dioxide partial pressure. As a result, a chronic state of hypocapnia is induced which manifests as a lower than

normal threshold for the triggering of acute hyperventilation and all its accompanying physiological sensations. That is, the presence of a reduced hyperventilation threshold means that even slight increases above the usual levels of respiratory rate or volume, as might occur in response to a minor stressor, can produce an attack of acute hyperventilation.

According to Ley (1987a, 1987b), panic arises only when the symptoms of stress-induced hyperventilation are misinterpreted as life threatening. The misinterpretation of symptoms increases the individual's fear and activates the autonomic nervous system (ANS) in preparation for a fight or flight response. ANS activation causes further increases in respiration rates which exacerbates the reduction of CO<sub>2</sub> in the blood and intensifies hypocapnic symptoms. Spiralling anxiety results in a panic attack. In the light of evidence that catastrophic thoughts about hyperventilation-induced symptoms often followed rather than preceded the phenomenal experience of panic (e.g., Wolpe & Rowan, 1988), Ley (1989) revised his theory to minimise the role of cognitions in the onset of a panic attack. He proposed that the fear experienced during a hyperventilatory panic attack was a direct response to the symptom of respiratory distress in a situation where the individual believed that he/she had no control over the conditions influencing his/her breathing difficulties and so experienced the symptoms as a threat of suffocation. In other words, he believed that the fear induced by the individual's perception of powerlessness and inability to breathe comfortably was sufficient to produce panic, without the need for the misinterpretation of hyperventilation symptoms. In summary, Ley (1991) proposed that heart palpitations and respiratory distress (which he argued were the primary symptoms of panic attacks) and other somatic complaints were the consequence of hyperventilation-induced hypocapnia; that the fear involved in a panic

attack was a consequence of the threat of suffocation; and finally, that the frequently reported experience of catastrophic thoughts was the consequence of an hyperventilation-induced reduction in oxygen flow to the brain.

In support of his theory of panic based on hyperventilation, Ley (1991) cited the findings of treatment studies that have demonstrated positive effects for breathing retraining with panic and agoraphobic patients (e.g., Bonn, Readhead & Timmons, 1984; Clark, Salkovskis & Chalkley, 1985; Rapee, 1985). Breathing retraining refers to a program designed to teach people to slow down their breathing and thus prevent or terminate hyperventilation. Furthermore, Ley (1988) interpreted the findings of biological challenge studies that have successfully used sodium lactate to induce panic attacks as consistent with his theory that hyperventilation is the basis of panic attacks. He suggested that lactate infusion, like hyperventilation-induced hypocapnia, causes an increase in the alkalinity of the blood that produces frightening symptoms reminiscent of those produced by hyperventilation. Indeed, Gorman et al., (1986) found that patients who panicked in response to lactate infusion exhibited signs of chronic hyperventilation. They also found that lactate-induced panic was characterized by acute hyperventilation that occurred in the context of chronic background hyperventilation.

Nevertheless, despite the fact that some studies exist whose findings support Ley's proposal that hyperventilation is the underlying physiological mechanism of panic disorder etiology, many more studies contradict this concept (e.g., Gorman et al., 1986; Gorman et al., 1989). For example, Gorman, Liebowitz, Fyer & Stein (1989) found evidence for chronic hyperventilation in only about 50% of their patients with panic disorder. Other studies have found no indications for chronic hyperventilation amongst panic patients at all (e.g., Zandbergen, van Aalst, de Loof,

Pols, & Griez, 1993). Furthermore, the findings of at least two studies indicated that acute hyperventilation preceded or accompanied the experience of panic attacks in only a minority of individuals (Garssen, Buikhuisen, van Dyck, 1996; Hibbert & Pilsbury, 1989). The authors of these studies concluded that there was no evidence to support the hypothesis that hyperventilation causes panic attacks or contributes to their severity. Furthermore, based on the literature investigating the effectiveness of breathing retraining (BT) in panic disorder treatment, Garssen, de Ruiter and van Dyck (1992) concluded that, although breathing retraining and related procedures were therapeutically effective, this was probably due to principles other than Ley's proposal, namely that BT decreased the tendency to hyperventilate. Instead they attributed the effectiveness of BT to a number of components of the treatment including the tendency of BT to induce a relaxation response, the presentation of a credible explanation for the threatening physical symptoms of panic, and the fact that practising BT offers an opportunity for distraction in the event of a panic attack, and promotes a feeling of control. Finally, Gorman and colleagues (1984) discovered that the inhalation of 5.5% CO<sub>2</sub> during biological challenge produces panic attacks in patients with panic disorder or agoraphobia despite the fact that its physiological effects are directly opposite to those of hyperventilation (and challenge with sodium lactate infusion).

It seems that some individuals with panic disorder exhibit a tendency to hyperventilate prior to, or during episodes of panic, and some show evidence for chronic hyperventilation. For these individuals, overbreathing may represent a habitual pattern of responding to chronic stress. In the event of a stressor that triggers further increases in respiration, acute hyperventilation may provide a source of disturbing bodily sensations that set the scene for a panic attack. However, it has been

demonstrated that hyperventilation is not a necessary component of panic, and there are many people who, in the event of experiencing unfamiliar body sensations, do not respond with panic. In his original hypothesis about the role and pathogenesis of hyperventilation in producing panic attacks, Ley insisted that hyperventilation must be accompanied by fearful cognitions for panic to eventuate. Even following his modification of this theory which was designed to minimise the role of cognition, Ley emphasised that the fear that arose in response to the threat of suffocation occurred in the context of the perception that the individual had no control over this threat. In this way, Ley's theories acknowledge that the existence of physiological mechanisms for the triggering of episodes of panic constitutes just one part of the larger phenomenon of panic attacks. The role of cognitive mechanisms must also be carefully investigated.

#### *1.4.1.2 CO<sub>2</sub> Sensitivity and the "False Suffocation Alarm" Theory*

Biological agents that have been identified as strongly provocative of panic attacks under laboratory conditions are sodium lactate, bicarbonate, and carbon dioxide. Although these substances are metabolised along different pathways which might involve increases or decreases to alkalinity/ acidity in the peripheral circulatory system, they are all eventually metabolised into carbon dioxide. As CO<sub>2</sub> is easily able to cross the blood-brain barrier, the final result of biological challenge using the above agents is an accumulation of CO<sub>2</sub> at chemosensitive regions of the brain stem. This accumulation triggers a vigorous hyperventilatory response by the body (Liebowitz et al., 1985a). With this biochemistry in mind, Gorman and Papp (1990) proposed that individuals with panic disorder might be characterised by hypersensitive CO<sub>2</sub> chemoreceptors in the brain. According to this theory, the receptors of patients with

panic disorder respond more vigorously than in healthy controls to equivalent increases in carbon dioxide. An exaggerated ventilatory response, along with respiratory distress, is experienced as suffocation which triggers intense fear. The fear in turn triggers hyperventilation and all its attendant physical symptoms (e.g., dizziness, tingling, disorientation). Gorman and Papp further proposed that, rather than being a cause of panic attacks, evidence for chronic hyperventilation amongst panic disordered patients might actually represent an adaptive response. That is, given that chronic hyperventilation resulted in the body maintaining low CO<sub>2</sub> levels, this function could serve the adaptive purpose of preventing the hypersensitive CO<sub>2</sub> chemoreceptors from firing, thereby avoiding the initiation of vigorous respiratory symptoms and ultimately hyperventilation and panic.

Consistent with the hypersensitive CO<sub>2</sub> chemoreceptor hypothesis, some studies have found that CO<sub>2</sub> inhalation triggers a stronger ventilatory response in panic patients than in healthy controls (e.g., Gorman et al., 1986; Lousberg, Griez & van den Hout, 1988; Papp et al., 1989). However, other studies have failed to replicate these findings (e.g., Pain, Biddle & Tiller, 1988; Zandbergen, Pols, de Loof, & Griez, 1991). Furthermore, studies investigating lactate-induced panic have produced findings that are inconsistent with CO<sub>2</sub> sensitivity theory. Indeed, Gorman and colleagues found that biological challenge with lactate infusion, using a variant isomer of sodium lactate that is not metabolized to carbon dioxide, was able to provoke panic in the laboratory (Gorman & Papp, 1990). Nevertheless, in a review of the biological aspects of panic disorder, McNally (1994) concluded that sufficient evidence existed in favour of CO<sub>2</sub> sensitivity theory to suggest that this mechanism might be implicated in at least a subset of people who develop panic disorder.

A popular extension of the carbon dioxide sensitivity model for panic disorder was proposed by Klein (1993). Drawing upon a diverse array of potentially converging sources of information including, but certainly not limited to, a) biological challenge data, b) the intense fears of suffocation encountered by the average person, and c) a rare congenital disorder known colloquially as Ondine's curse, in which an infant breathes normally while awake but stops breathing once asleep, Klein posited the existence of an evolved suffocation alarm system that is dysfunctional in people suffering from panic disorder. He defined this alarm system as a mechanism which triggered sudden respiratory distress and an adaptive desire in the individual to flee to an area of open space or fresh air, in the face of internal (e.g., the partial pressure of  $\text{CO}_2$  ( $\text{pCO}_2$ ) in the blood) and/or external (e.g., a stuffy, unventilated room) cues for possible asphyxiation. In individuals predisposed to panic disorder, Klein argued that the suffocation alarm was set to fire at a pathologically low threshold and thus acts as a false alarm for suffocation. Although Klein proposed that the false suffocation alarm was primarily a biological dysfunction, he acknowledged that psychological variables might also influence the development of the suffocation alarm system such that traumatic life events could modify the alarm threshold. In particular, Klein noted the frequent reports of separation anxiety, loss and grief in the histories of panic disorder patients, and proposed that these experiences might lower the suffocation alarm threshold in some causing chronic anxious symptomatology and heightening panic vulnerability.

Finally, by integrating the findings of numerous studies, Klein emphasised that naturally-occurring panic attacks were heterogeneous and could be primarily differentiated into two groups. He did this in order to clarify the difference between panic and panic-like anxiety states, as he felt that the confusion of the two was

confounding the literature and leading to inconsistencies amongst study findings. Klein classed one panic group as more typical of the spontaneous panic that was considered to be characteristic of panic disorder. This type of panic was considered by Klein to be characterized by respiratory distress (dyspnea); was responsive to imipramine (i.e., serotonin reuptake blocking antidepressants); regularly incited phobic avoidance; lacked acute HPA axis activation (N.B. HPA refers to the hypothalamic-pituitary-adrenocortical axis long recognised as part of the stress response), and was provoked by biological challenge agents such as lactate, bicarbonate and CO<sub>2</sub>. In contrast, Klein considered the second group of panic to more closely resemble fear-like surges (qualitatively distinct from panic). These “panic attacks” were characterized by pounding heart, sweating and trembling (but no respiratory distress); was responsive to benzodiazepines (not imipramine); reflected a hypersensitive HPA axis/ autonomic system; and was provoked by biological agents that produced varying degrees of autonomic surge and HPA activation, such as caffeine and yohimbine. He proposed that both acute disturbances could coexist and interact sequentially and synergistically and were not mutually exclusive. Moreover he postulated that for many panic patients, initial severe respiratory panics would eventually cause secondary fearful HPA-related surges.

Klein’s (1993) false suffocation alarm theory has experienced only limited and unconvincing empirical support (e.g., Asmundson & Stein, 1994; Gorman et al., 1994; Taylor & Rachman, 1994). For example, biological challenge studies using hyperventilation, lactate and/or CO<sub>2</sub> inhalation have demonstrated that a greater proportion of panic patients than healthy controls (e.g., Gorman et al., 1994) or patients with other forms of anxiety disorder (e.g., Liebowitz et al., 1985b) experience a panic attack in response to the challenge. These findings are consistent with the

hypothesis that panic disorder patients demonstrate a hypersensitivity to CO<sub>2</sub> as proposed by the false suffocation alarm theory. However while these and other findings offer support for Klein's work, they could also be interpreted as offering support to alternative explanations for panic etiology such as Ley's theory invoking the fear of uncontrollable respiratory distress as the instigator of panic (Ley, 1996), or cognitive theories such as Clark's (1986) 'catastrophic misinterpretation of bodily sensations'. For example, Salkovskis and Clark (1990) have argued that biological challenge agents such as CO<sub>2</sub> produce panic through their ability to create physical sensations that can be misinterpreted, rather than through some specific biochemical pathway. In addition, Schmidt, Telch and Jaimez (1996) noted that the findings from studies such as theirs in which individuals with panic disorder were exposed to a 35% CO<sub>2</sub> challenge were inconsistent with the possibility of a false suffocation alarm. Schmidt et al. found that, although 35% CO<sub>2</sub> inhalation induced a panic attack in a substantial proportion of their patients (about 45%), the majority did not panic during the challenge. The authors noted that, given that 35% CO<sub>2</sub> represents a concentration of CO<sub>2</sub> that is 875 times greater than that found in dry room air, one would expect this seemingly potent dose to cross even the least sensitive threshold, and thereby to trigger panic in 100% of panic disorder patients. Moreover given the intensity of the dose, one would expect it to exceed the alarm threshold even for normal controls. This is not the case (e.g., Gorman et al., 1994; van den Hout, van der Molen, Griez, & Lousberg, 1987).

#### *1.4.1.3 Genetic Studies*

That panic disorder runs in families is fairly well-accepted (van den Heuvel, van de Wetering, Veltman, & Pauls, 2000). In a review of genetic studies of panic

disorder, van den Heuvel et al. noted that increased rates of panic disorder were consistently reported among first-degree relatives of individuals with panic disorder as compared with relatives of controls. They found that the reported lifetime morbidity risk for panic disorder amongst first-degree relatives of PD patients varied between 7.7% and 20.5%.

Investigations into the possibility of a genetic component for panic disorder have produced mixed findings (e.g., Crowe, Noyes, Pauls, & Slymen, 1983; Judd, Burrows & Hay, 1987; van den Heuvel et al., 2000). For example, Crowe et al. examined the family histories of 41 panic disorder patients to assess whether the pattern of expression for panic disorder was consistent with a model predicting either a single-gene transmission or a multiple-gene model of transmission. He found that he could not exclude either model. Instead, more recent studies (e.g., Cavallini et al., 1999) have provided support for an additive model of genetic transmission for panic disorder, in which there is incomplete penetrance of genes, and “additional factors” determine whether the relevant genes are transmitted and/or expressed.

In their review of the concordance rates for panic disorder for monozygotic (identical) versus dizygotic (non-identical) twins, van den Heuvel and colleagues (2000) found monozygotic twins to be at least 2.5 times more likely to both present with panic disorder than were dizygotic twins. This finding was quoted as indicating support for the role of genetic factors for panic disorder etiology. In addition, based on twin studies, Kendler et al. (1993) estimated the heritability of panic disorder liability at 30 – 40%. Nevertheless, despite the empirical support for a genetic contribution to panic disorder etiology, van den Heuvel et al. noted that studies into the heritability of panic disorder have generally found concordance rates for monozygotic twins to be 40% or lower. As such, they concluded that both shared and

unique environmental factors must be considered to be of at least equal importance in the etiology of panic disorder.

#### *1.4.1.4 Summary*

Biological accounts of panic disorder etiology have done much to elaborate the physiological mechanisms implicated in the panic response. The findings from this research suggest that there may be a number of different pathways (e.g., hyperventilation; CO<sub>2</sub> hypersensitive chemoreceptors in the brain), with different underlying physiology, that all lead to a common end-point diagnosis of panic disorder. Other possible pathways for panic development that have received various degrees of support include noradrenergic dysfunction, mitral valve prolapse and audiovestibular irregularity. These will not be covered here. (For a comprehensive review, refer to McNally, 1994).

Biological theories have informed important advances in both pharmacologic treatment of panic disorder (with patients' responses feeding back to inform theoretical advances) and behavioural treatments (e.g., breathing re-training). Biological theories however are insufficient for accounting for the full spectrum of panic disorder phenomenology as they cannot account for a number of important findings, such as: a) the perception of control (or lack thereof) significantly mediating panic response (e.g., Sanderson, Rapee & Barlow, 1989); b) the presence of a 'safe' companion significantly reducing physiological arousal in response to CO<sub>2</sub> challenge (Carter, Hollon, Carson & Shelton, 1995); c) whether hyperventilation sensations are experienced as pleasant or unpleasant is a function of cognitive interpretation (Salkovskis & Clark, 1990); d) CO<sub>2</sub> challenge produces strikingly similar physiological and biochemical effects in both panic patients and healthy controls, yet

only the former routinely panic (Woods, Charney, Goodman & Heninger, 1988).

These and other findings indicate that psychological variables also play a fundamental role in panic disorder etiology. Indeed it would appear that these factors (i.e., perceptions of safety and/or controllability, and cognitive interpretation) cannot be easily separated (or dismissed) from the physiological experience of panic.

#### *1.4.2 Psychological Perspectives*

Psychological accounts of panic disorder aetiology have focused primarily on the concept of a ‘fear of fear’ which was first advanced by Goldstein and Chambless (1978). The ‘fear of fear’ concept manifests in panic disorder as the fear of having further panic attacks, otherwise known as ‘anticipatory anxiety’, and is a critical component of a *DSM-IV* diagnosis of panic disorder. This concept is thought to be responsible for causing the relatively common experience of panic attacks to spiral into disorder. The four most prominent contributing theories to our understanding of a ‘fear of fear’ are, a) conditioning theory, b) sense of control and predictability, c) catastrophic thinking in response to bodily sensations, and d) anxiety sensitivity.

##### *1.4.2.1 Conditioning Theory*

Early conditioning theories posited that, with repeated experience, certain stimuli, events or situations (i.e. conditioned stimuli (CSs) such as being in a supermarket, busy street, enclosed space, etc.) become paired with the occurrence of a panic attack and all of its associated physiological sensations. The learned association that occurs means that a panic attack is likely to occur when the CSs are encountered again. This basic conditioning theory was thought to explain the onset of agoraphobia and situational panic attacks via conditioning to external or exteroceptive cues.

However, with the introduction of the concept of ‘fear of fear’, Goldstein and

Chambless (1978) revised this theory to incorporate the idea of *interoceptive* conditioning. According to their postulation, low-level somatic sensations of anxiety or arousal were thought to become the CSs that provoked higher levels of arousal in the individual. Thus, early somatic components of anxiety, such as a pounding heart were posited as potential sensations that may lead to significant bursts of anxiety or panic. Goldstein and Chambless' model of interoceptive conditioning therefore provided an explanation for the experience of "spontaneous" or seemingly uncued panic attacks in the individual.

Although conditioning theories have yielded fruitful therapeutic approaches to panic disorder primarily in the form of exposure therapies that aim to extinguish the association with fear, criticisms have been levelled towards the conceptual basis of the theory (McNally, 1990, 1994; Klein, 1981). Primarily Klein criticised early conditioning theories (as well as psychoanalytic theories) of panic for not recognising anxiety and panic as distinct states. Furthermore, McNally (1994) argued that, in the context of interoceptive conditioning, the distinctions between the unconditioned stimulus (US), the conditioned stimulus (CS), the unconditioned response (UR) and the conditioned response (CR) have been blurred, with anxiety and/or panic indiscriminately attributed to any one of these positions. For example, low levels of physiological arousal act as the US which becomes paired with high levels of arousal (CS) to elicit the conditioned response of panic (also high arousal).

In an attempt to refute and clarify criticisms regarding conceptual confusion in conditioning theory, Bouton, Mineka and Barlow (2001) have produced a comprehensive modern learning theory account of panic disorder. Citing considerable experimental evidence, they argued that recent evidence suggests that the old

distinctions of US, CS, UR and CR *are* relatively arbitrary, overly simplistic and somewhat inaccurate representations of what occurs in learning behaviour.

The modern learning theory model for panic disorder as proposed by Bouton et al. posits that anxiety and panic are distinct biological systems providing different but complementary evolutionary services. Whereas anxiety prepares the organism for an anticipated trauma (primarily by mobilizing high levels of vigilance and alertness), panic deals with a trauma already in progress (by mobilizing the individual's fight/flight processes). Despite their status as distinct systems, panic and anxiety are seen to interact in a significant manner, in that anxiety has a role in potentiating panic. Furthermore, the development and presence of conditioned anxiety surrounding the panic attack serves to exacerbate subsequent attacks. Thus, as with early learning theory, a fundamental role is assigned to early conditioning episodes (e.g., the first panic attack) in the etiology of the disorder, for establishing conditioning. However, the modern learning theory perspective emphasizes that conditioning is not an inevitable consequence of CS-US pairings. "Instead, the extent to which conditioning develops depends on many additional factors, including the person's previous experience with the CS and the US and with the "informativeness" of the various CSs present on the conditioning trial and on other modulating factors in the background, and so on" (Bouton et al., 2001, p. 24).

Unfortunately however, although Bouton et al.'s modern learning theory embraces greater complexity than earlier versions, this model does little to promote clarity or to shape further investigation as the theory seems able to expand limitlessly to accept each new piece of information about PD pathogenesis. The authors do not propose hypotheses relevant to learning theory that can be unequivocally supported or

not supported by empirical testing. Furthermore, the model entirely fails to account for the occurrence of the initial panic attacks.

#### *1.4.2.2 Sense of Control and Predictability*

The extent to which an individual experiences aversive events as uncontrollable or unpredictable has an influence on the development of psychopathology. With respect to panic disorder, controllability and predictability have been discussed from two separate perspectives. Firstly from a developmental angle, prior experiences with control and mastery, and with unpredictability are suspected to contribute to a non-specific vulnerability for panic disorder (e.g., Bouton et al., 2001). Secondly, and more specifically to panic disorder research, the constructs of controllability and predictability have been manipulated in biological challenge studies to investigate the mediating effects they have on anxiety responses (e.g., Zvolensky & Eifert, 2001).

In their review of modern learning theory and panic disorder, Bouton et al. (2001) drew on evidence from laboratory studies with animals to demonstrate that unpredictable and/or uncontrollable aversive events have a more pronounced impact on anxiety than predictable or controllable aversive events. Furthermore, early developmental experiences with unpredictability and/or uncontrollability may predispose the individual to chronic and heightened anxiety in general in later life, and when exposed to novel and/or stressful situations. In humans, such developmental experiences might come in the form of unresponsive and/or unpredictable parenting, or in the form of a greater than usual impact from stressful life events (such as the loss of a parent).

McNally (1994) discusses the concept of predictability in terms of a person's expectation for catastrophic consequences in the face of panic, or for the occurrence of panic in itself (e.g., "If I drive through this tunnel, I'll have a panic attack). In line with this idea, investigators (e.g., Rapee, Mattick & Murell, 1986) have manipulated the instructions given to participants in biological challenge studies to determine the effect of expectation on anxiety response. Specifically, Rapee et al. found that providing information to panic patients about the effects of CO<sub>2</sub> inhalation reduced the degree of anxiety and catastrophic thinking relative to providing no information. Furthermore, if healthy subjects with high anxiety sensitivity but no history of panic were coached to expect relaxation following CO<sub>2</sub> inhalation, they exhibited higher rates of panic relative to those who were expecting high arousal (Telch & Harrington, 1992).

Other research (e.g., Lejuez, Eifert, Zvolensky, & Richards, 2000) offered support for the importance of predictability in relation to the onset of panic. For example, Lejuez et al. found that healthy participants demonstrated higher self-reported anxiety in response to unsignalled (i.e. unpredictable) CO<sub>2</sub> presentations relative to signalled gas presentations. Similarly, in examining the effect of pre-existing personality differences, Zvolensky, Eifert, and Lejuez (2000) assessed participants for their tendency to view anxiety-related events as predictable or unpredictable. They found that those with a predisposition to view events as unpredictable reported significantly greater emotional and cognitive distress in response to a voluntary hyperventilation task than those with a propensity to perceive predictability.

Zvolensky, Lejuez, and Eifert (2000) argue that control and predictability are linked in a unidirectional manner, insofar as control most often implies prediction.

Therefore, if one has control over an event, one can predict when it will begin and end. However, prediction does not necessarily imply control. In regard to controllability, Sanderson, Rapee and Barlow (1989) dramatically demonstrated the influence of perceived control in the occurrence of panic in 20 patients with panic disorder and agoraphobia. All participants underwent CO<sub>2</sub> inhalation, and were instructed that a light on the panel directly in front of them, if lit up, would indicate that they could reduce the amount of CO<sub>2</sub> they were receiving by turning a dial. In actual fact, the dial was inoperative whether or not the panel light was on, and had no effect on the concentration of CO<sub>2</sub> participants were inhaling. For half the participants the light was on throughout the experiment, giving participants' an illusion of control. For the other half, the light did not come on at all. Eighty percent of subjects with no perception of control reported panic attacks compared to only 20% in the illusion-of-control group. The illusion of control influenced both cognitive and somatic symptoms of panic. Zvolensky and colleagues conducted experiments in which half the participants' *could* actually control the amount of CO<sub>2</sub> that was presented to them. The authors found that participants who had some control over their conditions responded to the biological challenge with significantly less anxiety than those who had no control. These findings held true for both non-clinical participants with high anxiety sensitivity (Zvolensky, Eifert, & Lejuez, 1999; Zvolensky, Eifert, & Lejuez, 2001) and those with high levels of suffocation fears (Zvolensky, Lejuez, & Eifert, 1998).

Although not directly related to control, other studies have manipulated 'safety cues' to provide further support for the hypothesis that psychological factors influence anxious responding in the face of biological challenge. Carter, Hollon, Carson, and Shelton (1995) found that patients with panic and agoraphobia who underwent a CO<sub>2</sub>

challenge in the presence of their “safe” companion demonstrated less distress and physiological arousal than those who had no safe person present. Indeed the responses of those patients undergoing this challenge with their safe person resembled the responses of healthy participants who underwent challenge alone.

Collectively, these experiments demonstrate that the experience of unpredictable and uncontrollable periods of autonomic arousal evoke significant anxiety even among healthy individuals. The effect is greater for those with a predisposition to view the world as unpredictable, or for those fearful of the sensation of physical anxiety symptoms (i.e. those with high anxiety sensitivity). Assuming that laboratory-induced panic attacks are fair approximations of actual panic attacks, how much more frightening must these experiences be for those who experience them ‘out of the blue’, and who are most likely already sensitive to anxiety (given the likelihood of comorbid diagnoses). Clearly psychological variables including beliefs about control and predictability influence how frightening is the initial experience, (as well as how an individual will respond to the experience), and will subsequently impact on fears about further attacks.

#### *1.4.2.3 Catastrophic Misinterpretation of Bodily Sensations*

Modern learning theory (as elucidated above) acknowledges that cognitions can play a role in the development of panic disorder, however the role assigned to them is purely as a conditioned stimulus or as part of the context for the CSs. In contrast, Clark (1986, 1988, 1996) proposed a more central, causative role to the cognitions that develop around the somatic sensations of panic or anxiety. According to Clark, ambiguous physical sensations lead to panic due to the catastrophic misinterpretation of their significance. For example, heart palpitations might be

interpreted as an impending heart attack, dizziness as a sign of imminent collapse, and feelings of being detached from reality as a signal of insanity. The catastrophic cognitions generate increasing anxiety which in turn intensifies the bodily sensations seeming to confirm the individual's worst fears. Spiralling anxiety eventually leads to a panic attack. In this cognitive model, the experience of fear (of fear) in the first instance is deemed nonessential to the generation of a panic attack, as sensations may also arise from other emotional sources, such as anger. Furthermore, somatic sensations might arise from non-emotional sources such as caffeine ingestion.

McNally (1994) notes that the evidence in favour of this theory includes its ability to account for the findings from biological challenge studies, in response to which, patients with panic disorder are far more prone to panic than healthy controls despite the fact that the physiological profiles of the two groups in response to the challenge do not significantly differ. Ley (e.g., 1985) also noted that retrospective studies have indicated that panic patients report thoughts of imminent threat as typically accompanying their attacks and as occurring after the detection of the physical symptoms, but before the emergence of panic. Other studies however have not replicated this clear sequence of events, finding that catastrophic cognitions did not invariably precede panic (e.g., Wolpe & Rowan, 1988), and panic did not invariably follow from catastrophic misinterpretation.

Further empirical support for the central role of catastrophic cognitions in the development of panic lie with the studies mentioned previously that manipulated instructional information prior to lactate infusion challenges with panic disorder patients in order to minimize misinterpretation of the physical sensations elicited (Rapee et al., 1986). Significantly, only 30% of those patients instructed in the effects of lactate infusion panicked in contrast to 90% of controls. Still, it is interesting to

consider that 30% of patients responded with panic despite knowing the origins of their physical symptoms. This leads to another line of criticism for this model which questions why patients with panic disorder persist in their catastrophic misinterpretation of symptoms despite repeated experiences which signal that the panic attack symptoms were not indicators of impending physical or psychological impairment (i.e., cardiac arrest or insanity) (Seligman, 1988). This criticism is countered most effectively by the view held by proponents of the anxiety sensitivity model for the development of panic disorder. If the panic patients' cognitions in response to the physical symptoms of a panic attack centre on the fear of the attack leading to a heart attack, rather than it signalling a current heart attack, it is easier to understand the persistence of these maladaptive cognitions. After all, past experience with panic attacks ending relatively innocuously, does not preclude the possibility that the feared consequence will not happen the next time (McNally, 1994).

Most notably, problems arise for this theory when considering the subgroups of patients who experience nocturnal panic attacks and those who experience "nonfearful" panic. It has been demonstrated that nocturnal panic attacks usually occur in the transition from stage two to stage three sleep, rather than the rapid-eye-movement sleep associated with dreams. "Nonfearful" panic attacks are an interesting phenomenon in which people experience the physical symptoms of a panic attack but without cognitions of danger or threat. In both of these panic phenomena, the presence of any cognitions (catastrophic or no) that coincide with the attacks are seemingly undetectable. Clarke (1986), however addressed these problematic phenomena by suggesting that catastrophic cognitions occur unconsciously. Unfortunately this proposition creates some difficulties with regard to the testability and falsifiability of the theory (McNally, 1999).

#### 1.4.2.4 Anxiety Sensitivity

In a refinement of the Goldstein and Chambless (1978) model, Reiss and McNally (1985) deconstructed the “fear of fear” into two parts - anxiety expectancy and anxiety sensitivity. The anxiety expectancy component (which came to be known as expectancy theory) was aligned with conditioning theory in that it described the process by which an individual came to associate a given stimulus with fear or anxiety. The anxiety sensitivity component was a theoretically novel understanding of “fear of fear” that inspired a new direction in panic disorder research and, more recently, a new theory of human motivation (Reiss, 2000).

Anxiety sensitivity theory predicts that individuals with high anxiety sensitivity are more likely to panic in response to anxiety-related symptoms, and more likely to develop panic disorder (Reiss & McNally, 1985). Anxiety sensitivity (AS) refers to individual differences in what people think will happen to them when they experience anxiety (Peterson & Reiss, 1993). Those with high AS believe that anxiety has dangerous consequences and may lead to heart attacks, mental illness, or social embarrassment. They are therefore more likely to respond to a pounding heart with further anxiety and/or panic. Those with low AS believe that anxiety is an unpleasant but harmless experience.

Anxiety sensitivity theory differs from the other major psychological perspectives of panic disorder in a couple of important ways. With respect to conditioning theory, anxiety sensitivity theory fundamentally differs in its conceptualization of the sequence in which panic and the fear of fear occur. Whereas conditioning theory regards the fear of fear as a *consequence* of past experience with panic attacks, anxiety sensitivity is viewed as an enduring trait which *precedes* the development of panic disorder, and itself develops in response to a range of factors,

including, but not limited to panic experiences (Donnell & McNally, 1990). With respect to differences with the theory of catastrophic cognitions, Clark (1986) proposed that panic arose as a consequence of the catastrophic misinterpretation of benign somatic symptoms (e.g., a skipped heart beat is mistaken for a sign of impending heart attack). Anxiety sensitivity on the other hand, does not require that a person mistake somatic sensations for something else. Instead, particular bodily sensations (e.g., heart palpitations) are dreaded because they are associated with the onset of panic (not a heart attack), and a panic attack is considered highly aversive in and of itself (Reiss & McNally, 1985). This distinction is still somewhat controversial (see Cox, Borger & Enns, 1999). Further, anxiety sensitivity theory extends the theory of catastrophic misinterpretation by proposing that there is natural variation in the tendency to catastrophise in response to somatic symptoms. The former theory proposes that a select group at the high end of the anxiety sensitivity spectrum are more likely to experience the physical symptoms of panic as catastrophic.

The Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1993) was designed to assess the extent of an individual's fear of anxiety symptoms (see Taylor (1999) for review). Its development has spurred extensive interest and empirical study in the anxiety sensitivity construct and its relationship with panic disorder (and more recently with anxious and affective disorders in general). The ASI is a 16-item inventory and was originally proposed to yield a unitary factor structure. Early empirical evaluation of the factor structure of this inventory has yielded inconsistent findings and much debate (see Zinbarg, Mohlman & Hong, 1999 for review). However, consensus in the research has eventually been reached and suggests that the ASI has a hierarchically organized factor structure with a meaningful total score and three interpretable lower-order factors (Rodriguez, Bruce, Pagano, Spencer, & Keller,

2004). The three subscores of the ASI have been labelled physical concerns, concerns about mental incapacitation (a.k.a. psychological concerns) and social concerns. They each relate to a realm of feared consequences of anxiety symptoms, whether it be fear of heart attack or fainting, going crazy, or embarrassing oneself as a result of publicly observable anxiety symptoms. The ASI has been demonstrated to be a valid and reliable instrument (see Peterson & Reiss, 1993).

Clinical studies using the ASI have found that patients with panic disorder score about two standard deviations above the normative mean on the ASI, and significantly higher than those with generalized anxiety disorder, or social phobia (e.g., Taylor, Koch and McNally, 1992; see Cox, Borger & Enns, 1999 for a review). In nonclinical samples, individuals with a history of panic attacks, consistently score higher on the ASI than those without (e.g., Telch, Shermis & Lucas, 1989; Telch, Lucas & Nelson, 1989). Recent ASI item analyses indicate that fears of cardiorespiratory symptoms are especially prominent in panic disorder (Blais et al., 2001). In total, these findings led McNally (1992) to conclude that the fear of panic, as much as the occurrence of panic itself, may distinguish panic disorder from other anxiety disorders. The association between AS and agoraphobia is less well-supported than for AS and panic. Instead Cox, Borger and Enns (1999) proposed that panic expectancy variables might be more important than AS for predicting agoraphobia.

Biological challenge studies (e.g., Rapee, Brown, Antony & Barlow, 1992) and longitudinal studies of anxiety sensitivity (e.g., Schmidt, Lerew & Jackson, 1999; Maller & Reiss, 1992; Ehlers, 1995) have also supported the predictions of anxiety sensitivity theory. Indeed Rapee et al. (1992) found that high anxiety sensitivity was the only variable to predict an anxious response to hyperventilation and CO<sub>2</sub>

challenges in a large group of anxiety patients and healthy volunteers. Moreover, healthy individuals with high ASI scores responded to hyperventilation and biological challenge in the same way as panic patients, reporting more anxiety, more physical sensations (Holloway & McNally, 1987), and comparable rates of panic (Telch & Harrington, 1992). Recent studies, some reanalysing earlier research, (e.g., Zinbarg, Brown, Barlow & Rapee, 2001; Zvolensky, Feldner, Eifert & Stewart, 2001; Carter, Suchday, Gore, 2001) have shown that only elevated scores on the physical concerns subscale of the ASI were linked to a fearful response to biological challenge or hyperventilation. These findings are consistent with the notion advanced by McNally (1994, 2002) that biological challenge studies induce panic by provoking particular physical sensations that certain individuals find frightening.

Prospective studies of anxiety sensitivity have established it as a risk factor for the onset and maintenance of panic attacks and anxiety disorder (e.g., Ehlers, 1995; Maller & Reiss, 1992; Schmidt, Lerew & Jackson, 1999). Maller and Reiss (1992) followed-up college graduates who had scored either high or low on the ASI three years earlier. High-AS students were five times more likely than low-AS students to have a *DSM-III-R* anxiety diagnosis at follow-up. Furthermore, three of four students who had experienced panic attacks for the first time in the three-year follow-up period were from the high-AS group. In two separate studies, Schmidt and colleagues assessed a large group of cadets both before and after embarking on a highly stressful 5-week basic training program for the U.S. Air Force Academy. About six percent reported a spontaneous panic attack during basic training (approximately half of whom had no prior history of panic). Pre-test scores on the ASI predicted the emergence of spontaneous panic during basic training, even after controlling for trait anxiety and a history of panic. An examination of ASI subfactors indicated that only

the Mental Incapacitation subscale (MI-ASI) predicted panic in this group of cadets (Schmidt, Lerew & Jackson, 1997; Schmidt et al., 1999). That is, those people who experienced a panic attack in response to the basic training program were more likely than the other cadets to fear symptoms such as nervousness and an inability to concentrate as signs of impending mental illness as measured by the MI-ASI. Further analyses of the pre- and post- test data from Schmidt et al.'s studies revealed that while elevations on the ASI predicted panic, the occurrence of panic in turn increased anxiety sensitivity (Schmidt, Lerew, & Joiner, 2000). Taken as a whole, the above findings suggest that individuals who panic in response to biological challenge, have distinctly different cognitive concerns or profiles than those who panic in response to an emotionally stressful situation. This proposition supports the idea of panic disorder as a heterogeneous phenomenon.

As a result of evidence supporting its predictive validity, anxiety sensitivity has come to be viewed as an early risk factor for spontaneous panic, and panic disorder (McNally, 2002). Further support for this proposition comes from evidence that a subgroup of individuals scoring highly on the ASI have never panicked (e.g., Donnell & McNally, 1990), which McNally (2002) has argued is a necessary criterion for a risk factor. Donnell and McNally's examination of the relationship between anxiety sensitivity and panic attacks amongst American college students revealed that, of those demonstrating elevated ASI scores, approximately 70% had never had a spontaneous panic attack. In addition, Cox, Endler, Norton, and Swinson (1991) found that 50% of their sample of college students had never had a panic attack at all, neither spontaneous nor situational. These studies indicate that fear of anxiety symptoms can be acquired in ways other than through direct personal experience with panic.

The origins of anxiety sensitivity are purported to reflect both genetic and current cognitive components. That is, variability in anxiety sensitivity between individuals is a consequence of natural variation in the genes that render anxiety a displeasure, as well as individual differences in beliefs about the personal consequences of anxiety experiences (Reiss, 2000). Genetic studies have found support for a heritable component of anxiety sensitivity (Stein, Jang & Livesly, 1999; Schmidt, Storey, Greenberg, Sandiego, Li & Murphy, 2000; van Beek & Griez, 2003). In a twin study, Stein and colleagues (1999) found that additive genetic effects and unique environmental effects both emerged as strong influences on anxiety sensitivity. A later gender analysis of this data found that anxiety sensitivity factors were heritable only in women (accounting for approximately 45% of the variance), whereas environmental factors accounted for all of the variance for men (Jang, Stein, Taylor, & Livesley, 1999).

Reiss (2000) postulated that cognitive factors (in the form of beliefs about the consequences of anxiety symptoms) would significantly modify a person's inherited sensitivity to anxiety. Researchers have only begun to investigate and identify which experiences shape beliefs consonant with high anxiety sensitivity. The research that exists (i.e., Ehlers, 1993; Watt & Stewart, 2000; Watt, Stewart & Cox, 1998) has implicated both parental modelling and reinforcement of sick-role behaviour and fear reactions for both anxiety-related (e.g. dizziness) and non-anxiety related symptoms (e.g. colds or lumps) as being associated with the development of childhood high anxiety sensitivity. Interestingly, these studies demonstrated that, in contrast to the findings for high AS, only parental reinforcement of anxiety-related symptoms (and not general somatic symptoms) differentiated participants with a history of panic from those without. Using structural equation modelling, Stewart et al. (2001) concluded

that anxiety sensitivity acted as a mediating variable between childhood learning experiences and the development of panic attacks in young adulthood. However it should be noted that childhood learning experiences acted directly on both anxiety sensitivity AND panic frequency.

Childhood exposure to parental loss of control due to anger or alcohol (Watt, Stewart & Cox, 1998; Watt & Stewart, 2003), and parental threatening, hostile and rejecting behaviours (Scher & Stein, 2003) have also been implicated in the development of high anxiety sensitivity. For example, Watt and Stewart (2003) investigated the relationship between elevated anxiety sensitivity and parental dyscontrol related to drinking, anger or other negative emotional states. Specificity emerged between AS components and particular dyscontrol experiences. In particular, childhood exposure to parental loss of control related to negative emotional states (i.e., anger, anxiety, depression) was significantly positively correlated with all three AS dimensions, whereas exposure to loss of control associated with drinking was significantly correlated with the psychological concerns factor of the ASI only. Furthermore, the psychological concerns factor was the only one found to mediate the relationship between childhood exposure to parental dyscontrol and panic symptoms in adulthood. Interestingly Watt and Stewart found that, whereas panic symptoms appeared more specifically related to parental loss of control associated with anger and drinking, general anxiety symptoms were associated with parental dyscontrol arising for any reason.

Scher and Stein (2003) explored the role of exposure to parental threatening behaviour and hostile/ rejecting behaviour in the development of anxiety sensitivity and its lower-order components. Drawing on attachment theory and previous research they predicted that both sets of behaviour (i.e., threatening and

hostile/rejecting) would be related to the development of overall anxiety sensitivity, and that the behaviour sets would be differentially related to the ASI factors and in turn differentially related to anxiety versus depressive symptomatology. Their predictions were supported in a sample of nonclinical undergraduate students. Both sets of behaviours (i.e., threatening and hostile/ rejecting) significantly predicted overall anxiety sensitivity level, with parental threatening behaviours bearing the strongest relationship. Furthermore, parental threatening behaviours predicted fears of publicly observable anxiety symptoms (i.e., the social concerns factor of the ASI), which in turn predicted adult anxiety symptomatology. In contrast, parental hostile/ rejecting behaviours predicted fears of losing cognitive control (i.e., the psychological concerns factor of the ASI) which in turn predicted depressive symptomatology. Taken together with the findings of Watt and Stewart, Scher and Stein's results support the notion that individual differences in anxiety sensitivity may be attributable in part to differences in childhood experiences, perhaps specifically related to parent-child interactions.

Two major criticisms of the anxiety sensitivity approach have referred to a) the likelihood that the construct is seriously confounded with trait anxiety (Lilienfeld, Turner, & Jacob, 1989; Lilienfeld, Turner & Jacob, 1993), and b) its lack of specificity as a vulnerability factor for panic disorder (Bouton et al., 2001). With regard to the former criticism, multiple studies have since demonstrated that the ASI predicts variance in fearfulness beyond that predicted by measures of trait anxiety (e.g., McNally & Lorenz, 1987) and is better able to discriminate between panic disorder and other anxiety disorder populations ( e.g., Taylor, Koch & McNally, 1992). With regard to the latter criticism, and to comments that anxiety sensitivity is only a moderate, albeit significant, predictor of panic variables, McNally replied that

the ASI (like other measures of the fear of fear) is only one part of the causal picture. Furthermore, he argued that research into anxiety sensitivity had indisputably established that cognitive factors must figure in any satisfactory analysis of the etiology of panic disorder. To conclude, high anxiety sensitivity, although not uniquely associated with panic disorder, is postulated as a cognitive risk factor for the development of panic disorder (McNally & Lorenz, 1987).

#### *1.4.2.5 Summary*

Psychological accounts of panic disorder have primarily examined the influences of conditioning, cognitions and beliefs in the onset and maintenance of this condition. Research into the psychological variables of panic disorder pathogenesis has been instrumental in furthering our understanding of panic disorder development.

While the process of interoceptive conditioning, may contribute to the generation of panic attacks and to the development of a fear of further panic, the theories of catastrophic misinterpretation of bodily sensations and anxiety sensitivity have offered more parsimonious and compelling explanations of these components of panic disorder. Proponents of interoceptive conditioning theory such as Goldstein and Chambless (1978) have argued that, with repeated exposure, panic attacks become associated with, and subsequently are evoked by, particular physical symptoms such as a racing heart. In addition, the modern proponents of this theory (e.g., Bouton et al., 2001) have acknowledged that an individual's cognitions and beliefs about control, predictability and safety will have a moderating influence on the ability of the conditioned stimulus (e.g., heart palpitations) to evoke a panic attack.

As compared with theories on panic disorder development based on conditioning, beliefs and cognitions are afforded a more dominant role in the

development of panic disorder in the theories of catastrophic misinterpretation (e.g., Clark, 1986) and anxiety sensitivity (McNally, 2002). Proponents of both of these theories have argued that individuals with panic disorder respond fearfully to certain physical sensations because they believe that these sensations (might) signal catastrophic consequences such as an impending heart attack or mental illness. According to McNally (1994), a significant difference between the two theories is that anxiety sensitivity theory holds that certain individuals (i.e., those with high anxiety sensitivity) are more prone to fear the consequences of their anxiety symptoms than others (i.e., those with low to moderate AS), and therefore more likely to respond to anxiety symptoms with panic. Anxiety sensitivity theory in particular has undergone considerable research and received extensive support for its role as a cognitive risk factor in the development of panic disorder.

Despite significant advances in our understanding of panic disorder development, the knowledge base, as it stands to date, is far from complete. Research into the childhood experiences that shape anxiety sensitivity has only just begun and much work remains to be done. Similarly, little systematic research exists with regard to understanding the processes by which an individual comes to perceive the world as unpredictable, or to routinely and catastrophically misinterpret somatic sensations.

Research investigating the necessary conditions for individuals identified as at risk for panic disorder (e.g., those with high anxiety sensitivity) to actually develop it, and the conditions which protect others from developing the disorder is also rare. Schmidt and Lerew (2002) demonstrated a possible protective factor in their study of new air force cadets. They found that high individual perceptions of control regarding basic training were protective against panic for those with high anxiety sensitivity.

Finally, although research has suggested that the pathways to panic disorder may be heterogeneous, little research exists to actually examine and compare these potentially different pathways. For example, the research into anxiety sensitivity seems to indicate that some individuals with high AS are more prone to fear anxiety symptoms which they interpret as signalling mental illness, while others respond more fearfully to anxiety symptoms which they perceive as signalling a possible physical illness. It would be interesting to know whether these apparent differences between individuals who all present with high AS, have emerged from qualitatively different life experiences, and whether they would all manifest similar pathology in the event that panic disorder were to develop.

### 1.5 Chapter Summary

The *DSM-IV* specifies that, in order for a diagnosis of panic disorder to be made, an individual must have a history of both recurrent panic attacks, and also anticipatory anxiety – that is, or a fear of having further panic attacks. In general, biological accounts of panic disorder have focused on illuminating the physiological pathways and triggers that lead to a panic attack. By contrast, psychological theories of panic disorder have examined more closely the anticipatory anxiety component of panic disorder which is also referred to as the ‘fear of fear’. Both of these approaches have been important for furthering our understanding of panic disorder pathogenesis. Furthermore, much of the research that has been conducted, both biological and psychological, suggests that panic disorder may be a heterogeneous phenomenon, with multiple possible pathways or mechanisms to its development. For example, childhood anxiety disorders are prevalent for a proportion of cases of panic disorder patients; hyperventilation has been implicated in some cases of panic disorder but not

others; and although high anxiety sensitivity appears to be predictive of panic disorder development, people with low to moderate AS are certainly not precluded from it.

## Chapter 2: Attachment Theory and Psychopathology

Although current research into panic disorder has greatly expanded our knowledge of the possible mechanisms at work in the development of panic attacks and anticipatory anxiety, it has been less successful in providing a framework for understanding the broader emotional and interpersonal difficulties common to many panic disorder patients. By contrast, attachment theory has proven itself to be a valuable paradigm for understanding both normal human functioning and the development of psychopathology from a more holistic perspective.

Attachment theory proposes that a person's intrapersonal traits and interpersonal style/processes develop as a function of the security of his/her relationship with his/her primary caregiver (usually the mother) as a child. That is, proponents of attachment theory (e.g., Bowlby, 1969; Ainsworth, 1973; Main & Weston, 1982) have argued that an early relationship that is predominantly secure and is characterised by a caregiver who is sensitive to the needs of her child, and who is able to provide a predictable and relatively safe environment will produce a child (and subsequently an adult) who is secure in his/her ability to master the environment and his/her own emotions. By contrast, an early relationship with the caregiver that is characterised by unpredictability and/or rejection or hostility will produce a child, and subsequently an adult, who perceives the world as threatening and unsafe, and who responds to it accordingly. Thus attachment theory predicts that an individual developing out of the latter scenario, will be more likely to exhibit methods of thinking, behaving and relating to others that lead to maladaptive outcomes for mental health.

Evidence emerging from attachment studies (e.g., Ainsworth, Blehar, Waters, & Wall, 1978; Urban, Carlson, Egeland, & Sroufe, 1991; Sroufe, Carlson, &

Shulman, 1993), from neurological studies examining child brain development in the context of the infant-mother relationship (e.g., Schore, 1994), and from studies in developmental psychology (e.g., Stern, 1985) have generally supported the propositions of attachment theory. That is, that attachment relationships have fundamental implications for a child's emotional and relational development, and for that child's subsequent mental health.

### *2.1 Attachment Theory: A summary*

Bowlby (1969, 1973) proposed that human beings had evolved an innate and sophisticated behavioural system that was designed to protect their young, thereby increasing the odds for survival of the entire species. Essentially this behavioural system manifested itself as the reciprocal bond between mothers and their children, in which the mother noted and attended to her infant's signals of fear or distress by offering comfort and protection and a secure base from which the infant could explore its environment. Bowlby referred to the bond between the child and the mother as an 'attachment' which could be denoted as 'secure' or 'insecure'. He noted that, a secure relationship was one in which the child demonstrated confidence in the availability, accessibility and support of its attachment figures in the event that it encountered a difficult or alarming situation. The term 'attachment figure' generally referred to the mother, although it could in reality be any differentiated and preferred individual.

Bowlby (1969) described his theory as a marriage of ethology and psychoanalysis. He drew on both naturalistic observation of children in institutionalised care and animal behaviour, as well as laboratory studies with animals that manipulated rearing conditions to develop his basic principles of infant development. Later theorists (e.g., Ainsworth, Bell, & Stayton, 1972; Blehar, Lieberman, & Ainsworth, 1977; Grossman & Grossman, 1991; Main & Weston,

1981) further developed attachment theory by drawing on observations of ‘normal’ infants and children both under naturalistic and experimental conditions. Whereas Bowlby theorized about the evolutionary role of attachment for the human race as a whole, Mary Ainsworth’s work, in particular, contributed significantly to the understanding of individual differences in attachment behaviours (e.g., Ainsworth, 1973; Ainsworth et al., 1978). Specifically, Ainsworth and her colleagues developed a system designed to test and categorise the quality of a child’s attachments. This protocol, known as the ‘strange situation’ test, played a major role in the empirical validation of many of Bowlby’s theoretical constructions.

Based on direct observations of young children who had been admitted to hospital and who were not permitted access to their parents (e.g., Bowlby, Robertson, Rosenbluth, 1952), Bowlby observed that a temporary separation from the mother evoked a predictable pattern of attachment behaviours in the child. These behaviours included protest at the separation (e.g., crying and screaming), actively seeking the mother (e.g., following the mother, or searching for her when separated), together with watchful monitoring of the environment. If separation was brief and uncomplicated, then there was pleasure upon reunion. At this stage the child still retained a strong expectation of the mother’s return and alternative attachment figures were rejected. In the event that separation was prolonged, protest seemed to give way to despair, and then detachment. During the *despair* stage there appeared to be an increasing sense of hopelessness in the child, with the child becoming pre-occupied, withdrawn and inactive. There was also a decrease in the child’s physical movement. Beyond this stage, Bowlby noted that the child appeared to partially recover, in that s/he accepted care from alternative figures, however s/he appeared to relate to others

superficially. Upon subsequent reunion with the mother, the child was likely to initially reject her or relate in a superficial and detached manner.

In order to confirm Bowlby's observations, Ainsworth and colleagues (Ainsworth et al., 1978) developed the 'Strange Situation' test. This protocol involved the close observation of mothers and their 12-month old infants during very brief separations and reunions in a laboratory setting. The Strange Situation protocol allowed Ainsworth to observe and classify the qualitatively distinct patterns of attachment behaviours that emerged from their observations of the infant-mother dyads. Specifically, Ainsworth and colleagues observed three general attachment styles – secure, insecure- avoidant, and anxious-ambivalent, which captured the main ways that infants tended to cope with the stress of being left alone by their mothers in a 'strange situation' involving novel toys and an unfamiliar experimenter.

The secure attachment category applied to the majority of infants (about 65%) observed by Ainsworth and colleagues. These infants became somewhat distressed or subdued in their mother's absence, but expressed warm, relieved greetings at her return, were quickly soothed by her and were subsequently able to resume a relaxed play.

Those infants not classified as secure were all considered to demonstrate insecure attachment. These remaining infants however demonstrated two distinctly different ways of coping with the strange situation. Some (about 20%), classified as 'avoidant', demonstrated minimal interest in the caregiver both during her presence and absence. Upon being re-united with the mother, the avoidant child did not approach her for comfort, either ignoring or avoiding her, and appeared to be prematurely self-reliant. The final group (representing about 15%), labelled anxious-ambivalent or resistant, protested and cried upon separation with the mother and in

her absence. Upon reunion with her, the ambivalent child sought to be held by the mother but continued to be angry, distressed and unpacified by her presence.

Furthermore, anxious-ambivalent children remained unable to return to exploratory play.

Interestingly, Ainsworth and colleagues found that the children's reunion behaviours were more indicative of their attachment status, than their level of distress at separation. Amongst the secure group there were some infants who showed little distress and sought minimal contact with the mother at reunion, similarly to the avoidant group, and some who were greatly distressed and were slow to be comforted following reunion, as with the anxious-ambivalent group. Nevertheless, it was their competence in expressing their needs to their mothers, their generally positive approach to her, and an unambivalent acceptance of her ministrations that led to them being classified as secure.

At a later date, Main and Solomon (1986, 1990) reviewed the data of many children who had been unable to be assigned to the existing categories. This work led to the recognition of a fourth, less common and more severe, style of insecure attachment denoted as 'disorganised/ disoriented'. These children demonstrated unusual behaviours such as freezing, hitting the mother, and avoiding approaching the mother directly even when they were very distressed. A child who was classified as 'disorganised' was also assigned a "forced" classification of one of the other three categories that best represented them in order to provide some information as to their attachment strategies in the strange situation.

A child's attachment style, as assessed by the strange situation, has been correlated with a variety of indicators of social adjustment (e.g., Urban et al., 1991) and cognitive competence (e.g., Matas, Arend, & Sroufe, 1978). For example, two

year olds classified as secure in infancy have been demonstrated to have longer attention spans than toddlers classified as insecure infants. As two year olds, securely-attached infants also demonstrated more complex symbolic play (Slade, 1987), more confidence in using tools (Matas et al., 1978), and were more likely to elicit their mother's help in difficult tasks (Bretherton, 1985). Furthermore, the positive effects of a secure-attachment in infancy appeared to extend into early childhood, so that 6 year olds with secure attachment histories were seen to play more concentratedly and for longer, were more socially skilful in handling conflict with their peers, and had more positive social perceptions compared with children classified as insecure in infancy.

As preschoolers (Sroufe, 1979, 1983; Sroufe, Fox, & Pancake, 1983) children with securely attached histories showed themselves to be less dependent on teachers than those with insecure histories. These children *did* seek teacher attention, but in more positive ways, and not at the expense of peer relations. Furthermore, children with secure histories were rated as more ego-resilient by their preschool teachers (who were unaware of the children's attachment histories) and as more socially competent with their peers than children with anxious attachment histories. Differences in dependency and social competency continued to be manifest at ages 10 (Urban et al., 1991) and 15 (Sroufe et al., 1993) with adolescents with avoidant and ambivalent histories showing more dependency on adults than those with secure histories, and greater acceptance and ease of interaction with peers (Shulman, Elicker, & Sroufe, 1994; Sroufe, Bennett, Englund, Urban, & Shulman, 1993). Taken together, the above findings suggest that the security of the infant-mother attachment relationship appears to have important implications for the child's ongoing adaptive functioning.

A central tenet of Bowlby's (1969, 1973, 1980, 1988) theory on attachment was that a child's early attachment experiences with his/her primary caregiver were internalised as working models that served as a template on which notions of the self, significant others and their inter-relationship were represented. These internal working models (IWMs) thus formed a prototype for future relationships with significant others. In addition, Bowlby proposed that IWMs contained the unwritten rules for how a child experienced, expressed, and coped with distressing emotions. As such, the three attachment classifications delineated by Ainsworth et al. (1978) were seen to be outward manifestations of the child's IWMs, and thus to represent and reflect the child's organised strategies for managing stressful situations in the context of a mother who was/ was not able to assist with his/her efforts to regulate distress. Finally, Bowlby envisioned that an individual's early relationship with his/her primary caregiver would have a lasting influence that would manifest itself in his/her future style of relating, both with him/herself and with the world in general.

Some interesting studies (e.g., Cassidy, 1988; Cassidy, Kirsch, Scolton, & Parke, 1996) have provided indirect support for the influence of internal working models on a child's perceptions of him/herself and the world in which he/she lives. For example, Cassidy (1988) found that securely attached children at age six were more able to give a balanced account of themselves as good, but not perfect, while insecurely attached children saw themselves as either faultless or bad. In addition Cassidy et al. (1996) found that securely attached children in kindergarten and Year 1 were more likely to assign benign motives to story characters depicted with ambiguous intent in the context of a negative peer-related event. In contrast, insecurely attached children were more likely to infer hostile intent. Most

importantly, these representations of peer relations were found to predict individual differences in peer social status.

Finally, Ainsworth and colleagues found that the mother's ability to be responsive to the child's distress as assessed by the strange situation was observed to be an important determinant of the child's attachment-security status (e.g., Blehar et al., 1977). Other authors (e.g., Kagan, 1982) have proposed that a child's temperament plays at least as significant a role in determining his/her later attachment status as maternal responsiveness.

## 2.2 *Determinants of Infant Attachment Status*

Research has indicated that the security of an infant or child's attachment status is primarily determined by the extent of its mother's responsiveness and her sensitivity to its needs (e.g., Bell & Ainsworth, 1972). Infant temperament has also been implicated as a determinant of attachment status (e.g., Kagan, 1982).

The extent to which maternal responsiveness and temperament either contribute uniquely or interact to determine an infant's attachment status, as assessed by the Strange Situation, has been keenly debated (see Vaughn & Bost (1999) for a review). On the one hand, attachment theorists argued that relationship history totally transformed constitutional temperament variation such that temperament made little or no contribution to the quality of attachment (e.g., Sroufe, 1985). On the other hand, temperament theorists argued that infant temperament (particularly the infant's susceptibility to distress) played a significant, albeit small, role in determining resistant/anxious-ambivalent behaviour (e.g., kicking legs, pushing away upon reunion); a role that was argued to be at least as important as the role played by maternal sensitivity (e.g., Goldsmith & Alansky, 1987). Furthermore, Kagan (1982) advanced the claim that temperament was the principle determinant of those

behaviours used by the Strange Situation to evaluate attachment security. Thus he claimed that assessments of attachment security using the Strange Situation were confounded by measures of temperament.

Those studies conducted by attachment theorists (e.g., Ainsworth & Wittig, 1969; Bell & Ainsworth, 1972; Blehar et al., 1977; Bretherton, 1991; Main & Weston, 1982; Grossman, Grossman, & Schwan, 1986; Sroufe, 1979) have demonstrated that a child's attachment status at 12 months of age is more strongly correlated with the mother's responsiveness to and attunement with the child, than with other infant-derived variables such as temperament. For example, Bell and Ainsworth (1972) conducted a naturalistic, longitudinal study in which 26 infant-mother pairs were observed at home at approximately three week intervals over the course of the infants' first year of life. They found that maternal responsiveness, in terms of the mother's consistent and prompt responding to the infant's cries, was associated with a decline in the frequency and duration of infant crying over the year. More specifically, they found that, by the time the infants were 12 months of age, individual differences in crying reflected the mothers' history of responsiveness, rather than any initial constitutional differences in infant irritability.

Overall, infants classified as secure at 12 months of age in the Strange Situation have been identified as having mothers who were rated in earlier interactions with her infant as more responsive and encouraging of interaction than the mothers of insecurely-attached infants (e.g., Blehar et al., 1977). In addition, mothers of avoidant infants have been classified as consistently unresponsive and averse to close physical contact, while mothers of anxious-ambivalent infants were rated as inconsistently responsive (Ainsworth et al., 1978).

With regard to the relative contributions of temperament and maternal sensitivity in determining infant attachment status, a review of both attachment and temperament studies by Vaughn and Bost (1999) led them to state definitively that neither attachment security nor temperament could be considered redundant in explanations of personality or qualities of interpersonal interaction. However, whereas maternal sensitivity contributed strongly to both secure and insecure attachment classifications, infant temperament variables such as proneness-to-distress, appeared to play a significant role in determining an insecure, but not a secure attachment organisation.

Crockenberg's (1981) findings support the idea that temperament and maternal responsiveness combine to determine the infant's attachment status. In particular, he found that infant irritability interacted with a mother's low levels of social support and/or economic resources to predict a subsequent insecure categorisation on the Strange Situation, but only for the more difficult, irritable infants. This finding suggests that attributes of the infant may potentiate inadequate caregiving practices (i.e. reduced maternal sensitivity) when caregivers themselves are also stressed. In this regard, van den Boom (1990) found that an intervention designed to heighten maternal sensitivity for mothers of irritable infants, had the further effect of improving attachment quality.

In line with the above findings, Crockenberg (1981) proposed a model of Goodness of Fit to explain the interaction between maternal characteristics and infant temperament. This model predicted that attachment security (as well as other aspects of interpersonal relationships) emerged as a consequence of the interplay between both members of the infant-mother dyad. Consistent with this view, Mangelsdorf, Gunnar, Kestenbaum, Lang, and Andreas (1990) found that an insecure attachment

relationship was more probable when infants who were prone to high levels of distress had mothers who were high on a measure of constraint/ rigidity. Finally, Belsky, Fish and Isabella (1991) concluded from a review of the literature that infant temperament influenced attachment via its mediating influences on the processes of infant-mother interaction. They predicted that similar mediating effects would occur for all other factors that impacted on the proximal processes of the parent-child interaction, such as the psychological health of the mother, the quality of her marriage, and degree of social support.

The implications for the overall findings that maternal responsiveness and temperament interact to determine the infant's attachment security, are that maternal sensitivity entails sensitivity to a particular infant with a particular set of characteristics and demands. Eagle (1995) proposed that proper maternal responsiveness to a very difficult infant would enable that infant to become securely attached. However he argued that the greater demands on the mother of a very difficult infant might mean that its demands were NOT successfully met. As such a mother viewed as adequately responsive by observer standards could theoretically be experienced and perceived differently by infants endowed with different temperaments. Indeed, Eagle's proposition embodies the concept of the 'Goodness of Fit' model.

Suomi's (1997) work with Rhesus monkeys has lent considerable weight to the proposition that the mother-infant goodness of fit plays an important role in determining healthy outcomes for the child. Suomi found that about 20% of infant rhesus monkeys exhibit inborn profiles of inhibition in the face of novelty, which was similar to the profiles of behavioural inhibition (BI) in children. In children, BI refers to the likelihood that the child will respond to novel stimuli with distress and

avoidance, and is considered to be a temperamental trait. Suomi found that when these behaviourally inhibited, and highly physiologically reactive rhesus infants were fostered by highly nurturing rhesus mothers, they left their mothers earlier, explored their environment more, and became more socially adept, than those highly reactive infants who were fostered by normal mothers. Furthermore, the infants reared with highly nurturing mothers developed into more well-adjusted, independent adolescents than even those monkeys who were initially considered to be physiologically non-reactive (that is, they demonstrated the equivalent of a non-anxious temperament). This study highlights the importance of assessing the contributions of both the mother and the infant to the security of the mother-infant attachment relationship.

Bowlby (1973) proposed that attachment behaviours become activated from about six months of age, when the infant begins to be mobile. Prior to six months of age, the infant does not seem to be overly discriminant in identifying with any one constant figure, but relies on the caregiver attending to its needs in a timely manner. Under normal circumstances, attachment behaviours are dormant and unobservable but are triggered by separation (or the threat of separation) from attachment figures, or by the occurrence of frightening or unfamiliar stimuli. They can then be terminated or assuaged only by proximity to the attachment figure. Bowlby observed that the degree of proximity required for the termination of attachment behaviours (e.g., eye contact alone versus close proximity and physical contact) varied as a function of the nature and intensity of the apparent threat.

Although attachment behaviours are most easily observable in infancy and childhood, Bowlby insisted that attachment was a lifelong process, from infancy through childhood, adolescence and adulthood. It involved a gradual shifting of the balance of responsibility for the maintenance of proximity – from the caretaker to the

developing child; and a change in the figure(s) towards whom attachment behaviours were directed – from parental figures to peers or other adults (including the individual's own adult children in later life). Observable attachment behaviour in adults, in terms of becoming especially demanding of others, was proposed to be most likely elicited in times of sickness and calamity, or sudden danger or disaster. Bowlby insisted that it was a mistake to see such behaviour as regressive and indicative of pathology as this kind of behaviour was a fundamental part of human experience. Just as Ainsworth's work was instrumental to the understanding of individual differences in attachment styles amongst normal (that is, nonclinical) infants, Main and colleagues (e.g., Main, Kaplan, & Cassidy, 1985) and Hazan, Shaver and colleagues (e.g., Hazan & Shaver, 1987; Shaver, Hazan, & Bradshaw, 1988) have been instrumental in establishing systems capable of classifying attachment differences amongst adolescents and adults.

### *2.3 Assessment of Attachment in Adolescence and Adulthood*

In order to enable attachment research to extend beyond the study of infants, Main and her colleagues developed the Adult Attachment Interview (AAI) as a means of assessing adult attachment status (Main et al., 1985). The AAI is a semi-structured interview for adults about their childhood attachment experiences and the meanings they currently assign to past attachment-related experiences. Adults were classified into attachment categories based on the discourse analysis of their AAI transcripts. Discourse analysis was designed to assess an individual's ability to give an integrated and believable account of his/her personal experiences and the meanings he/she attributed to them. Main and colleagues interviewed mothers whose infants had been classified according to the strange situation. Based on these interviews they generated four attachment categories that corresponded well with the infant classification

scheme. Adults classified as *autonomous* (corresponding with the infant classification of *secure*), appeared to value the importance of intimate relationships. They were able to provide a coherent narrative with confirming details of both positive and negative childhood experiences. They were also able to be thoughtful about both their parents' and their own contributions to these experiences. Those adults classified as *dismissing* (which was associated with the infant classification of *insecure-avoidant*), tended to minimize the effect of intimate relationships in their lives. They tended to idealise their relationships with their parents, and had difficulty providing concrete details to illustrate the generalizations they made about their childhoods. By contrast, adults in the *preoccupied* group (which corresponded with *insecure-ambivalent / resistant* infants) had little reflective understanding of their attachment histories. They quickly became entangled in details of their early experiences and were still engaged in struggles with their parents. The unresolved group (which corresponded with the *disorganized* infant group) demonstrated lapses in coherency and disorganization of thought when discussing events concerning attachment, loss or trauma, such as would normally occur at the time of the actual experience (e.g., references to a deceased parent as if s/he were still alive) (refer to Crowell, Fraley & Shaver, 1999 for a more complete discussion of the AAI and its correlates).

Grossman and Grossman (1991) proposed an interesting interpretation to explain the finding of an apparent relationship between the mother's ability to give a coherent account of her own experiences, and her behavioural sensitivity to her child. They observed that the infant's cry or distress appeared to alarm its parent and elicit ambivalent feelings from her. They argued that mothers who exhibited insecure attachment states of mind, may have themselves lacked the experience and model of an attachment figure who had been capable of integrating the negative feelings

associated with a crying infant into emotional empathy and active assistance to the child. If, instead of empathy and comfort, these mothers had experienced frequent rejection or unsupportiveness in response to their own distress, such parents might seek to exclude from their perception the reminder of their own former misery, which their infant's distress represented, by ignoring the infant's crying. Such a pattern would be expected to manifest in observations of low maternal sensitivity in a strange situation, as the mother essentially turns away from her infant's distress management to the management of her own emotional conflicts. As a consequence, the infant would experience its mother on the whole as emotionally unavailable when it needed her most. In accordance with attachment research (e.g., Ainsworth et al., 1978) one would then expect the infant to develop an avoidant attachment pattern in response to its mother's emotional unavailability.

Due to the labour-intensiveness of the AAI classification system, questionnaire measures of adult attachment were developed to capture adults' perceptions of their childhood attachments with their parents. A profusion of self-report techniques (primarily questionnaires but also some interviews) have also been developed to assess attachment quality and patterns between partners in romantic relationships for adolescents and adults, and to assess attachment styles to parents and peers in adolescence and adulthood. They differ from those methods accessing autobiographical discourse as they require a conscious self-assessment of relationship patterns that is not required in the former assessment technique.

Measures assessing romantic adult and adolescent attachments are based primarily on the work of Hazan and Shaver (1987; Shaver et al., 1988). These brief questionnaires asked subjects to nominate a series of descriptions of relationship styles as being relevant (or not) to them. Hazan and Shaver derived their relationship

descriptions from Ainsworth's three infant attachment styles of secure, insecure – ambivalent and insecure- avoidant. From scores of studies with college students and adults (see Shaver & Hazan (1993), and Rothbard & Shaver (1994) for reviews), a portrait has emerged of the three kinds of individuals identified by this attachment measure. Securely attached adults are self-confident, socially skilled, open to and interested in close relationships with romantic partners, and likely to form relatively stable and satisfying long-term relationships. Anxious (or anxious–ambivalent) adults lack self-confidence, are worried about rejection and abandonment, are prone to bouts of jealousy and anger at relationship partners who are perceived as untrustworthy, are eager to become involved in romantic relationships despite their perils, and are likely to engage in inappropriately intimate self-disclosures, to fall in love quickly (and perhaps indiscriminately), and to experience frequent break-ups and reunions. Avoidant adults may or may not be interested in close relationships, but nevertheless they are uncomfortable with closeness, are disinclined to become involved in long-term romantic relationships, are uncomfortable with self-disclosure, and are relatively inhibited and socially unskilled.

Hazan and Shaver's (1987) three-fold typology of attachment styles was subsequently elaborated by Bartholomew (e.g., Bartholomew & Horowitz, 1991) into a two-dimensional, four-fold model. Conceptually, Bartholomew (1990) based her model on Bowlby's (1973) ideas that an individual's internal working models contained a representation of the self as either positive or negative, and a general representation of significant others as either positive or negative. Conceptually, individuals with a secure attachment style possess relatively positive (+ve) models of both self and others, whereas those with anxious-ambivalent styles (whom Bartholomew renamed "preoccupied") possess a +ve model of others, but a negative

(-ve) model of self. Notably Bartholomew differentiated Hazan and Shaver's insecure-avoidant attachment style into two different types of avoidant attachment. According to Bartholomew, Hazan and Shaver's avoidant type seemed too vulnerable and low in self-esteem when compared to Main's (Main et al., 1985) description of 'dismissing' adults. Bartholomew labelled one of her avoidant categories 'fearful avoidant' and the other 'dismissing avoidant'. Fearful avoidants were considered to be akin to Hazan and Shaver's avoidant type. Conceptually they are thought to possess -ve models of both self and others, so that they both desire and fear intimacy. In contrast, dismissing avoidants possess a +ve model of the self but a -ve model of others. Bartholomew and Horowitz (1991) demonstrated that the two kinds of avoidants differed as expected. Although both kinds identified themselves as "socially avoidant", fearful avoidants scored lower on measures of self-esteem as compared to dismissing avoidants. In addition, based on self- and peer reports, dismissing individuals were described as "cold", "competitive", "autocratic" and "introverted". Conversely, fearful avoidants were described as "submissive", "sub-assertive", "introverted" and "exploitable".

Concerns about the reliability of both Hazan and Shaver's (1987) and Bartholomew's (1990) measures of attachment style due to the small number of items on each, led to the development of further measures of attachment style which use multiple items to make their assessments (e.g., Collins & Read, 1990; Feeney, Noller, & Hanrahan, 1994; Griffin & Bartholomew, 1994; Simpson, 1990). Considerable statistical analysis (e.g., Brennan, Clark & Shaver, 1998) of these multiple-item attachment measures led to the conclusion that they all loaded substantially on the same two major factors. These two factors could be thought of in terms of an affective-behavioural framework, or in terms of Bowlby's (1980)

cognitive/representational model of self and other. In the context of the former, the two derived factors could be conceptualised as ‘anxiety’ - referring to anxieties about abandonment or insufficient love; and ‘avoidance’ - referring to avoidance of intimacy and emotional expression. From Bowlby’s cognitive/representational framework, the two dimensions referred to a conceptual model of the self and a conceptual model of the other, either of which could be positively or negatively configured.

In both the frameworks described above (i.e., affective/behavioural, and cognitive/representational), the two derived factors could be combined to produce four categories that were consistent with Bartholomew’s model of attachment styles. As such, a secure attachment style would be represented by a configuration of low anxiety/ low avoidance or a positive (model of self) / positive (model of others). The dismissing group would be characterized by low anxiety/ high avoidance or positive (self) / negative (other). The preoccupied pattern would refer to high anxiety / low avoidance or negative / positive. Finally, the fearful pattern would be reflected by scores representing high anxiety / high avoidance or negative / negative models of self and other.

The dimensions of anxiety and avoidance, and/or positive versus negative models of self and other, contain within their labels implications for mental health and social functioning. Indeed, a number of cross-sectional studies investigating correlates of adult attachment style have demonstrated that attachment style is associated with a broad array of social and psychological variables, including relationship functioning (Brennan & Shaver, 1995; Collins, Cooper, Albino, & Allard, 2002), personality (Collins & Read, 1990; Mickelson, Kessler, & Shaver, 1997; Shaver & Brennan, 1992), psychopathology such as anxiety and depression (Brennan & Shaver, 1998; Carnelley, Pietromonaco, & Jaffe, 1994), substance use (Brennan &

Shaver, 1995; Senchak & Leonard, 1992), and social support (Simpson, 1990; Simpson, Rholes, & Nelligan, 1992).

With regard to attachment style and relationship functioning, Collins, Cooper, Albino, and Allard (2002) assessed the attachment styles of over 200 adolescents aged between 13 and 19 years. Six years later they re-interviewed these participants along with their romantic partners. They found that insecure attachment in adolescence was a risk factor for adverse relationship outcomes in adulthood, although the effects were most consistent for avoidant attachment. Avoidant adolescents were involved with partners who had less healthy personality profiles. Furthermore, they were involved in relationships that they rated as less satisfying overall, with both them and their partners engaging in less pro-relationship behaviour than their secure counterparts.

Mickelson et al. (1997) examined a wide range of correlates of attachment style in a large community sample ( $N=8098$ ) that was representative of the demographics of the national population. With regard to personality traits in particular, they found that secure attachment was related to higher self-esteem, an internal locus of control, extroversion, and openness to experience. In contrast, both avoidant and anxious attachment styles were associated with an external locus of control, neuroticism, introversion, and lack of openness to experience.

That the above correlates are fairly wide-ranging is consistent with the idea that adult attachment styles reflect an individual's fundamental assumptions about interpersonal interaction, that is, the individual's internal working models. Thus, given the broad implications of attachment style for relationship and personality functioning, one would expect attachment to be significantly related to psychopathology.

## 2.4 *Insecure Attachment and Psychopathology*

Bowlby (1969, 1973) considered the anxiety experienced by the child in response to possible or actual separation from its carer to be a realistic, normal and instinctual response. He proposed that when attachment behaviours were activated (either by separation itself or by the presence of novel or frightening stimuli) and proximity to the attachment figure could not be maintained, the consequent searching and striving for the missing person was accompanied by a sense of disquiet, more or less acute. This sense of disquiet was defined as separation anxiety. A person in the midst of experiencing separation anxiety would be expected to be further sensitized to anxiety activated by unfamiliar or alarming circumstances, and to be further inhibited in their exploratory behaviour. That is, an individual would be likely to respond more fearfully to certain situations if he/she were insecure about the availability or responsiveness of key attachment figures. Again, it is important to emphasise that Bowlby considered these responses to be normal. Furthermore, Ainsworth et al.'s (1978) work using the strange situation confirmed that a healthy and securely attached child would be expected to exhibit anxiety and distress in response to its mother's exit from the room. However the defining characteristic of a securely attached child was its ability to be soothed by the mother upon her return, as opposed to maintaining a detached or persistently distressed presentation. It should be emphasised however that neither avoidant nor ambivalent attachment styles are pathological in themselves but rather represent aspects of the normal variation of possible attachment presentations. Nevertheless, insecure attachment styles are highly associated with indices of maladaptive functioning and psychopathology. In fact, Dozier, Chase, Stoval, and Albus (1999) concluded from their review of adult psychopathology and attachment states of mind as assessed by the AAI, that psychiatric disorder was nearly

always associated with a nonautonomous classification (which is analogous to an insecure attachment style). Similarly, in their large community study of attachment and its correlates, Mickelson et al. (1997) found that almost all forms of psychopathology including mood disorders, anxiety disorders, and substance abuse were negatively correlated with a secure attachment style, and positively correlated with the insecure attachment style. Only alcohol abuse and drug dependence disorders differentiated between the avoidant and anxious-ambivalent attachment styles, both of which were more characteristic of people with avoidant than with anxious attachments.

Other studies have also reported associations between insecure attachment and psychopathology. For example, Hazan and Shaver (1990) found that adults with insecure attachments reported more depression and physical symptoms than those with secure attachments. Similarly, Carnelley et al. (1994) found that mildly depressed women were insecure in their romantic relationships. While Marcaurelle, Belanger, Marchand, Katerelos, and Mainguy (2005) found that attachment insecurity predicted greater symptom severity in patients with panic disorder and agoraphobia. Finally, some studies have found that certain disorders may be linked with specific attachment styles. For example, Pettem, West, Mahoney, and Keller (1993), and Zuroff and Fitzpatrick (1995) found that depression was linked specifically with anxious attachment. While Patrick, Hobson, Castle, and Howard (1994) found that patients with borderline personality disorder were more likely to have an anxious attachment style, whereas those with dysthymia were more likely to have an avoidant attachment style.

Although attachment research has established that attachment style and psychopathology are significantly associated, whether attachment style could be said

to be a causal influence on adult psychopathology is still very much in question. An alternative explanation is that having a psychological disorder distorts an individual's cognitive processes or internal working models of self and others so that they are more likely to report an insecure attachment style. Unfortunately, very few prospective studies of attachment exist to offer support for either explanation. Nevertheless, those few longitudinal studies that have been conducted (e.g., Carlson, 1998; Warren, Huston, Egeland, & Sroufe, 1997) appear to favour the interpretation that attachment style does have a causal influence in the development of psychopathology.

Warren et al. (1997) investigated the specific proposition that anxious-ambivalent attachment as assessed at 12 months of age by the strange situation would predispose infants to anxiety disorders later in life. They made this prediction based on the observation that anxious-ambivalent infants overtly experienced and displayed their anxiety about caregiver availability in a chronic manner, whereas avoidant infants appeared to displace and avoid their anxious feelings. The authors indeed found that a history of ambivalent attachment was specifically and uniquely associated with anxiety disorders at 17.5 years of age. Interestingly, early attachment status was predictive of later anxiety disorder above and beyond predictions based on infant temperament.

In a separate study that nevertheless involved the same sample of families (i.e., those families who had participated in the Minnesota Parent-Child Project), Carlson (1998) examined the relationship between disorganised attachment style and later psychopathology, particularly dissociative symptomatology. She found that the combination of avoidant and disorganized attachment histories (as assessed at 12 and 18 months of age) accounted for a significant proportion of variance in the incidence

and severity of subsequent psychopathology (assessed at 17 ½ years of age). Later assessments (including behaviour problems in primary school, and parent-child relationship quality assessed at 13 years of age) increased the predictability of pathology, ultimately accounting for 31% of the variance. However a disorganised attachment history remained a significant predictor of psychopathology even after controlling for the other variables. With respect to the specific prediction of a relationship between a disorganised attachment style and dissociative symptomatology, infant disorganisation was associated with both higher teacher ratings of dissociative symptoms (in primary and high school), and higher self-reported dissociative symptoms at 19 years of age. No significant associations emerged between disorganised attachment and any of the variables assessing endogenous vulnerability, including temperament.

Given that so few prospective studies of attachment and psychopathology exist, a number of authors have examined the relationship between attachment-relevant childhood events and the development of later psychopathology (e.g., Faravelli, Webb, Ambonetti, Fonnesu, & Sessarago, 1985; Harris, Brown, & Bifulco, 1990a). Those childhood events that are considered to be relevant to attachment include separation from and/or loss of significant attachment figures, and the quality of care received from parents. With regard to the quality of caregiving received, studies have examined both the abstract qualities of parenting such as the perceived warmth, rejection and overprotectiveness offered by parents, as well as more concrete aspects of parenting such as physical and/or sexual abuse and serious neglect. Based on his clinical experience and on reviews of case studies, Bowlby (1980) himself specifically implicated the actual loss of a parent during a child's formative years as a vulnerability factor for the onset of depression in later life. In addition, he

emphasised that particular patterns of interaction between the parent and child, such as being repeatedly told that he was unlovable, incompetent or inadequate, were likely to promote helplessness in the child, and so make him/her more vulnerable to depression in adulthood. Similarly, Bowlby (1973) implicated childhood experiences with threatened loss (as in the case of an attachment figure enduring a serious illness), bereavement or prolonged separation during formative childhood years as predisposing factors for the development of separation anxiety in childhood, and agoraphobia in adulthood. Here too he proposed that pathogenic family interactions were another possible route to these disorders. For example, Bowlby emphasised that the use of repeated and/or intense threats of abandonment or withdrawal of love by a parent to induce good behaviour from the child was a significant and under-rated contributor to anxious attachment in childhood and even into later years. Bowlby proposed that severe anxious attachment was the fundamental pathological process underpinning both school refusal/ separation anxiety in childhood, and agoraphobia in adulthood.

With regard to Bowlby's (1980) views on the pathogenic nature of childhood experiences with loss and bereavement, the evidence for a clear link with adult depression specifically (as Bowlby predicted) has been inconsistent. For example, whereas Harris et al. (1990) found that a child whose mother had died before he/she was 11 years of age was at increased risk for later depression, Tennant (1988) found no evidence for parental death as a significant risk factor of depression in a review of the literature. He did find some evidence however that separations, particularly those occurring in the context of family or parental discord and divorce, contributed to adult depression. Similarly, Mickelson et al. (1997) found that parental divorce but not parental death in childhood was associated with an insecure-ambivalent attachment

style in adulthood. In addition, Lewis, Feiring, and Rosenthal (2000) found that parental divorce during childhood was significantly related to an insecure attachment classification at 18 years, as well as to self-ratings of maladjustment.

As with depression, the attachment literature supports a link between childhood loss and separation anxiety disorder in childhood, and panic disorder and agoraphobia in adulthood (e.g., Brown & Harris, 1993; Faravelli et al., 1985), however the relationship, although reasonably strong, is certainly not specific to these disorders (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1992; Thyer, Himle, & Fischer, 1988). For example, Faravelli et al. (1985) found that persons with agoraphobia had experienced early separation from their mothers or parental divorce significantly more often than a control group with no psychiatric disorder. Similarly, Brown and Harris (1993) found that persons with panic disorder had frequently experienced early loss of a caregiver or extremely inadequate caregiving compared with persons who had no psychiatric diagnosis. However, both Thyer et al. (1988), and Kendler et al. (1992) found that childhood experiences with parental loss were not specific to panic disorder or agoraphobia, with similar incidences of loss occurring in the histories of patients with depression, generalized anxiety disorder and simple phobias.

Taken as a whole, and integrated with other research into the effects of parental psychopathology on children (e.g., Ge, Lorenz, Conger, Elder, & Simons 1994; Rutter & Quinton, 1984), Holmes (1993) has argued that parental disruption and disturbance are a more potent cause of later difficulty and depression than childhood loss itself. That childhood bereavement is certainly a vulnerability factor for later psychopathology is probably linked with disruptions to good parental care - a result that is often the consequence of such loss. A nurturing relationship with the

mother prior to her death, and nurturing, continuous relationships with the father or other caregivers, appears to protect the child from the effects of her loss with respect to vulnerability for later anxiety and/or depression (Brown & Harris, 1993; Harris, Brown, & Bifulco, 1986) For example, Harris et al. (1986) found that inadequate care following parental loss, particularly in cases of separation rather than death, doubled the risk of depression in adulthood. As such, Holmes argued that the development of later pathology is likely to be a cumulative result of vulnerabilities acquired in childhood and adolescence, combined with the circumstances surrounding adult onset of disorder, including contemporaneous perceptions of social support.

With regard to evidence for a relationship between particular patterns of parental caregiving and psychopathology, Dozier et al. (1999) concluded from a review of the literature that inadequate caregiving (and perceptions of past inadequate caregiving) have been linked to almost all forms of psychopathology. The outcomes of dysfunctional parenting appear to be non-specific for children with respect to internalizing and externalizing disorders, however some research has suggested that particular styles of caregiving may predict specific disorders (e.g., Berg-Nielsen, Vikan, & Dahl, 2002; Dozier et al., 1999). For example, affective and anxiety disorders tended to be associated most frequently with parental affectionless control (i.e., a combination of rejection or low warmth, and overprotection); whereas antisocial personality disorder was most likely to be associated with rejection, harsh and inconsistent disciplining, and inadequate control. However little evidence exists to suggest that anxiety disorders can be differentiated by perceptions of parental care. For example, Chambless, Gillis, Tran, and Steketee (1996) found that both patients with panic disorder and agoraphobia, and patients with obsessive-compulsive disorder report parental affectionless control. A difference related to parental care that did

emerge however was in the underlying personality cluster. That is, persons who engaged in more avoidant behaviours reported that their mothers had been neglectful, whereas persons who engaged in dependent or passive-aggressive behaviour reported that their mothers had been overprotective.

Finally, reports of childhood abuse have been demonstrated to predict multiple disorders, including depression and anxiety (Tennant, 2002), borderline personality disorder, dissociative disorder and antisocial personality disorder (Dozier et al., 1999). According to attachment theory, the implications of abuse for personality development and psychopathology are enormous. Dozier et al. (1999) concluded that children with caregivers who do them harm experience unresolvable internal conflict, as the very persons whom they look to for protection from threat are themselves threatening. Whereas the distribution of attachment classifications among children from normal populations is approximately 65% secure, 20% avoidant and 15% resistant (with about 15% given the additional classification of disorganised) (Goldberg, 1995), the proportion of 'insecure' families rises sharply in populations with proven physical or sexual abuse to about 50% avoidant, 30% disorganised and only 10% secure (Belsky & Nezworsky, 1988).

To summarize, both attachment-related events such as parental loss and divorce, and insecure attachment styles appear to create a vulnerability for the later development of psychopathology. Possibly the relationship between attachment-relevant events and psychopathology is mediated by the development of an insecure attachment style. However, the relationship between attachment and later psychopathology is probably a function of deficiencies in ongoing parental care throughout formative childhood years, rather than a result of singular events, however intense. Indeed, Rutter and Quinton (1977) based on a review of the literature

identified that childhood psychopathology could be predicted only by the cumulative risk of multiple social factors. These included marital conflict, a large family size, low socioeconomic status and childhood exposure to maternal psychopathology. None of these factors individually contributed significantly to the child's risk for disorder, but taken together they had a significant impact. Similarly, one would not expect parental divorce in and of itself to be pathogenic, if it were handled with reasonable sensitivity towards the children by both parents, and it was not compounded substantially by other risk factors.

### *2.5 Attachment and Emotion Regulation*

In outlining his conceptual framework of Attachment Theory, Bowlby (1980) commented that the formation, maintenance, disruption and renewal of attachment relationships engendered “many of the most intense emotions” (p. 40). He observed that the finding and maintaining of love evoked feelings of intense joy and security, whereas the threat of loss aroused anxiety, and actual loss invoked sorrow. In addition, each of the last two experiences was likely to arouse anger. Given these connections between emotions and the state of an individual's relationships Bowlby proposed that the psychopathology of emotion (as seen for example in anxiety and affective disorders) was to a large extent a reflection of the psychopathology of “affectional bonds”. As such, it seems worth considering further the ways in which attachment processes contribute to the capacity for emotion regulation in the individual.

The individual's capacity for emotion regulation, including the ability to modulate and cope with anxiety, anger and extreme joy, develops in the context of the family environment. As infants mature into children and children into adults, emotion becomes regulated in increasingly sophisticated ways. Developmental

psychopathologists emphasize multiple, additive and interactive influences on the capacity for adaptive emotion regulation. Particular emphasis is afforded to the roles of inherited vulnerabilities such as anxious temperaments and behavioural inhibition in the face of novelty in young children as well as the mother-child attachment quality and later family processes (for example, parental modelling of problem-solving) (Dadds & Roth, 2001). Given that attachment and family processes can significantly ameliorate the influence of inherited vulnerabilities on maladaptive emotion regulation, it is worth understanding the ways in which such processes operate to facilitate optimal emotion functioning.

Thompson (1994) has offered a definition of emotion regulation that has multiple components. He writes that emotion regulation consists of both external and internal processes that monitor, evaluate and modify emotional reactions, especially with respect to their intensity and timing, in order to accomplish specific goals. The goals referred to might be as straightforward as feeling good/better, or as socially complex as inducing guilt in another person to spur a particular action. The 'external processes' that constitute a significant part of emotion regulation refer to the influences of others in evoking or modulating emotional reactions. Such forces are most evident early in life when caregivers devote considerable efforts to interpreting and managing the emotions of young children (Thompson, 2001). Indeed neuropsychological studies have demonstrated that early parental influences actually play a major role in shaping the infant's developing brain structures which eventually enable the capacity for self-regulation and the adaptive management of stress, among other functions (Schore, 2001).

From birth onwards, an infant uses its expanding sensory and cognitive capacities to interact with its social environment, most notably its mother. During

these interactions, a secure and emotionally attuned mother is continuously and intuitively regulating her baby's variable arousal levels, and therefore emotional states. This process begins to be more easily observable when the infant is about 8 weeks of age, at which time a maturational advance occurs in the primary visual cortex of the infant (Yamada et al., 2000). This neurological activity coincides with the infant demonstrating an intense interest in the emotionally expressive face of the mother (Feldman, Greenbaum, & Yirmiya, 1999). The infant tracks the mother's face in space and gazes intently at her. The infant's gaze in turn evokes the mother's gaze and periods of intense mutual gazing occur. These interactions are highly arousing for both mother and infant, and this high positive arousal is regulated by the synchronization of the intensity and timing of their behaviour, within lags of split seconds (Feldman et al., 1999). By varying the intensity of her verbal/auditory (e.g., the tone, pitch, and volume of her voice, or the tempo of her speech) and nonverbal / visual behaviour (e.g., the brightness of her gaze and facial expression, the tenseness of her posture and speed of body movement) the mother can increase or decrease her baby's excitement / arousal as he synchronizes his affective behaviour to hers and vice versa (Stern, 1985). These episodes of affect synchrony are the first expressions of social play, and are generally patterned by an infant-leads-mother-follows sequence (Feldman et al., 1999), although the mother may intervene first to modulate nonoptimal high and/or low levels of stimulation, thus preventing hyper- or hypo-arousal in her infant (Stern, 1985). Finally, not only the tempo of their interaction, but also the pattern of their disengagement and reengagement is synchronized by mother and baby (Lester, Hoffman, & Brazelton, 1985). Moments of disengagement provide important opportunities for both partners to regulate their arousal levels on their own, but in the presence of the other. This process facilitates the development of

the infant's capacity for emotional self-regulation which develops significantly at about 18 months of age (Sander, 1988). The ability to adaptively move between interactive regulation and autoregulation of negative affective states, depending on the social context, remains important throughout the life span, and emerges out of a history of secure attachment interactions with an attuned social environment (Sander, 1988).

The degree of synchrony in the mother-infant interaction is determined by the mother's level of sensitivity in a number of key moments especially: a) the extent to which she is able to attune her activity level to the infant during periods of social engagement; b) the extent she can allow him to recover quietly in periods of disengagement; and c) the sensitivity with which she can attend to the infant's initiating cues for reengagement (Lester et al., 1985). But even the most sensitive mother is not always attuned to her infant. In fact, frequent moments of misattunement are the norm and represent ruptures of the attachment bond that induce a stress response in the infant (Schorre, 1994). However, in these instances, a sufficiently sensitive caregiver can usually re-establish her external regulation of the infant's negative emotional state relatively quickly, thereby enabling the mother-infant dyad to negotiate a successful recovery. Indeed Schorre (2001) postulated that this process of positively reconnecting with the mother following a negative experience might teach a child that negative emotional states can be endured and even overcome, and as such may well contribute to stress resilience in infants.

The above description of the manner in which mothers regulate their infant's emotional experience through the process of affective synchrony have emerged from the integration of observations of mothers and infants by developmental psychologists

such as Stern (1985) with findings from neurological studies (e.g., Yamada et al., 2000).

Based on the integrated evidence of these two disciplines, Schore (1994) proposed that environmental experience (most significantly in the form of infant-mother interaction) was crucial to the differentiation of the infant's actual brain tissue. Further, he noted that the quality and type of environmental experience affected the structure and function of the brain in a manner that had the potential to either enable or constrain further development. Schore (2001) cited evidence from neurological studies which indicated that from late pregnancy through to the second year (around 18 to 24 months), the brain was in a crucial period of accelerated growth. During this period, cortical and subcortical networks were generated by an initial overabundant production of synaptic connections, which was followed by an environmentally driven process of competitive interaction to select those connections that were most effectively entrained to environmental information. As a result of synchronized emotional transactions, the organization of the infant's right brain hemisphere in particular showed increased coherence, hierarchical organization and connectivity between higher and lower cortical and subcortical structures. This increased coherence enabled the right-hemisphere to act as a self-regulating whole, thereby allowing for increasing complexity with ongoing maturation, and in response to new environmental experience (Schore 1994, 2001).

Again based on interdisciplinary research, Schore (2001) indicated how early attachment transactions shaped the infant's capacity to respond to stressful stimuli. In line with its capacity for emotion regulation, the right brain hemisphere is known to exert major control over both the sympathetic and parasympathetic nervous systems that mediate stress responses (Spence et al., 1996). The sympathetic nervous system

(NS) is responsible for excitatory functions (e.g., the fight-flight response), and the parasympathetic NS for inhibitory responses (e.g., switching off the stress response). These systems are also activated during attachment transactions. When mothers and infants engage in periods of mutual gazing, elevations in sympathetic arousal have been demonstrated in both partners, followed by increases in parasympathetic activity when gaze is disengaged. This process is very similar to that required for adaptive emotional functioning in response to stress, in that novel or stressful stimuli generate a rapid neurochemical response which is then terminated at the appropriate time, or gives way to counter-regulatory measures to prevent an excessive reaction. There is some indication that individuals prone to anxiety and panic disorders have difficulty turning off their stress response (e.g., Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996).

Together the sympathetic and parasympathetic NSs constitute the autonomic NS, which is responsible for the somatic sensations of all emotional states, (Spence et al., 1996), and for transmitting information about these somatic components of emotion to the right hemisphere. Schore (2001) noted that, along with receiving this information about the bodily state, the right hemisphere also received and processed all external socioemotional information. These two sources of information ultimately enabled the individual to meaningfully integrate the internal somatic information of emotion with the external context that evoked it. Finally Schore indicated that other known right hemisphere brain functions included the ability to empathize with the emotional states of others, the storage of representations of oneself in terms of autobiographical memory of one's history, and the storage of internal working models of the attachment relationship. With respect to the latter function, the right hemisphere is thought to implicitly encode strategies for affect regulation that

maintain basic regulation and positive affect even in the face of environmental challenge that occurs in the absence of the original primary caregiver and in later life. Schore (2001) proposed that this process allowed for the development of an internal sense of security and resilience that emerged from the intuitive knowledge that one was able to regulate the changes in one's bodily-based emotional states, either in one's own or within a relationship with caring others.

A good demonstration of the relationship between early attachment history and later emotional functioning with respect to empathy and emotion regulation was provided by Weinfield, Ogawa, and Sroufe (1997). Weinfield et al. asked teachers who were blind to the children's attachment histories to provide written descriptions of the children. In each case children who were described as empathic had secure attachment histories, whereas those described as "mean" were always avoidant. From Weinfield et al.'s observations of the children's free play, they noted that, when a child was distressed, children with secure histories were seen to be more empathic than children with avoidant histories. Interestingly, children with ambivalent/resistant histories did not differ significantly from either of the other attachment groups, but showed trouble maintaining a boundary between their own and other children's distress. That is, they appeared to become distressed in response to witnessing distress in another. Overall, Weinfield, Sroufe, Egeland, and Carlson (1999) concluded that attachment research investigating differences in the regulation and expression of anxiety, anger and empathy amongst children has demonstrated that children with resistant histories are the group most likely to have problems with anxiety, perhaps in response to the constant vigilance they have developed in their early attachment relationships. In contrast, children with avoidant or disorganized histories are most likely to show hostile, aggressive behaviour both with parents and

peers, perhaps as a response to chronic rejection and insensitivity from caregivers.

Finally, children with secure histories seem to have acquired a foundation for empathy and for dyadic affect regulation in which the one who is not distressed helps to regulate the other.

In summary, neuropsychological studies have demonstrated that, from the time the infant is as young as eight weeks, mother-infant attachment transactions are fundamental to the infant's developing capacity for emotion regulation, and these transactions actually influence the structure and function of the brain, particularly the right hemisphere. In addition, many of the functions known to be controlled by the right hemisphere of the brain are relevant to attachment theory in terms of the measured outcomes for secure attachment, such as the demonstration of empathy in childhood, a coherent autobiographical account, positive internal working models of oneself and others, and resilience and adaptability in the face of stress. Finally, Schore (2001) noted that throughout the life span the right hemisphere evidenced continuing periodic growth spurts, probably in the context of right brain synchronization with others, and in response to the developmental stresses that were intrinsic to later stages of life. These growth spurts indicate that, just as Bowlby proposed, internal working models that encode strategies for emotion regulation, can be somewhat modified later in life in response to relevant interpersonal and developmental challenges. As such, one would imagine that attachment processes, operating beyond mother-infant attunement and into childhood and adolescence, offer powerful opportunities for the modification or reinforcement of particular strategies for emotion regulation.

Recall Thompson's (1994) definition of emotion regulation presented earlier which emphasized that emotion regulation was a goal-directed process and one that

was influenced by external figures. On the whole, Thompson argued that the emotional management efforts of caregivers were effortlessly integrated into the child's expanding repertoire of self-regulatory strategies, as both the child and caregiver were forwarding the same emotional goals for the child. Together the child and caregiver aimed to reduce distress, improve feelings of well-being and constructively channel a child's feelings of anger, fear and other emotions to accomplish positive outcomes (Thompson, 2001). Such a situation represented a synchrony between child and caregiver in the direction of optimal growth for the child.

In less than optimal circumstances however, the goals of child and carer for emotion regulation may chronically conflict, or the child might have multiple goals that are mutually inconsistent. For example, when an abusive or rejecting parent ridicules a child's genuine fear or insecurity, or when confronting a depressed and critical parent enables the maintenance of feelings of self-esteem, but exacerbates feelings of guilt and responsibility for the adult's emotional condition (Thompson & Calkins, 1996). In other circumstances, there might be a synchrony between the caregiver's management of emotion with the child's emotional goals, but in maladaptive directions. This can occur, for example, when an adult cooperates with a child's efforts to avoid fear-provoking events - a not uncommon situation when the parent suffers from an anxiety disorder (e.g., Vasey & Dadds, 2001). The result of these difficult situations for the child is often the entrenchment and reinforcement of emotion regulatory strategies that are adaptive within the contexts from which they emerged, but maladaptive in other situations in which they continue to be used. Often this translates to the propagation of strategies that have immediate benefits in

managing the situation at hand, but long-term costs in terms of emotional and interpersonal functioning.

In this way, insecure attachment styles in young children can be viewed as reflecting adaptive strategies for coping with the fundamental and immediate difficulty of maintaining proximity to a rejecting or unpredictable caregiver. In avoidant attachment, the benefits of maintaining attachment, albeit distantly, with a chronically rejecting caregiver, occur at the expense of having to chronically suppress feelings of neediness, anger and anxiety over the abandonment or unavailability of the caregiver (Ainsworth et al., 1978; Bowlby, 1973; Main & Weston, 1982). In ambivalent attachment the benefits of maintaining close attachment to an unpredictable caregiver through the use of excessively clingy and/or submissive behaviour or role-reversal, occurs alongside the reinforcement of hyperarousable and hypervigilant coping strategies, and at the cost of autonomous development. Individuals are likely to suppress feelings of resentment towards their caregiver and self-loathing over their own sense of utter dependency, focusing instead on anxiety and anger over rejection and abandonment.

Anger in particular, is thought to pose a problem to the insecurely-attached child and has been described as characteristic of both avoidantly attached individuals who, whilst seen to be generally emotionally detached, also reportedly experienced irregular bursts of aggressive behaviour (Ainsworth et al., 1978; Main & Weston, 1982 – in Holmes) *and* anxiously attached individuals, who have been found to be highly expressive of anger (Ainsworth et al., 1978; Shaver & Clark, 1994). Anger is thought to be especially incompatible with the need for retaining a close attachment, because of fears by the child that expression of anger will lead to actual abandonment (Bowlby, 1973; Main & Weston, 1982, Stott 1950). This fear may lead to an

escalation of anxiety or to the general (not entirely successful) suppression of anger. Essentially, insecure attachments are reflective of situations in which an infant-caregiver dyad has been unable to competently regulate overwhelming and difficult emotions for the child, especially anger and anxiety. As a result, the child is faced with the dilemma of either suppressing and denying these emotions completely (albeit not necessarily successfully) for fear of being overwhelmed, or existing in a state of almost chronic and heightened anger and anxiety to the exclusion of other emotional experiences. Both options are emotionally restrictive and pose problems for intimate relationships in later life.

Consistent with the above ideas and findings, a number of recent studies provide evidence of attachment style differences in the nature of emotional experience and psychological adjustment in both childhood (e.g., Belsky, Spritz, & Crnic, 1996; Laible & Thompson, 1998) and adulthood (see Shaver & Clark (1994) and Shaver & Hazan, (1993) for reviews). For example, Belsky et al. (1996) found that three-year-old children with secure attachment histories remembered positive events more accurately than negative events. The converse was true for children with insecure attachment histories, although no differences were found on attentional measures. A further investigation of the relationship between attachment and emotional processing found that securely attached children scored higher on two assessments of emotional understanding than insecurely attached children (Laible & Thompson, 1998). This result was primarily due to secure children's greater competence in understanding negative emotion. Laible and Thompson reasoned that the security of attachment fostered greater understanding (as opposed to avoidance) of negative emotions and their consequences.

In adulthood, the relationship between insecure attachment and emotional adjustment manifests itself in terms of evidence that individuals with an insecure, rather than secure, attachment are more likely to report greater loneliness, shame proneness, anger, resentment, anxiety, depression, paranoia, fear of evaluation, self-consciousness, pathological narcissism, and somatic symptoms, as well as lower self-esteem, less self-confidence and maladaptive strategies for coping with negative affect (e.g., Brennan & Shaver, 1995; Feeney & Noller, 1990; Hazan & Shaver, 1987).

Kobak and Sceery (1988) offer a concise summary of attachment theory's account of individual differences in emotion regulation.

Secure attachment [is] organized by rules that allow acknowledgment of distress and turning to others for support, avoidant attachment by rules that restrict acknowledgment of distress and the associated attachment attempts to seek comfort and support, and [anxious-] ambivalent attachment by rules that direct attention toward distress and attachment figures in a hypervigilant manner that inhibits the development of autonomy and self-confidence. (p.142)

Finally, the central problem of attachment and emotion regulation is that early attachment transactions encode emotion regulation strategies that enable a child to feel more or less confident in their ability to manage particular emotional experiences. Insecure attachment patterns ultimately embody regulatory strategies that, despite being functional within the context of the early attachment relationship from which they emerged, if repeated in all relationships in later life, are maladaptive. Unfortunately, the likelihood of repetition of these relating styles is high. For one thing, the process of emotional suppression occurs just beneath conscious awareness

and is not easily accessible to rational, conscious thought. Fundamentally this means that internal working models, which also exist beneath conscious processes, cannot be easily updated in the light of the new experience. The resulting lack of opportunity for emotional processing of painful affective experience may lead to the persistence of primitive feelings of hate and abandonment, restricting emotional growth and development.

### *2.6 Continuity of Attachment Status from the Cradle to the Grave*

A basic assumption of attachment theory is that attachment styles established early in life are relatively stable throughout the lifespan (Eagle, 1995). Some theorists have commented that findings of ongoing continuities in attachment style are most probably the result of ongoing stability in the quality of parenting received, rather than a reflection of the determinative role of early experiences (e.g., Holmes, 1993, Eagle, 1995). In contrast, Weinfield et al. (1999) argue that, although stability of the surrounding environment is a partial explanation for the stability of attachment styles, the environment itself is influenced by the individual's history.

To elaborate, Bowlby's (1980, 1988) concept of "internal working models" of self and relationships are central to the assumption of attachment style stability. Bowlby proposed that early experiences of sensitive or insensitive care contribute to the growth of broader representations concerning a caregiver's accessibility and responsiveness, as well as to beliefs about one's worth with respect to the deservingness of such care. Such expectations enable assessments of the likelihood of sensitive caregiver responsiveness in the immediate context, but also influence future relational expectations and choices, self-appraisal, and behaviour toward others. For example, Bowlby believed that individuals with secure working models of relationship, seek and expect to find supportive and satisfying encounters with their

relational partners. Moreover, the decision rules for relating to others that are implicit in secure individuals' representational models of relationships cause them to behave in such positive and open ways, as to ultimately elicit the support they expect to receive. By contrast, individuals with insecure working models will select, elicit, and interpret reactions from their environment that are likely to be consonant with their own experience-based history. This experience bias in turn limits the opportunities that individuals have for modifying their representational models.

The findings of a number of studies provide indirect support for the existence of internal working models and their role in shaping beliefs, behaviours, and ultimately, relationships. For example, infants assessed as anxious avoidant at 12 months, were more likely as children to isolate themselves (Sroufe, 1983), to interpret ambiguous or even supportive efforts of others as hostile (Suess et al., 1992), and to be rejected by both peers (Jacobson & Willie, 1986) and teachers (Sroufe & Fleeson, 1988). Indeed Sroufe (1990) noted that the behaviour of teachers who were blind to children's attachment histories was reminiscent of what the children had experienced with their caregivers, and therefore likely to confirm the children's working models of self. It is because children have a role in creating their own later experiences that the influence of the environment and the individual's history cannot be viewed independently of one another.

The caregiver's influence on developing representational models is not limited to his/her responsiveness to the needs of the infant. The young child's representations of personal experiences, self and relationships are also shaped in the context of shared discourse with others (Fivush, 1994; Nelson, 1993; Oppenheim & Waters, 1985). Through their own interpretations of significant events in the child's life, caregivers can influence how children perceive the experiences they encounter in terms of their

personal meanings, and their early constructions of self, emotion and morality. Thus attachment figures doubly influence working models – both through the quality of care they provide and through the interpretation of events they offer within shared conversations with the child (Thompson, 1999). Through these processes, secure or insecure representations of relational experience are engendered and maintained in young children.

A third pathway by which parents affect infant representations of relational experience is through the parent's influence on affect regulation - a process that is intrinsically linked with the infant's developing ability to organize, regulate and make meaning of its own emotions. Given that the mother and infant's interaction function to dyadically regulate the infant's emotional states, and that this process actually influences the neural connections and structure of the brain, one would expect these early interactions to exert pressure towards the maintenance of a stable effect of early attachment processes.

With respect to findings of stability of attachment, two research groups assessed attachment security in 6-year-olds using a 1-hour separation and reunion procedure. Both Grossman and Grossman (1991) and Main and Cassidy (1988) found that over 80% of children could be reliably classified as securely or insecurely attached at 6 years of age based on their classification by the Strange Situation at 1 year. Furthermore, the Grossmans found that avoidance ratings on a 7-point scale in infancy were highly significantly related to avoidance ratings at 6 years.

Some studies examining the stability of attachment over even longer periods, have noted remarkable consistencies of about 75% between infant attachment classification at 1 year and secure vs. insecure delegations of attachment representations in early adulthood (from 17 – 22 years of age) using the Adult

Attachment Interview (Hamilton, 2000; Waters, Merrick, Treboux, Crowell, & Albersheim, 2000). Changes in security status were found to be significantly related to experiences with negative life events.

Indeed Waters et al. (2000) found that amongst participants originally classified secure as infants, 85% who had experienced no adverse life events (as defined by the study) were still secure at the 20 year follow-up. In contrast, only 33% of participants with a secure-history who had experienced one or more negative life events were still judged to be secure 20 years later.

In contrast to Waters et al. (2000), Weinfield, Sroufe and Egeland (2000) found no evidence for continuity between infant and adult attachment amongst a population at high-risk for poor developmental outcomes. In the context of findings of attachment continuity amongst middle-class samples, Weinfield and colleagues interpreted their findings as consistent with attachment theory's assertion that early attachment status is open to modification by later, significant, attachment experiences. They observed that their high-risk population had endured more turmoil and general familial instability (e.g., higher rates of divorce and single-parent families) than the average population, as was characteristic of lower socio-economic groups. As such, they found that the discontinuity of attachment that they observed amongst their participants was lawful. Continuous and discontinuous groups were successfully differentiated on the basis of child maltreatment, maternal depression, and family functioning in early adolescence. By contrast, Lewis, Feiring and Rosenthal (2000) found no significant evidence for continuity or for lawful discontinuity in their longitudinal study. For comparative purposes, it should be noted that the methodology and analysis employed by Lewis and colleagues were slightly different to those employed by the studies above. They did find however that an insecure

classification at 18 years of age was significantly related to earlier parental divorce. They also found that indices of adolescent maladjustment were significantly related to attachment security assessed at 18 years, but not to attachment in infancy.

Indeed Thompson (1999) concluded, based on a review of the attachment literature, that attachment security has a much stronger contemporaneous than predictive relationship with measures of psychological adjustment, social competence and emerging representations of self and others. Although some well-thought out longitudinal projects such as the Minnesota Parent-Child Project have reported impressive findings for the predictive validity of early attachment security, Thompson noted that attachment studies on the whole which have investigated the predictive validity of infant attachment security for a diverse range of long-term sociopsychological outcomes have painted a very mixed picture.

All in all, the evidence suggests that the continuity of early relational influences is complex and multifaceted, and that expectations that early attachment security would foreshadow long-term and diverse benefits for children may have been overly simplistic. Instead, Thompson (1999) proposed that an individual's internal working models are based on a network of developing representations that emerge successively and interactively with age. In order to understand how attachment security in infancy foreshadows later functioning, we need to understand the processes by which the infant's simple social expectations about the mother's availability, which embody Bowlby's attachment theory, evolve into the more complex representational systems of later ages.

It is likely that emerging characteristics of the parent-child relationship play an important role in mediating the transition from the infant's simple expectations of the mother, to later complex representations of relationships. As a consequence, early

maternal responsiveness to the dependency needs of the infant is likely to be less relevant to current adjustment correlates than more contemporary indices of parent-child dynamics.

Indeed, Thompson (1999) noted that changes to parent-child relationships can occur as the result of normative developmental processes, whereby the emerging capabilities of the child impose new demands on parents. He emphasized that parents who found it easy to respond sensitively to the dependency needs of the infant, may have more trouble when faced with the task of sensitively balancing the toddler's strivings for independence with his/her ongoing need for security and closeness. Furthermore family events, such as the birth of new babies are likely to challenge parent-child relationship dynamics and as such the child's felt security and capacity to cope with current developmental challenges.

A number of research findings support Thompson's view. For example, Teti, Sakin, Kucera, Corns and Das Eisen (1996) found that firstborn preschoolers experienced a reduction in attachment security following the birth of a sibling. Conversely, attachment security in infants has been demonstrated to increase following infant-mother psychotherapy. Such changes in felt-security are generally lawful, with the most potent factors identified empirically thus far being changes in caregiver life stress, parental, social support and depression (Erickson, Sroufe & Egeland, 1985; Lewis et al., 2000; Pianta et al., 1990).

Taken together, the attachment literature on the continuity of early attachment suggests that attachment security can be stable from infancy through to early adulthood and that changes in attachment security are meaningfully related, and vulnerable, to changes in the family environment.

Bowlby (1973) used the metaphor of branching railway lines to illustrate his elaborated “developmental pathways” model (adapted from Waddington’s (1957) theory of epigenesis) for psychological health. Infants with emotionally available caregivers begin their development on qualitatively different paths than those with emotionally unavailable caregivers. Future experiences with caregivers, and experiences of loss and abuse have differential effects on these children because they are on different branching pathways (Sroufe, 1997). No single experience fully directs or constrains development, however particular paths become more or less likely in response to the consolidation within the child of an organized system for coping with her experiences (Sroufe, 1996). In line with this model, it should be noted that insecure attachment styles are not pathological in themselves. Rather they may render the individual more vulnerable in terms of psychological health if maladaptive insecure attachment strategies are later evoked under pressure or in the face of stress (Eagle, 1995; Grossman & Grossman, 1991).

Finally, Pearson, Cohn, Cowan and Cowan (1994) studied differences in reported depression between two groups of women both classified autonomous according to the AAI. One group was classified “earned autonomous” to reflect that they had developed autonomous states of mind despite reporting difficult life circumstances. The other was termed “continuous autonomous”, and described basically loving parent-child relationships throughout their lives. Interestingly, women in the earned autonomous group reported significantly more depression than women in the continuous autonomous group. This finding strongly suggests that early adverse life experiences have significant implications for later functioning. It also illustrates that the relationships between early attachment, later attachment,

adverse life events and later sociopsychological functioning are incredibly complex, but at the same time, lawful and in need of further elaboration.

## 2.7 *Summary*

In essence, Bowlby's (1969, 1973, 1980) attachment theory refers to the bond between a child and its caregiver, and the extent to which their relationship confers a sense of security and confidence to the child in his/her interactions with the environment, especially in uncertain, threatening or dangerous circumstances. Ainsworth and her colleagues (e.g., Ainsworth et al., 1978) developed a protocol based on a series of brief separations and reunions that enabled the characterization of three individual styles of attachment among 12 month old infants. These were termed secure, insecure-avoidant, and insecure-ambivalent. The three attachment styles could be most clearly differentiated on the basis of the child's ability to seek and accept comfort from its mother during periods of distress. Ainsworth found that the infant's attachment style was most strongly correlated with the mother's behavioural sensitivity and responsiveness to her child's distress. In addition, temperament theorists have found that an infant's temperament, especially with respect to its proneness to distress, may have an impact on the infant-mother relationship, thereby predicting an insecure attachment for the child, especially in cases where the mother is already under-resourced.

Bowlby predicted that the security of an infant's attachment emerged from predictable and repeated interaction with the caregiver in which the caregiver attended to the infant's physical and emotional needs. He proposed that these repeated interactions would be internalised by the child to form a blueprint on which notions of the self, significant others and their inter-relationship were represented. These representations were termed 'internal working models' and would form the prototype

for future interactions with the world, including the ways by which the child would tend to regulate his/her distressing emotions, and the manner in which he/she would engage in future relationships with significant others. Thus attachment theory predicts that the quality of an individual's internal working models will protect or predispose him/her to the later development of psychopathology as a function of their role in determining the individual's ability to successfully regulate his/her distressing emotions either alone or in the context of a significant other.

The work of Main (e.g., Main et al., 1985), Hazan and Shaver (e.g., 1987), and Bartholomew (e.g., 1990) has enabled attachment research to extend beyond the study of infants, to adolescents and adults. Their work has confirmed that an individual's attachment style has ramifications for the quality of his/her relationships, adaptive emotional functioning, and mental health. In addition, attachment and developmental research has indicated that the quality and style of caregiving received by the child, and his/her experiences with parental loss, particularly in the context of parental divorce, also have implications for his/her future mental health. It is less clear however whether specific experiences with childhood adversity lead to specific mental health outcomes, or whether childhood adversity has a more general predisposing effect for later emotional difficulties.

### Chapter 3: Panic Disorder and the Attachment Paradigm

Research into panic disorder has revealed themes in the histories of patients, and in their present-day interpersonal and emotional functioning that parallel the findings for individuals with insecure attachments. These themes include: an overrepresentation of childhood behavioural inhibition (a temperament-based construct), both in the histories of panic disorder patients and amongst their children; a history of childhood separation anxiety; perceptions of parents as rejecting and overprotective; indications of relationship problems; and difficulties with being assertive and expressing anger in relationships. For the most part, the presence of the above attachment-relevant themes in the lives of panic disorder patients have been ignored in the pursuit of cognitive-behavioural and pharmacological treatments for panic symptoms. Yet, research (e.g., Marcaurelle et al., 2005; Mennin & Heimberg, 2000; Noyes et al., 1990) into the personality and interpersonal functioning of panic disorder patients has indicated that these variables may predict a more chronic and severe course of panic disorder, in spite of treatment. As such, clarifying the roles of the above-mentioned attachment-relevant variables amongst panic disorder patients is considered to be crucial for better understanding the psychological mechanisms which may cause and/or maintain the disorder in at least a proportion of patients. Such knowledge would inform improvements in current assessment and treatment practices for panic disorder.

#### *3.1 Developmental Antecedents of Panic Disorder*

Examination of the case and family histories of individuals with panic disorder has revealed the presence of common developmental features. More specifically, there appears to be an overrepresentation of childhood behavioural inhibition amongst panic disorder patients and their children. Behavioural inhibition is a behavioural

construct that is considered to be an index of an anxious temperament. In addition, a significant proportion of panic disorder patients have reported histories of childhood separation anxiety, and/or experienced their parents as rejecting or overprotective in childhood. Whether these developmental experiences are specific to a developmental pathway for panic disorder, or whether they predispose individuals more generally to the development of mental illness has been a matter for some debate. In either case, it is important to establish their influence in panic disorder pathogenesis, to ensure that these issues are adequately addressed both in future research and in clinical assessment and intervention.

### *3.1.1 Behavioural Inhibition (temperament)*

“Behavioural inhibition to the unfamiliar” refers to a temperamental tendency to show fear, withdrawal or quiet restraint in the face of unfamiliar people or situations (Kagan, Reznick, & Snidman, 1988). It is a laboratory-based construct, in that behavioural inhibition (BI) was originally (and is generally) assessed by observing children’s responses to a series of structured and moderately unfamiliar situations, such as new people, objects or non-invasive test procedures. The procedures used to assess behavioural inhibition have varied over time, and across assessment of different developmental ages and stages, and have only been standardised over the last 10 years (see Kagan, 1994 for standardised procedures).

Behavioural inhibition has been demonstrated to be a moderately stable trait (e.g., Garcia-Coll et al., 1984; Hirshfeld, Rosenbaum, & Biederman, 1992; Kagan et al., 1984; Kagan et al., 1987) with a significant but not exclusive genetic component (DiLalla, Kagan & Reznick, 1994; Robinson, Kagan, Reznick & Corley, 1992).

Children assessed as behaviourally inhibited were noted to be easily aroused to distress and motor activity as infants (Fox, Henderson, Rubin, Calkins, & Schmidt,

2001; Kagan, Snidman, & Arcus, 1998), shy and fearful as toddlers, shy with strangers and timid in unfamiliar situations as preschoolers, and cautious, quiet and introverted at school age (Kagan, 1994). Furthermore, Kagan and colleagues (e.g., Garcia Coll, et al., 1984; Kagan et al., 1984; Kagan et al., 1987) noted neurophysiological similarities amongst children with behavioural inhibition. Specifically, these children demonstrated elevated cortisol rates and heart rates that were higher and less variable than behaviourally-uninhibited children in response to cognitively challenges and unfamiliar events. Based on this evidence, Kagan et al. (1987) proposed that behaviourally inhibited children have a generally lower threshold of reactivity in limbic structures that mediate fear and distress, particularly in the amygdala and hypothalamus. As such, these children's inhibited behavioural response (that is, reluctance to approach a novel stimulus) might be viewed as an adaptive effort to cope emotionally with the heightened physiological arousal they experience in these circumstances.

In the general population, behavioural inhibition has an estimated prevalence of 10-20% amongst four and six year old children. Another 10% of children give indications of being behaviourally uninhibited (Garcia Coll et al., 1984; Kagan et al., 1984; Kagan et al., 1987). The children of panic disorder patients, however, are at significantly greater risk for behavioural inhibition than the average child (e.g., Battaglia et al., 1997; Rosenbaum et al., 1988; Rosenbaum et al., 2000). For example, Rosenbaum et al. (1998) found BI to be more prevalent among 33 children of patients with panic disorder than among 23 children of patients with other psychiatric conditions. Similarly, Battaglia et al. (1997) found that 19 children of panic disorder patients exhibited more inhibited behaviours than did the 16 children of comparison parents. Interestingly, the work of Rosenbaum et al. (2000) indicated that the children

of parents with major depressive disorder were also at increased risk of behavioural inhibition, with comorbid panic disorder (PD) and major depressive disorder (MDD) conferring the highest risk to children. Amongst a sample of 2-6 year old children, the authors found that 29% of children whose parents had both PD and MDD were behaviourally inhibited, whereas only 12% of children of comparison subjects (with neither PD nor MDD) had BI. Children whose parents had one or other disorder had intermediate rates of BI that were not significantly different from either the comparison group or the comorbid group. Rosenbaum et al. concluded from their results that individually, the 2 disorders (i.e., PD and MDD) were unlikely to confer a differential risk for BI in the children of these patients. These findings actually contradict those of an earlier study by Rosenbaum and colleagues (i.e., Rosenbaum et al., 1988) which found a progression of increasing rates of BI from the psychiatric comparison group without PD or MDD (15.4%), to MDD (50%), to comorbid PD and MDD (70%), to PD (84.6%). The findings of this study however were more limited by a small sample size than the later study (i.e., 56 children versus 284 children). Nevertheless these findings need to be replicated by other studies in order to establish more firmly the relationship between PD and MDD, and the increased risk for BI amongst children of patients with these disorders.

A number of clinical and empirical findings led researchers to propose that children with BI were at risk for the later development of anxiety disorders, particularly panic disorder. Firstly, descriptions of inhibited school-age children were similar to the clinical descriptions of children whose parents had panic disorder, and to retrospective descriptions of childhood by adults with panic disorder or agoraphobia (e.g., Berg, 1976; Klein, 1964; Pollack et al., 1996). For example, Gersten (1989) found that behaviourally inhibited children showed school refusal and

separation anxiety, much like the children of agoraphobic patients, and agoraphobic patients themselves as children. In addition, evidence for increased arousal in the limbic-sympathetic axes of the nervous system of children with BI (e.g., Kagan et al., 1987) fit well with hypotheses about the underpinnings of anxiety disorders, and possibly panic disorder (e.g., Charney et al., 1990).

Nevertheless, despite the above findings suggesting that childhood BI and panic disorder may be linked, cross-sectional and longitudinal studies (e.g., Biederman et al., 1990; Biederman et al., 1993; Biederman et al., 2001) have established that, although children with BI appear to be at increased risk for anxiety disorders, the effect may be more specific for social phobia than for panic disorder. For example, Biederman et al. (2001) found that 5-6 year old children with BI were at increased risk for social anxiety disorder compared with non-inhibited children. This effect was strongest for behaviourally-inhibited children who also had parents with both panic disorder and comorbid depression.

Some major limitations to Biederman et al.'s (2001) study, however, make their conclusions regarding BI and later social anxiety disorder rather speculative. Firstly children were assessed for psychopathology using the mothers' reports on the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Epidemiologic Version (Orvaschel, 1994) and the Child Behavior Checklist (Achenbach, 1991). The diagnostic criteria for these instruments are unlikely to be equivalent to standard diagnostic instruments such as the *DSM-IV*. In fact, given that about four percent of children appeared to be diagnosed with panic disorder, when PD is a condition that emerges only very rarely in pre-pubertal children, one must assume that the diagnostic criteria used are very over-inclusive and thus have questionable validity.

Secondly, when the children were divided into diagnostic groups, the resultant sample sizes used for comparative purposes were quite small, and thus of questionably statistical reliability. For example, only 18 children (made up of both children with and without BI) had both social anxiety disorder and a parent with PD and comorbid MDD.

Finally the authors found a significant difference between children with and without BI only for avoidant disorder, but combined this group with children with social phobia on the grounds that the two conditions were similar. They then labelled this combined group “social anxiety disorder”, and drew conclusions about the combined group as a whole. The validity of this action is also questionable.

Nevertheless, taken together with other research, it seems clear that children with BI are at an increased risk for childhood anxiety disorders, possibly for multiple disorders (e.g., Biederman et al., 1993; Hirshfeld et al., 1992), with possible specificity for social anxiety (e.g., Hayward, Killen, Kraemer, & Taylor, 1998; Schwartz, Snidman, & Kagan, 1999).

For example, Hayward et al. (1998) assessed BI in over 2000 Year 9 students using a retrospective self-report measure. They subsequently interviewed those students identified as high and low on childhood BI, at yearly intervals through high school, for symptoms of depression and social phobia. The authors found that those students showing indications of high BI in childhood were at a greater than 5-fold increased risk for developing social phobia than those low in BI. In addition, Schwartz et al. (1999) found significantly higher rates of social anxiety amongst 13 year olds who had been classified as behaviourally inhibited at 21 or 31 months, as opposed to those teenagers who had been classified as uninhibited. Nonetheless, further studies with better designs and longer follow-up intervals are needed to

confirm the predictive validity of behavioural inhibition for later psychopathology, and to uncover any possible links with the development of panic disorder in early adulthood.

Although behavioural inhibition is generally considered to be an index of temperament, and is moderately heritable, the fact that the earliest assessments of BI are made at about 2 years of age, leaves ample room for the possibility that BI might be just another aspect of an insecure attachment, or that BI could be the product of an interaction between temperamental indices such as proneness to distress, and the attachment quality. As such, Fox and Calkins (1993) assessed children's behavioural and physiological responses to novelty and restraint, maternal ratings of child temperament, and attachment style at multiple intervals from birth to 24 months. Infants could be grouped reliably into two groups according to their physiological reactivity to novelty (e.g., a peek-a-boo game with a stranger). Fox and Calkins suggested that the high reactivity group showed temperamental features that could be considered to be early indications of behavioural inhibition. However, they subsequently found no relationship between the early measures of infant reactivity and BI as measured at 24 months, suggesting that early temperament alone was not sufficient to account for the development of BI. Furthermore, Fox and Calkins found that attachment style interacted with the infant's early reactivity levels to predict behavioural inhibition at 24 months. Specifically, those infants who were highly reactive and had anxious-ambivalent attachments tended to demonstrate higher levels of behavioural inhibition. These findings suggest that BI is not simply an index of the temperamental characteristics of the child, but is as much a function of the early attachment relationship between mother and child.

A different study by Nachmias et al. (1996) sought to examine the relationship between attachment theory and behavioural inhibition in predicting physiological stress responses in 78 eighteen-month-old toddlers. Specifically, Nachmias et al. measured salivary cortisol levels (which reflect the activation of neuroendocrine responses in the HPA system) to assess the children's physiological stress reactions in the face of three novel events (i.e., a vibrant live clown, a robot clown, and some lively puppets). The children were rated as having BI according to their "ease of approach" to the novel stimuli, and the strange situation was conducted a week later to assess attachment style. Interestingly, the authors found that only children who were both insecurely attached AND behaviourally inhibited were unsuccessful at reducing the activation of their neuroendocrine response to stress. No other group, neither securely attached- inhibited children, nor secure/ insecure- noninhibited children showed similar elevations in cortisol levels after exposure to the novel, arousing events. The findings of this study suggest that BI in and of itself is insufficient to predict a child's vulnerability for the development of anxious psychopathology in particular. Instead, the combination of behavioural inhibition and an insecure relationship with their caregiver is likely to be a superior barometer of early risk for later anxiety disorders.

Finally, Warren et al. (2003) conducted a comprehensive study that examined the relationship between temperament, BI, attachment styles, and parenting behaviours amongst infants of mothers with panic disorder. Research (e.g., Gersten, 1989; Rosenbaum et al., 1988) has demonstrated that these infants are already at an increased risk for both behavioural inhibition and childhood anxiety disorders by virtue of their mothers' psychopathology. Warren et al. recruited from the community a total of 52 mothers with PD, and 42 mothers without psychopathology to act as

controls. The mothers' infants were aged either four or fourteen months, with the different age groups undergoing slightly different testing procedures according to age-appropriateness.

Interestingly, Warren et al. found few differences between infants of mothers with PD and infants of control mothers. Infants with PD mothers did not show higher reactivity (rated for 4 month-old infants only as high or low levels of crying and motor activity in response to novel stimuli), they did not show higher rates of behavioural inhibition at 14 months, nor did they demonstrate any differences in attachment security (assessed by the strange situation at about 15 months of age). Of the infant outcome variables, only two significant differences emerged, and these were higher salivary cortisol levels and more disturbed sleep amongst infants of PD mothers than infants of controls.

Thus, the infants of PD mothers showed few behavioural indications of temperament or attachment-style differences compared with infants of control mothers, although they did show neurophysiological differences that could be indicative of higher arousability. In line with their findings, Warren et al. (2003) proposed that behavioural inhibition might emerge later in childhood, possibly in response both to a different neurophysiological profile, and environmental experience, especially with respect to the infant's relationship with the mother. Other studies finding higher rates of BI for infants of PD mothers have generally assessed the children for BI at 24 months of age or later (as opposed to 14 months of age in this study).

Indeed, despite finding few differences amongst the infants in their study, Warren et al. found that mothers with PD responded to their infants in ways that were significantly different from control mothers. Specifically, the PD mothers of four-

month-old infants demonstrated less sensitivity (that is, they ignored or misread their infants' signals) and more intrusiveness (that is, they limited or interrupted the infants' activities) towards their infants than controls. In addition, PD mothers of 14-month-old infants reported engaging in more ineffective disciplinary practices than control mothers. Specifically, they indicated that they were more likely to become angry with their children in response to relatively minor offences (e.g., yelling, arguing, and insulting). The authors suggested that these results might be an indication of emotion regulation difficulties amongst PD mothers, particularly with regard to anxiety and anger. They observed that these mothers seemed to be less attentive to their children and more distracted, which could be a result of preoccupation with their own anxiety. Finally, PD mothers were more likely than controls to engage in sleep practices for their children that were associated with more disturbed sleep and difficulty settling to sleep. For example, these mothers were more likely to feed their infants to sleep, to sleep in the same bed with them, and less likely to put them to bed awake. Finally, the authors noted that all of the parental behaviours described above (i.e., less sensitivity, more anger in disciplinary situations, and more use of potentially problematic sleep strategies) have been associated with an increased risk of psychopathology for children.

Only further longitudinal studies would be able to determine whether or not the children of PD mothers continued on to develop higher rates of behavioural inhibition or mental health problems than the children of controls. However, even if this finding eventuated, the result would not be able to be attributed exclusively to temperament (as behavioural inhibition theorists such as Kagan, Reznick, and Snidman (1988) propose) but must be considered, to some extent, to be also a product of particular family environments.

In summary, the children of panic disorder patients demonstrate higher rates of both behavioural inhibition and childhood psychopathology than the children of parents without psychopathology (e.g., Gersten, 1989; Rosenbaum, 1988). Parental depression, either with or without panic disorder, has also been associated with childhood behavioural inhibition (Rosenbaum et al., 2000). Studies of children who exhibit BI have indicated that they are at increased risk for childhood psychopathology, particularly childhood anxiety disorders. Further, there may be some specificity for childhood psychopathology involving anxiety about social evaluation (e.g., Biederman et al., 2001). Whether children with BI are at greater risk for the later development of panic disorder has yet to be established. Finally, although BI has been put forward as an index of endogenous variables such as temperament and higher infant arousability (e.g., Kagan, Reznick, & Snidman, 1988), the evidence (e.g., Fox & Calkins, 1993) suggests that it is more likely to be the later product of both infant temperament and mother-infant interactional style. In addition, the later development of anxious psychopathology may be best predicted by a combination of behavioural inhibition and attachment style (e.g., Nachmias et al., 1996).

### *3.1.2 Perceived Parental Behaviours*

Bowlby (1973) suggested that maternal overprotectiveness was a key factor in the genesis of separation anxiety and agoraphobia. Some early clinical observations of families of patients with agoraphobia appeared to support this assertion (Buglass, Clarke, Henderson, Kreitman, & Presley, 1977; Solyom, Silberfeld, & Solyom, 1976; Terhune, 1949; Tucker, 1956; Webster, 1953). For example, Solyom and colleagues (1976) found that mothers of agoraphobics rated higher on measures of overprotectiveness and anxiety than a control group. Snaith (1968) however found no

differences in perceptions of parental overprotectiveness amongst a group of patients with agoraphobia, patients with specific phobias, and a control group.

Parker, Tupling, and Brown (1979) developed the Parental Bonding Instrument (PBI) in order to allow for the quantification and empirical assessment of clinical observations of differences in parenting behaviours between clinical and nonclinical groups. The PBI is a retrospective self-report questionnaire that allows adults to rate their early parenting on dimensions of 'care' and 'overprotection'. Studies using the PBI have found that patients with panic disorder/ agoraphobia (e.g., Faravelli et al., 1991; Silove, 1986) typically characterized their parents as uncaring or rejecting. Parental overprotectiveness however was reported among agoraphobic and panic disordered patients with somewhat less consistency, despite Bowlby's early observations (e.g., deRuiter & van Ijzendoorn, 1992; Pacchierotti et al., 2002; Parker, 1979a; Silove, Parker, Hadzi-Pavlovic, Manicavasagar, & Blaszczyński, 1991). For example, Parker (1979a) found that agoraphobic patients rated mothers as uncaring but not overprotective compared to controls. In contrast, no differences emerged for perceptions of paternal parenting behaviours. Similarly, Pacchierotti et al. (2002) found an association between less caring parental reports (for both mother and father) for *DSM-III-R* diagnosed panic patients, with no differences found for the protection dimension. On the other hand, Silove (1986) found that *DSM-III*-diagnosed agoraphobic patients scored both parents as significantly less caring and significantly more overprotective than a control group. A number of other researchers have similarly found that panic disordered patients rated their parents as low on caring and high on overprotection relative to controls (Faravelli et al., 1991; Leon & Leon, 1990; Wiborg & Dahl, 1997). Interestingly Wiborg and Dahl (1997) found this pattern of adverse parenting to hold true only for those panic patients with comorbid major

agoraphobia. Those with minor agoraphobia were not distinguishable from controls on measures of parental perceptions. On the whole, Silove (1986) concluded that overprotective parenting might be pathogenic for the later development of agoraphobia only when combined with a lack of perceived care. Other researchers, such as McNally (1994), have concluded that experiences with adverse parenting are a generic risk factor for subsequent psychopathology, rather than specifically related to panic disorder or agoraphobia per se. This conclusion came in light of findings that uncaring and/or overprotective parental representations were also found to be associated with other anxiety and depressive disorders (e.g., Harris, Brown, & Bifulco, 1990b; Leon & Leon, 1990; MacKinnon, Henderson, & Andrews, 1993; Parker, 1979b; see Rapee, 1997 for a review). Alternatively, Parker (1979b), based on his findings from a nonclinical sample, proposed that low maternal care might act as a general predisposing factor for later psychopathology, whereas maternal overprotection could provide a more specific additional risk factor for anxiety disorders. Specifically, he administered the PBI to a sample of psychology students to examine for possible differential effects of parenting styles on trait anxiety and trait depression. He found that, whereas maternal care was the strongest predictor of both trait anxiety and depression, only maternal overprotection explained additional variance in trait anxiety scores.

Manicavasagar and colleagues proposed that changes to the way agoraphobia has been diagnosed over the years could account for the discrepancies between Bowlby's initial observations of parental overprotection in the families of patients with agoraphobia, and the findings of more recent research (e.g., Manicavasagar, Silove, & Curtis, 1997; Manicavasagar, Silove, Wagner, & Hadzi-Pavlovic, 1999). Based on their research, these authors proposed the existence of a distinct adult

anxiety disorder that was similar to childhood separation anxiety disorder in that a patient's fears centred primarily on issues of separation from significant attachment figures. They labelled this syndrome 'Adult Separation Anxiety Disorder' (ASAD). Manicavasagar and colleagues suggested that ASAD was most consonant with the conception and assessment of agoraphobia prevalent in Bowlby's time, and therefore would be more likely to demonstrate a clear relationship with maternal overprotection than current present-day *DSM-IV* diagnoses of agoraphobia (that is, panic disorder with agoraphobia). Furthermore, they proposed that ASAD and panic disorder would have high rates of comorbidity. In a study designed to test these propositions, Manicavasagar et al. (1999) recruited two groups of participants. The first group consisted of 34 adults, recruited from the community based on their self-reported anxieties regarding separation from significant attachment figures. The second group consisted of 37 patients referred by an anxiety clinic and diagnosed with panic disorder with/without agoraphobia. Consistent with their expectations, Manicavasagar et al. found that about  $\frac{3}{4}$  of the PD patients and  $\frac{1}{2}$  of the ASAD group were comorbid for both anxiety disorders. Furthermore, they administered the PBI to all subjects and discovered that maternal overprotection (and not maternal care or either of the paternal dimensions) emerged as a significant predictor of ASAD status for a combined sample of ASAD and/or PD-Ag sufferers. By contrast, neither parental care nor overprotection (for mothers or father) predicted assignment to the PD group in the combined PD and ASAD sample. Furthermore, maternal overprotection scores for patients with PD but no comorbid ASAD were no different from the scores of separate control group who were recruited consecutively from amongst the patients of a general practice service. These promising findings confirm Manicavasagar et al.'s proposition that historical shifts in the diagnostic criteria for

agoraphobia and panic disorder which combine the two syndromes and make agoraphobia secondary to panic, have confounded recent research attempts to examine the developmental antecedents of panic disorder and agoraphobia.

Specifically, these findings suggest that inconsistencies in recent research with regard to the presence of perceived parental overprotection in PD samples could be the result of variability between studies in the comorbidity rates for an ASD diagnosis.

Nevertheless, the findings of Manicavasagar et al. need to be replicated by different research groups in order to support the need for a new anxiety diagnosis before any firmer conclusions can be drawn.

In summary, the evidence suggests that aberrant parenting is clearly associated with the development of both childhood and adult psychiatric disorders (e.g., Mackinnon, et al., 1993; Parker, 1979a; 1979b; Torgersen, 1990). Less clear is the issue of whether specific parenting styles predispose children to the development of specific psychopathology. Patients with panic disorder and agoraphobia tend to perceive their parents as more rejecting and possibly more overprotective than do normal controls. However similar patterns of parental perceptions have also emerged for patients with depression and other anxiety disorders such as obsessive-compulsive disorder (e.g., Chambless et al., 1996). The lack of clear evidence for a specificity hypothesis between parenting style and psychiatric disorder might reflect the supposition that aberrant parenting acts as a general risk factor for the development of psychopathology. Alternatively, the lack of evidence for specificity might reflect inadequacies in the instruments used to assess parenting style such as the PBI. Specifically, the two scales of the PBI may be unable to pick up specific and subtle nuances in family dynamics that might actually predispose individuals to specific disorders. In addition to possible measurement limitations, investigations into a

specificity hypothesis are further complicated by such issues as high comorbidity rates between depression and anxiety, and between various anxiety disorders. For example, depression occurs comorbidly in as many as 90% of PD patients (e.g., Noyes et al., 1990). Furthermore, PD populations might also be confounded by the presence of comorbid but undiagnosed adult separation anxiety disorder. The latter finding is an interesting possibility which requires further validation and replication. If supported, the possibility of a diagnosis of adult separation anxiety disorder that is often misdiagnosed as panic disorder or is a frequently undiagnosed comorbid condition with panic disorder, as Manicavasagar et al. (1999) propose, is likely to have major implications for research into the etiology and course of panic disorder. For example, childhood separation anxiety disorder has been proposed as a possible developmental antecedent of panic disorder with/without agoraphobia, however the evidence for this connection has been inconsistent. Manicavasagar et al. have proposed that this inconsistency has occurred as a result of PD being confounded by comorbid or misdiagnosed ASAD.

### 3.1.3 *Childhood Separation Anxiety Disorder*

Bowlby asserted that separation anxiety was a normal developmental phenomenon. However, Gittelman and Klein (1985) defined anxieties about separation as clinically significant when they exceeded normal developmental expectations, or when they interfered markedly with an individual's life. The *DSM-IV* (American Psychiatric Association, 1994) characterizes childhood separation anxiety disorder (SAD) as a condition involving excessive fears for close attachment figures (e.g., fears of someone significant coming to harm), or of being separated from people or places of safety. SAD is the only remaining anxiety disorder relegated exclusively to childhood in the *DSM-IV*. Other childhood anxiety diagnoses (e.g. overanxious

disorder) have been subsumed under adult disorders (i.e. generalized anxiety disorder), with diagnostic criteria being revised to include relevant descriptors for children and adolescents. This move in the most recent *DSM* edition has reflected a trend to emphasize the continuity between anxiety disorders in children and adults (Craske, 1997). Nevertheless, equivocal research findings have meant that SAD has defied this trend of linking it with a particular adult diagnostic category.

Bowlby (1973) argued that a severe, underlying anxious attachment was the basis for the development of school refusal (a condition that is now subsumed under the diagnosis of SAD according to the *DSM-IV*) in childhood and agoraphobia in adulthood. Other authors (e.g., Klein, 1981; Shear, 1996; Silove & Manicavasagar, 2001) have since proposed that childhood SAD and adult agoraphobia are likely to be continuous. It is important to note that, as the *DSM-IV* now links agoraphobia inextricably with PD, theorists who continue to support the view that childhood SAD and agoraphobia are continuous, must now refer to this continuity as occurring between SAD and PD (with or without agoraphobia unless otherwise specified).

Klein (1964) proposed that the regularity with which PD patients reported a history of early SAD suggested that the two types of anxiety arose from a common developmental pathway. For example, Klein observed that 50% of his severely agoraphobic patients showed distinct evidence of premorbid separation anxiety in childhood. Furthermore he noted that the patients' initial panic episodes had often been preceded by the loss of a significant attachment figure. These findings led Klein to posit a theory in which the symptoms of separation anxiety disorder and later panic disorder with agoraphobia are the manifestation of a pathologically sensitive neurophysiological alarm system that was designed to promote evolutionary survival by protecting the young from actual or threatened ruptures to primary bonds. The

apparent success of the anti-depressant imipramine in the treatment of his agoraphobic patients, led Klein to propose a trial of imipramine in a childhood population with school phobia. As mentioned, school phobia or refusal has often been used inter-changeably with separation anxiety disorder in the literature, and is characterized as one of the manifestations of separations anxiety disorder in the *DSM-IV*. Indeed, Gittelman-Klein and Klein (1973) found imipramine to be significantly superior to placebo in the treatment of school phobia. This result was seen to provide support for Klein's theory of continuity between childhood separation anxiety and panic disorder. Subsequent research by Klein and his colleagues however (i.e., Klein, Kolplewicz, & Kanner, 1992), failed to replicate previous findings of imipramine efficacy for childhood separation anxiety disorder, casting doubt on the veracity of Klein's proposal.

Nevertheless, several lines of research have supported a link between childhood separation anxiety and agoraphobia. A number of studies have consistently found that about 20 – 50% of PD patients report a history of childhood separation anxiety (Aronson & Logue, 1987; Breier, Charney & Heninger, 1986; Gittelman & Klein, 1985; Klein, 1964; Klein, Zitrin, Woerner, & Ross, 1983). In addition, Faravelli et al. (1991) found that more agoraphobic patients than healthy control subjects reported distressing childhood events involving separation and loss from parental figures, which was consonant with the proposition that separation anxiety evolves as a result of such experiences with loss. In addition, Tweed, Schoenbach, George, and Blazer (1990) reported that adults whose mothers had died before they were ten years old were almost seven times more likely than those without a history of early maternal death to be diagnosed with *DSM-III* agoraphobia with panic attacks. Furthermore, those adults whose parents had separated or divorced before they were

10 were four times more likely to be diagnosed with agoraphobia with panic attacks than those without a history of early parental separation. Indeed, the evidence for a history of separation anxiety amongst PD patients has added weight to the notion, popular with attachment theorists (e.g., Shear, 1996), that PD and/or agoraphobia represent an unresolved “attachment-autonomy conflict” characterized by high levels of SA.

Despite the empirical support for a positive connection between SA and PD/Ag, the evidence in support of the proposition of a *specific* link between the two is less convincing. Whereas Weissman, Leckman, Merikangas, Gammon, and Prusoff (1984) found that children of parents with comorbid depression and panic disorder with agoraphobia were at a greater risk for separation anxiety than children whose parents had depression alone, other studies comparing retrospective reports of separation anxiety between PD patients and other clinical groups have had more equivocal results. That is, there appear to be as many studies finding significant differences between clinical groups (e.g., Gittelman & Klein, 1985) as there are finding none (or results in the opposite direction) (e.g., Thyer, Nesse, Cameron, & Curtis, 1985; Thyer, Nesse, Curtis & Cameron, 1986). For example, while Gittelman and Klein (1985) found that more than twice as many women with panic and agoraphobia, than women with specific phobias reported histories of separation anxiety (i.e., 48% vs. 20%); Thyer and colleagues (1985, 1986) found no differences between these two diagnostic groups. In addition, whereas Aronson and Logue (1987) found that 20% of their PD patients reported a history of separation anxiety, twice as many reported histories of overanxious disorder.

Finally, some studies such as those by Lipsitz et al. (1994) and Aschenbrand, Kendall, Webb, Safford, and Flannery-Schroeder (2003) have suggested that rather

than being a predisposing factor for a specific psychiatric disorder, early separation anxiety might predispose individuals to the general development of more severe anxious psychopathology in later life. For example, Lipsitz et al. examined reports of early separation anxiety in 252 patients attending an anxiety clinic. The patients' diagnoses included panic disorder, simple phobia, social phobia, obsessive-compulsive disorder (OCD), and generalized anxiety disorder (GAD). A history of SAD was found to be greater amongst patients with two or more lifetime adult anxiety disorders than among those with one (37% vs. 14% respectively). There were no differences in rates of SAD however between specific diagnostic groups. Most recently, Aschenbrand et al. (2003) conducted a longitudinal study with children who had completed treatment for an anxiety disorder (SAD, social phobia, or GAD) about 7 years earlier on average. They found that children who had been diagnosed with early SAD were at no greater risk for developing PD (or GAD, depression or social phobia) than the other clinical groups. They were, however, more likely to meet criteria for other anxiety disorders (such as specific phobia, post-traumatic stress disorder, OCD and acute stress disorder. The authors concluded that there was no evidence for a specific link between childhood SAD and adult PD.

Nevertheless, based on a review of the evidence for a link between SAD and PD, Silove, Manicavasagar, Curtis, and Blaszczynski (1996) concluded that, taken as a whole, the literature supports a hypothesis of continuity between the conditions, but the specificity of that relationship required further clarification. They noted that particular methodological problems inherent in the available research may have been contributing to variability in the findings. Specifically, the authors targeted possible inconsistencies in measuring separation anxiety symptoms, high comorbidity rates amongst PD patients for other Axis I and II disorders (see Dick et al., 1994), and

demographic changes to the PD population corresponding with changes in diagnostic criteria. In response to the issue of the absence of a standardized instrument for assessing separation anxiety symptomatology, Silove et al. (1993a) developed the Separation Anxiety Symptom Inventory (SASI).

Using the SASI, Manicavasagar, Silove, and Hadzi-Pavlovic (1998) found that about 50% of PD patients returned high SASI scores related to childhood separation anxiety. In addition, they found that patients with a lifetime history of PD returned higher SASI scores than other patients suffering from anxiety disorders that did not involve panic such as GAD (Silove et al., 1995; Silove, Manicavasagar, O'Connell, & Blaszczynski, 1993b). As such, they proposed that childhood SA exerted a graduated risk, with moderate levels predisposing subjects to GAD and higher levels increasing the risk to panic attacks and/ or PD. The authors also noted however that, despite some empirical support for the hypothesis that SA is linked to risk of later PD, the relationship does not appear to be highly specific or exclusive. That is, early SA may be associated with other anxiety-related outcomes, and a large portion of persons with adult PD do not report heightened levels of early SA. As such, Silove and Manicavasagar (2001) proposed that the developmental pathway leading to the common final outcome of PD was likely to be heterogeneous, with early SA relevant in only a minority of cases.

Furthermore, as mentioned previously, Silove and Manicavasagar (2001) proposed that some cases of childhood SAD might persist into an adult form of the disorder that was diagnostically separate from other anxiety disorders. They suggested that this disorder, which they labelled adult separation anxiety disorder (ASAD), could overlap substantially with PD populations, such that it might often be misdiagnosed as PD, and/or might be inaccurately treated as a secondary condition,

given the lack of a clear diagnostic category for adulthood. Indeed, their research provided some support for this formulation (e.g., Manicavasagar, Silove, Wagner, & Hadzi-Pavlovic, 1999). In a study described earlier that examined parental perceptions and separation anxiety symptoms in a combined sample of ASAD and PD sufferers, Manicavasagar et al. (1999) found that participants assigned to the ASAD category (which included ASAD only, and ASAD plus PD patients) reported significantly higher levels of childhood separation anxiety than those with PD alone. In contrast, the combined PD patients with or without comorbid ASAD reported elevated separation anxiety levels that were identical to the ASAD only group. An additional study by Manicavasagar and colleagues that emphasised the strong comorbid and probably causal nature of the relationship between separation anxiety and PD, found that the majority (89%) of a sample of people recruited from the community and diagnosed provisionally with adult separation anxiety disorder, also had comorbid depression or panic disorder with/without agoraphobia. Nevertheless, 75% of this group stated that their separation anxiety symptoms had preceded the onset of comorbid disorders and were more debilitating than the panic or depressive symptoms they experienced (Manicavasagar, Silove, & Curtis, 1997). Taken together, these findings support the propositions that a) separation anxiety and panic disorder are strongly associated, and b) differing rates across studies of undiagnosed ASAD amongst patient groups might contribute to the variability of findings with regard to childhood SAD.

In summary, the literature supports the existence of a relationship between childhood separation anxiety disorder and panic disorder, despite findings that this relationship may not be highly specific or exclusive. The finding that childhood SAD may be more specifically continuous with an adult form of the disorder is an

interesting one that requires further replication and examination. In addition the possible relationship between adult SAD and PD requires further explication. Based on their findings, Manicavasagar et al. proposed that ASAD and PD might be linked such that, under conditions of interpersonal stress, people suffering from ASAD might be at risk for developing secondary symptoms of panic attacks, which then escalate into panic disorder and possibly become further complicated by symptoms of agoraphobia. Since separation anxiety disorder is not yet widely recognised as a diagnosable condition in adulthood, an alternative view of the relationship between SAD and PD could be set out according to Silove and Manicavasagar's (2001) proposal noted earlier. That is, that PD is a heterogeneous condition arrived at via a variety of possible developmental pathways, one of which is characterized by ongoing separation and relationship issues. As such, certain children who have experienced early insecure attachment relationships might be predisposed to experience panic attacks under conditions of interpersonal stress in later life. These individuals might then be at risk for developing panic disorder with or without agoraphobia. The evidence that at least some adults with panic disorder demonstrate interpersonal issues and personality traits consonant with a history of insecure early relationships provides support for this view.

### *3.2 Adult Personality and Interpersonal Functioning*

Based primarily upon clinical observation, people with panic and agoraphobia have been frequently described as unassertive, anxious, passive, shy, and dependent, (Chambless, Hunter, & Jackson, 1982; Emmelkamp, 1980; Shafar, 1976; Torgersen, 1979) with pervasive feelings of helplessness and powerlessness (Fisher & Wilson, 1985; Torgersen, 1979). On an interpersonal level, high levels of marital conflict (Bland & Hallam, 1981; Hafner, 1982), insecure attachment styles (Mannassis,

Bradley, Goldberg, Hood, & Swinson, 1994) and sexual dysfunction (Buglass et al., 1977; Webster, 1953) have been reported. In addition, panic disorder patients have been characterized as having general difficulties in tolerating and expressing feelings of anger, frustration and disapproval (or criticism) (Brehony & Geller, 1981; Clarke & Wardman, 1985; Emmelkamp & Bouman, 1991; Korn, Plutchik, & van Praag, 1997), which has been associated with a marked tendency towards dependent and avoidant coping styles in response to conflict. In line with these findings, Chambless, Renneberg, Goldstein, and Gracely (1992) found that dependent personality disorder was the most common comorbid Axis II diagnosis amongst a group of panic disorder patients. Indeed, as mentioned previously, Axis II comorbidity with panic disorder is common, affecting between 40 and 65% of patients (Brooks et al., 1989). Further, Noyes et al. (1990) found that the greater the predominance of agoraphobic behaviours displayed in a population, the higher the incidence of co-existing personality disorder.

Unfortunately, the finding that PD appears to be strongly associated with additional interpersonal and personality difficulties, provides no indication as to whether such characterological difficulties predated the development of PD, or arose in response to it. Kleiner and Marshall (1987) investigated this issue of causality by carefully interviewing 50 patients with a *DSM-III* diagnosis of agoraphobia and panic. They found that the avoidant and dependent behaviours characteristic of agoraphobia, appeared to develop in response to a history of spontaneous panic attacks. The onset of panic attacks however was itself generally preceded by a period of severe and prolonged interpersonal conflict, and a moderate degree of general stress that was unrelated to interpersonal conflict. Indeed Kleiner and Marshall found that 84% of subjects reported marital or relationship conflict that had existed for over a year, and

commonly for several years. In addition, 64% of patients reported prolonged family conflict. On the whole, subjects described premorbid histories of unassertiveness, fear of negative evaluation, strong feelings of insecurity and a tendency toward compliant and dependent behaviours, especially in close relationships. They further reported particular difficulties in expressing anger and in coping with disapproval, which appeared to be associated with a passive or avoidant response to interpersonal difficulties.

Unfortunately, the retrospective nature of this study and its lack of a comparison group limit the generalisability of its conclusions. Nevertheless, this study adds to the growing and compelling evidence that a substantial proportion of panic disorder patients exhibit premorbid and longstanding characterological and interpersonal problems. Indeed, both Chambless (1982) and Mathews et al. (1981) reviewed the panic disorder literature for evidence of underlying dependency traits amongst this patient group. They both found overall support for the proposition that characterological dependency often precedes the onset of panic disorder, despite finding a lack of uniformity across studies. Mathews et al. noted that inconsistencies in the research findings were heavily compounded by discrepancies between measuring instruments and retrospective data collection. As such he strongly recommended the development of standardised and relevant measures and methodologies to enable further and systematic investigation of this issue.

With regard to personality traits, such as assertiveness, that impact on interpersonal functioning, Chambless et al. (1982) found that agoraphobic patients were significantly more socially anxious with respect to fears of negative evaluation by others, and significantly less assertive than a sample of college students. Patients were also considerably more uncomfortable about behaving assertively. Also

examining interpersonally-relevant emotions, Dadds, Rosenthal Gaffney, Kenardy, Oei and Evans (1993) investigated differences in the level and quality of hostility between four different *DSM-III* anxiety disorder groups, namely generalized anxiety disorder, panic disorder, agoraphobia with panic and social phobia. They found that differences in hostility existed between the different anxiety diagnoses, especially with regard to intropunitiveness (i.e., hostility directed toward the self in the form of guilt and self-criticism). Scores on intropunitiveness were higher than extrapunitiveness (i.e., hostility directed outwards in the forms of urges to act on hostility, the criticism of others and the projection of delusional hostility) indicating that all groups displayed more hostility towards themselves than toward others. In addition, the scores on intropunitiveness were considerably higher for the diagnostic groups than for norms reported in the literature. Interestingly Dadds et al. found that intropunitiveness could be predicted by the severity of anxiety symptoms for agoraphobia with panic, panic disorder and social phobia groups. However, the relationships went in the predicted direction for the agoraphobia and panic groups only, meaning that the greater the intensity of anxiety symptoms experienced by these patients, the higher their levels of inwardly-directed hostility. The authors concluded that hostility, especially that directed at the self, may play an important role in the development and maintenance of anxiety disorders.

Finally, Manassis et al. (1994) conducted a preliminary study into attachment style and anxiety disorders, particularly panic disorder. Specifically they examined attachment in 18 women with anxiety disorders, 14 of whom had panic disorder, and their 20 preschool children. The authors found all the women to have insecure attachment styles, with the group demonstrating a significantly higher rate of preoccupied attachment compared to literature norms. In addition, 16 of the children

had abnormal attachment, with the attachment style matching the mother's in 65% of the cases. Although more studies of attachment style amongst PD patients are required, particularly longitudinal studies to establish whether or not an aberrant attachment style preceded PD onset, this study contributes to those above in indicating that PD is strongly associated with interpersonal vulnerabilities.

In summary, much work remains to be done to establish systematically the existence and manner of interpersonal and personality difficulties experienced by individuals with panic disorder. Longitudinal studies in particular would be helpful in order to determine whether such problems might make individuals vulnerable to the development of panic disorder, or whether panic disorder itself has a substantial and adverse impact on relationships and personality functioning. In either case, these difficulties need to be more adequately addressed in treatment programs for panic disorder as they are likely to play a role in the maintenance of the condition. Nevertheless, despite the limitations in the research, the panic disorder literature to date suggests that interpersonal and personality difficulties, especially with respect to issues of dependency, avoidance, assertiveness and the regulation of anger, predate the onset of panic disorder and may play a role in predisposing individuals to this condition. Specifically, difficulties with regulating anxiety and anger in the context of relationships may predispose some individuals to experience escalating panic symptoms under conditions of interpersonal stress. In turn, anxieties about separation and/or autonomy may contribute to the development of agoraphobic complications, as individuals with agoraphobia often become heavily reliant on significant attachment figures in order to function with some degree of normality. Given the above evidence, it seems highly probable that for at least a subgroup of people, panic

disorder develops along a pathway that is characterized by early, and possibly ongoing, insecure attachments, and associated emotion regulation problems.

### *3.3 Summary of PD research in light of attachment theory:*

#### *Rationale for current study*

Studies of panic disorder (PD) with or without agoraphobia, have revealed that these patients report a higher proportion of anxiety disorders in childhood than healthy controls, with childhood separation anxiety disorder specifically occurring in 20-50% of patients. They also report more distressing childhood events involving separation and loss from parental figures relative to healthy controls, and this association might be more closely related to PD than other forms of anxiety. The offspring of PD patients demonstrate elevated rates of behavioural inhibition to the unfamiliar relative to patients with non-panic conditions (although depression might be an exception). As children, panic disorder patients experienced their parents as more rejecting than healthy controls, and/or more controlling, intrusive and discouraging of autonomous behaviour. A significant majority of panic disorder patients indicate prolonged conflict within both their romantic relationships and within their family of origin prior to onset of the disorder. They describe themselves as more anxious about negative evaluation by others, less comfortable or practised in assertive behaviour than controls, and more likely to display characterological overdependence prior to panic disorder onset. Furthermore, they may have particular difficulties with regulating the expression/ experience of anger. Finally, high rates of Axis I and II comorbidity predominate in panic disorder populations. These correlates of panic disorder may or may not be specific (are not necessarily highly specific) to the disorder but predict a wide range of later pathology. Nevertheless, the wide-ranging implications and associations of panic disorder for individual and

interpersonal functioning are likely to reflect fundamental and long-standing personality vulnerabilities, rather than represent a disorder that can randomly strike otherwise psychologically healthy populations.

Based on the literature, the general description of a panic disorder patient as offered above, is highly reminiscent of an insecure attachment profile. Specifically, insecure attachment is characterized by less actual sensitive responding by the mother to the infant, and retrospective recall of parents as less warm and more rejecting than seen for securely attached individuals. Furthermore, an insecure romantic attachment style in the adult is associated with lower self-esteem, greater loneliness, less relationship satisfaction, more difficulties with regulating negative emotions such as anger and sadness, more anxiety, depression, fear of negative evaluation and more somatic symptoms than secure attachment styles. Individuals classified as insecure in adulthood are also more likely to have experienced parental divorce or other adverse childhood events, and are more likely to have been classified as insecurely attached in childhood (and so to have had issues with separation anxiety). Furthermore, although infant temperament does not contribute to a secure attachment, a difficult temperament may interact with deficient parental resources to potentiate inadequate caregiving and increase the chances of an insecure attachment. Given the increased likelihood of an inhibited temperament amongst PD offspring, the increased risk for anxiety disorders in general and PD specifically, conferred by familial association with PD, and the challenge to social and emotional resources of parenting sensitively whilst contending with a mental health problem, one would expect higher than normal rates of insecurely-attached infants amongst PD-Ag patients. Finally, individuals with an insecure attachment are at greater risk for later psychopathology. Indeed

psychiatric disorder is nearly always associated with nonautonomous (i.e. not secure) attachment states of mind.

Guidano (1987) and Liotti (1991) have proposed complementary developmental models for panic disorder etiology that link early attachment experiences with the child's subsequent ability to regulate emotion – particularly with regard to negative and/or attachment-related emotion. These models also take into account findings regarding the panic disordered patient's sensitivity to issues of control and predictability, sensitivity to the sensations/symptoms of anxiety, and cognitive bias towards interpreting ambiguous internal sensations as threatening.

In their model, panic disorder pathogenesis begins with an anxious attachment between a caregiver (let's say 'mother') and her infant. Overprotective and/or rejecting parental behaviours contribute to the development of an anxious attachment and interfere with the child's autonomous emotional development.

The consequence for the child at this stage is the development of a representation of the self as frail and incompetent. Furthermore, the caregiver's attitude towards the child's emotional expression, especially with regard to those emotions related to anxious attachment such as anxiety and hostility, lead to the prohibition of these emotions and/or to the mislabelling of them by the child. For example, the child may become fearful of losing the parent's love altogether if he/she engages in autonomous or assertive behaviour. As such, the child develops a marked sensitivity to both the loss of freedom and the loss of protection, with these systems assuming an antagonistic polarity so that separation is seen to necessarily exclude all attachment.

In order to cope with his/her own alleged frailty (or perhaps the parent's frailty) the child attempts to overcontrol interpersonal interaction and interpersonal

emotion through the use of avoidant behavioural and cognitive strategies such as compliance and distraction/ diversion of its attention. The consequence of the premature and excessive use of control over his/her emotions means that early emotional schemata (consisting of relatively concrete memories of the visceral reactions and motor patterns evoked and the situations that elicited them) are prevented from evolving into more complex meaning structures concerning representations of the self and other people, and the attribution of causality and context to feelings, and of purpose to behaviour is prevented from being verbalized.

As a consequence of the difficulty with verbalization of thoughts and feelings, negative emotions are likely to be easily evoked by difficulties in relationships and expressed through physical sensation rather than verbal language. Moreover, the experience of frequent unexplained bodily sensations coupled with the need to divert attention from emotions and interpersonal contexts results in the experience of these particular emotional schemata as somewhat frightening. The resultant physical sensations are thus likely to be interpreted concretely and catastrophically as signs of impending physical or mental illness. Particularly in a context of heightened attachment anxiety, the misinterpretation of somatic sensations activates a further fear reaction invoking greater physiological arousal that magnifies the original sensation and intensifies the fear. Panic is generated by this vicious cycle escalation.

A small number of other authors have proposed models for panic disorder and/ or agoraphobia pathogenesis that take particular note of clinical observations of the patient's sensitivity to interpersonal conflict (or personal conflict about separation and autonomy), and/or his/her difficulty assigning uncomfortable emotional responses to their causal events (e.g., Busch et al., 1991; Goldstein & Chambless, 1978; Pam, Ighilterra, & Munson, 1994; Shear, 1996). Guidano and Liotti's formulation is

compelling for a number of reasons. Firstly it is complex and integrative, broadening the exclusive focus on panic symptomatology that is characteristic of many of the biological and cognitive-behavioural theories of panic disorder etiology, and incorporating evidence of interpersonal and personality difficulties. Secondly, it convincingly draws together findings from research on attachment disturbance with evidence of these precise disturbances in the cognitive, physiological and neurobiological structures of panic patients. Unfortunately, relatively little work has been done over the years to pursue a scientific approach to validating either the above model, or the clinical observations which underpin it. This is likely to be in part due to the complexity of such research with regard to defining the target variables (e.g., ability to accurately attribute emotions to their causes) and developing valid and reliable measures to assess them. Some inconsistencies in the findings for developmental antecedents and interpersonal correlates of panic disorder, especially with regard to a hypothesis of specificity for panic disorder over other anxiety and affective disorders, may have also hampered efforts to investigate attachment-based models. The inadequacies of current assessment measures and the shifting diagnostic criteria for panic disorder with/without agoraphobia, as well as high comorbidity rates with other affective and anxiety disorders have indubitably contributed to a lack of clarity in the question of panic disorder etiology. Furthermore, a general assumption of homogeneity in the panic disorder population which has been inherent in much panic research, may well have contributed to authors concluding that inconsistencies in the research reflected nonsignificant findings (rather than heterogeneity) and hence were not worth pursuing further.

With regard to heterogeneity, a literature search of “panic and subtype or hetero\* [to cover the terms heterogeneity or heterogeneous]” using the PsychInfo

database reveals that numerous recommendations have been made for PD to be classified into subtypes according to various criteria. These include classification on the basis of the primacy of respiratory symptoms in panic attacks, response to biological challenge, the accompaniment of fear with a physiological panic attack, the extent/presence of familial panic disorder, the position of panic disorder as the primary or secondary Axis I diagnosis, the presence/ extent of agoraphobia, and the age of onset of PD(early vs. late). Further sources of evidence that panic disorder might be heterogeneous are the existence of two possible peaks for age of onset, and the fact that some PD-Ag patients show anxiety sensitivity, whereas others do not.

With the heterogeneity of panic disorder patients in mind, Goldstein and Chambless (1978) proposed a model for agoraphobia that differentiated “complex agoraphobia” from “simple agoraphobia”. In the latter agoraphobic symptoms were precipitated by panic attacks produced by drugs or illness and recovery was generally fast after these conditions had been attended to. Also in keeping with a proposition of heterogeneity, Manicavasagar and Silove (e.g., 1997; Manicavasagar, Silove, & Curtis, 1997; Manicavasagar, Silove, Wagner, & Hadzi-Pavlovic, 1999) have proposed that panic disorder samples are confounded with comorbidity or misdiagnosis for patients whose primary disorder would be better characterized as adult separation anxiety disorder.

### 3.3.1 *Aims*

The purpose of this study, in light of the above information, is to investigate the relationships between panic disorder vulnerability and attachment, while addressing the limitation of an assumption of homogeneity inherent in much of the past research.

Specifically, this study aims:

1. to investigate the proposal that the pathway to panic disorder is not homogeneous. It is the contention of this study that a definable subset of panic sufferers will demonstrate histories suggestive of significant attachment-related issues in childhood.
2. to establish the relationship between early experiences with certain parenting styles based on retrospective recall, the development of childhood separation anxiety and current attachment style functioning for individuals with panic disorder.
3. to investigate the relationship between attachment style and panic attacks, such that panic expression would be expected to be a function of emotion regulation.

The clinical importance of clarifying the issue of heterogeneity for panic disorder etiology cannot be overstated. While it is likely that “uncomplicated” subtypes of the disorder respond reasonably well to pharmacological or cognitive-behavioural treatments, clinical studies have shown that a significant proportion of panic disorder patients either do not respond to treatment, or suffer relapses following treatment. It is likely that this group with treatment-resistant panic disorder will require more intensive treatment to address chronic avoidance behaviours. Moreover the underlying psychological and interpersonal difficulties noted amongst at least a subset of panic disorder patients, are likely to reflect more fundamental pathological processes that will need to be addressed in any treatment applied, if significant and stable clinical gains are to be made. The ability to identify panic disorder subtypes will allow for precious clinical resources to be distributed more effectively according to need. Furthermore, establishing the importance of attachment-related variables on panic disorder pathogenesis and patient functioning will enable treatment programs to

be devised that focus in on relevant maladaptive relationship patterns that may be contributing to current difficulties. A clear focusing and refocusing by practitioners on the most salient issues for panic disorder patients will contribute to more effective treatment practices.

### 3.3.2 *Hypotheses*

With respect to the issue of heterogeneity, it was the contention of this study that individuals with panic disorder, will differ with respect to their level of reported separation anxiety in childhood.

Furthermore, it is predicted that a definable subset of panic disorder patients will be able to be identified on the basis of childhood separation anxiety symptoms, perception of having experienced maladaptive parenting and a current insecure attachment style.

Finally, it is expected that the regulation of anger will mediate the relationship between insecure attachment dimensions and panic attack frequency, such that the greater the degree of suppression of anger (or holding it in), the higher the frequency of panic attacks.

### 3.4 *Design*

The above aims will be achieved through the use of a quantitative study using self-report questionnaires to assess panic disorder status, childhood separation anxiety symptoms, attachment style and the regulation of anger. Descriptive statistics and a logistic regression will be used to assess panic disorder heterogeneity, and to determine the relationships between panic disorder, parental perceptions, separation anxiety and attachment style. Furthermore, a series of linear regressions will be used to establish whether anger suppression mediates a relationship between insecure

attachment and panic frequency. All statistical analyses will be conducted using SPSSx version 11.0.

## Chapter 4: Method

### 4.1 Participants

Participants were undergraduate students from the Psychology and Nursing departments of an Australian university. The students accessed were from different year levels within their respective courses. The majority of participants (i.e., 78%) were enrolled in full-time courses. All participation was completely voluntary. In total, 242 people participated in the study, 79% of whom were female. Subjects were aged between 17 and 55 (mean = 23.3, s.d. = 8.0).

### 4.2 Materials

The materials for this study consisted of a self-report survey (refer to Appendix A) that was distributed to subjects. The survey contained an information sheet and consent form; a section that requested basic demographic information; and a section that was designed to assess panic and agoraphobic symptomatology. The remainder of the booklet comprised a number of previously published questionnaires. These included: -.

- *Anxiety Sensitivity Index* (Peterson & Reiss, 1992)
- *Separation Anxiety Symptom Inventory* (Silove et al., 1993)
- *Parental Bonding Instrument*. (Parker, Tupling & Brown, 1979)
- *Relationship Scales Questionnaire* (Griffin & Bartholomew, 1994)
- *State-trait Anger Expression Inventory - RRE* (Spielberger, 1988)

The survey is described in more detail below.

#### 4.2.1 *Assessment of panic symptomatology*

This section of the survey was divided into two parts. The first part asked for general demographic information such as age and gender. Part two consisted of a number of questions which asked specifically about symptoms of panic disorder, and agoraphobic avoidance behaviours. The first 7 questions were designed to enable a gross diagnosis of panic disorder according to *DSM-IV* criteria. Subjects were asked to read a short description of a panic attack and to indicate whether they had ever experienced a panic attack as so defined. If so, subjects were asked to indicate: what symptoms they experienced during a typical panic attack, the number of panic attacks experienced in the past week and month, the timing of their very first panic attack and the circumstances that surrounded it, the severity and disruptiveness of the panic attacks, and whether they had ever experienced persistent worry about having further attacks. Participants were then asked to report on the extent of any avoidance behaviours on a series of three 5-point scales that ranged from 'never' to 'always'. Finally participants were asked whether they had ever received a diagnosis by a psychiatrist or psychologist.

#### 4.2.2 *Anxiety Sensitivity Index*

The Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992) is a 16-item self-report inventory. Participants are asked to rate the extent to which they agree or disagree with each item - pertaining to beliefs that anxiety symptoms are signs of harmful or aversive consequences, by selecting one of five points on a Likert scale. The scale ranges from *very little* (0) to *very much* (4). The total ASI score is obtained by summing the item scores, and can range from 0 to 64. Considerable evidence supports the good psychometric properties of the ASI, including evidence of its high

internal consistency (with alpha coefficients ranging from .82 to .91; Peterson & Reiss, 1992); satisfactory test-retest reliability over 3 years ( $r=.71$ ; Maller & Reiss, 1992); good construct validity as a measure of fear of anxiety as distinct from trait anxiety; and excellent criterion validity in distinguishing anxiety disordered patients from controls, and distinguishing patients with panic disorder from other anxiety disordered patients. Panic disorder patients generally score about two standard deviations above the normative mean, whereas patients with other anxiety disorders score about one standard deviation above the normative mean (see reviews by Peterson & Reiss, 1992; Taylor, 1995).

#### 4.2.3 *Separation Anxiety Symptom Inventory*

The Separation Anxiety Symptom Inventory (SASI; Silove et al., 1993a) is a 15-item scale that inquires into memories of separation anxiety in the first 18 years of life. Individual items are rated on a four-point scale ranging from *I never had this feeling* (0) to *This feeling occurred very often* (3). A summary score on the measure is derived by adding item scores. The authors of the scale recommend the application of a square root transformation to total scores. The SASI has been demonstrated to have good psychometric properties with a coherent factor structure, high internal consistency (Cronbach's Alpha > 0.80), and sound test-retest reliability (intra-class correlation coefficients ranging from 0.86 to 0.98). SASI scores remain stable over time despite changes in adulthood anxiety levels (as measured by the Spielberger State-Trait Anxiety Inventory). Furthermore, SASI scores correlate highly with independent *DSM-III-R* diagnoses of lifetime juvenile separation anxiety disorder ( $r=.79$ ,  $p<0.001$ ) and overanxious disorder ( $r=.72$ ,  $p<0.001$ ) (Silove et al., 1993a).

#### 4.2.4 Parental Bonding Instrument

The Parental Bonding Instrument (PBI; Parker et al., 1979) is a 25-item self-report instrument designed to assess the parental contribution to the parent-child bonding relationship. Participants are asked to rate the extent to which each item is applicable to their mother or father (separately) as they remember him/her in their first 16 years of life, by selecting one of 4 options on a Likert scale. The scales range from *very like*[him/her] (0) to *very unlike* [him/her] (3).

Factor analysis of the original PBI by Parker et al. (1979) resulted in 3 factors. The first factor, labelled “parental care”, was comprised of a total of 12 items (6 reversed). This care scale referred to parental interest, sensitivity and warmth, and ranged from neglect to affection. Parker et al. combined the second and third factors to form an “overprotection” subscale, which implied control and intrusiveness. This subscale ranged from the allowance of independence to strict control. These two subscales of the PBI have been extensively used, and have demonstrated good psychometric properties. Test-retest studies have demonstrated a high level of reliability with scores remaining consistent in spite of changes in respondents’ mood states (Gerlsma, Emmelkamp & Arrindell, 1990; Parker, 1990). Validity studies using corroborative witnesses (e.g., clinical interviewers, mothers, siblings) have indicated that perceived parenting as recorded on the PBI does not differ substantially from observer reports. Nevertheless debate over the factor structure has persisted, especially with respect to whether the overprotection subscale is best represented by one or two factors (e.g., Cox, Enns & Clara, 2000; Lizardi & Klein, 2002; Murphy, Brewin, & Silka, 1997; Reti et al., 2002).

From a number of proposed alternative scoring systems for the PBI, Kendler’s (1996) three factor model has received support for being the most robust with respect

to age and gender (Cox et al., 2000; Sato et al., 1999), and for being more sensitive to differences than the two factor system with regard to differentiating between depressed and non-depressed samples (Lizardi & Klein, 2002). This model used a reduced 16-item form of the PBI and found three factors: - warmth (analogous to Parker et al.'s (1979) "care" factor), protection, and authoritarianism (the latter two combined make up Parker et al.'s (1979) overprotection factor). Kendler's "protection" and authoritarianism factors can be viewed as representing restriction over the individual/personal domain, and the social domain respectively (Martin, Bergen, Roeger, & Allison, 2004). For example, item 9 of the protection factor, states "tried to control everything I did", and item 21 of the authoritarianism factor states "gave me as much freedom as I wanted".

#### 4.2.5 *Relationship Scales Questionnaire*

The Relationship Scales Questionnaire (RSQ; Griffin & Bartholomew, 1994) is a 30-item self-report measure designed to assess adult attachment style in accordance with Bowlby's theory of attachment. Respondents rate themselves using a five-point scale ranging from *not at all like me* (1) to *very much like me* (5) on a series of statements about their close relationships (e.g., I find it easy to get emotionally close to others"). The statements were derived from the Relationship Questionnaire (RQ; Griffin & Bartholomew, 1994) which consists of four short paragraphs describing the secure, preoccupied, dismissing and avoidant attachment styles. The paragraphs read as follows:

Secure	It is easy for me to become emotionally close to others. I am comfortable depending on them and having them depend on me. I don't worry about being alone or having others not accept me.
Dismissing	I am comfortable without close emotional relationships. It is very important to me to feel independent and self-sufficient, and I prefer not to depend on others or have others depend on me.
Preoccupied	I want to be completely emotionally intimate with others, but I often find that others are reluctant to get as close as I would like. I am uncomfortable being without close relationships, but I sometimes worry that others don't value me as much as I value them.
Fearful	I am uncomfortable getting close to others. I want emotionally close relationships, but I find it difficult to trust others completely, or to depend on them. I worry that I will be hurt if I allow myself to become too close to others.

The Relationship Scales Questionnaire was designed to enable continuous and reliable ratings (derived from multiple items) of individuals on the four prototypes of secure, dismissing, fearful and preoccupied. The preoccupied and fearful subscales are comprised of four items each, while the other two contain five items. The additional 13 items were derived from Hazan and Shaver's (1987) three-category measure of attachment style, and can be used to compute other attachment scales.

Siegert, Ward, and Hudson (1995) assessed the RSQ's structure via a factor analysis using all 30 items of the RSQ and found little evidence to support the four proposed subscales. Instead they found a robust two-factor structure accounting for 29% of the total scales variance (Siegert, personal correspondence), with one factor relating to anxiety/ security and the other concerned with closeness/ independence. This two dimensional factor structure is consistent with other analyses of the factor structure of attachment measures generally (e.g., Brennan, Clark, & Shaver, 1998). It is also theoretically consistent with the underlying dimensions that Bartholomew

(1990) proposed for her four-categories of attachment style. Further, the bi-dimensional classification system enables the derivation of four categories that are synonymous with the four attachment styles of secure, dismissing, fearful and preoccupied if required.

#### 4.2.6 *Anger Expression - State-trait Anger Expression Inventory*

The State-Trait Anger Expression Inventory- Revised Research Edition (STAXI; Spielberger, 1988) is a self-report measure designed to assess an individual's experience and expression of angry emotion. Three of the STAXI's main scales (i.e., the anger expression scales; AX-STAXI) were specifically designed to measure the expression of anger. Anger expression is conceptualized as having three major components: an outward expression (ax/out); an inward expression, also referred to as anger suppression (ax/in); and control (ax/con). These three scales are comprised of 8 statements each and presented in random order. Respondents are asked to rate each statement on a 4-point scale ranging from *almost never* (1) to *almost always* (4) according to how often they generally react as described when angry or furious. For example, item 6 states "When angry or furious...I withdraw from other people". People with high ax/in scores frequently experience intense angry feelings, but tend to suppress these feelings rather than express them either physically or verbally. On the other hand, people with high ax/out scores frequently experience anger which they direct in aggressive behaviour towards other persons or objects in the environment, including through the use of sarcasm and criticism. People with high ax/con scores tend to invest a great deal of energy in monitoring and preventing the experience and expression of anger.

The STAXI has been demonstrated to have good psychometric properties, with a consistent factor structure (Forgays, Kirby Forgays, & Spielberger, 1997), good

internal reliability (Alpha coefficients range from .73 to .85) and good convergent and predictive validity (Spielberger, 1988).

### 4.3 Procedure

Ethics approval was obtained from the Faculty of Arts Human Research Ethics Committee (refer to Appendix B). After obtaining permission from the appropriate teaching staff, students were approached during their lectures or laboratory tutorials and invited to participate in this research project. The aims of the research were briefly explained, with verbal and written assurances that participation was voluntary and that participants' responses would be kept completely confidential. Students were asked to complete a survey in their own time and return them to their tutorial/ lecture in the following week when they would be collected by the researcher or their lecturer/ tutor. Students were also offered the option of mailing in their responses, with self-addressed envelopes provided.

Respondents were classified into panic groups according to whether or not they endorsed items suggestive of a lifetime diagnosis of panic disorder according to the diagnostic criteria of the *DSM-IV*. Individuals were classified into the "panic" group if they met the diagnostic criteria for panic disorder, and into the "no panic" group if they had never had a panic attack. Those participants who reported having experienced panic or agoraphobic symptoms but at insufficient levels for a lifetime panic disorder diagnosis, were classified as having subclinical panic (i.e., "subpanic"). The specific hierarchical criteria used to determine classification into panic groups are detailed in Appendix C.

## Chapter 5: Results

All statistical analyses were conducted using SPSS for Windows (version 11). Parametric statistics were primarily used to test the study's hypotheses. These consisted predominantly of regressions (both linear and logistic) and Pearson's correlations.

Factor analyses were conducted for both the Parental Bonding Index (PBI) and the Relationship Scales Questionnaire (RSQ) due to debate in the literature about their respective internal structures. For the PBI, a principal components analysis with varimax rotation was conducted. This analysis yielded three clear factors for the 25-item PBI that accounted for 60% of the total variance. These factors were almost identical with Kendler's (1996) three-factor model (except that they contained a breakdown of all 25 items, rather than Kendler's reduced pool of 16 items) that has been demonstrated to have psychometric superiority to other PBI models (Cox et al., 2000).

The derived factors were labelled parental warmth, overprotection (items 8, 9, 10, 13, 19, 20, 23) and authoritarianism (items 3, 15, 21, 22, 25). The "warmth" factor contained items referring to perceptions of the parent as loving and affectionate (e.g., item 6, "Was affectionate to me") and had a very high internal reliability coefficient of .95 for the maternal responses, and .96 for paternal responses following the removal of items 2 and 12. This factor consisted of items 1, 4, 5, 6, 11, 14, 16, 17, 18, 24. The "overprotection" factor referred to the fostering of dependency and parental intrusiveness (e.g., item 13, "Tended to baby me"). "Authoritarianism" contained items that referred to the encouragement or discouragement of independence and autonomy (e.g., item 21 "Gave me as much freedom as I wanted"). All items, except for item 7, loaded on identical factors for both the mother and father

reports. Item 7 loaded moderately on all three factors for both parents, but loaded slightly more highly on the overprotection factor for the mother reports, and slightly more highly on the authoritarianism factor for the father reports. For this reason, item 7 was deleted from any further analyses. Both the overprotection and authoritarianism factors had good internal reliability with *Alpha* coefficients of .82 and .80 respectively for maternal responses, and .77 and .83 respectively for paternal responses.

For the RSQ, all 30 items of the scale were entered into a principal components analysis with varimax rotation. The resulting solution produced two strong factors, together accounting for 37.3% of the total scale's variance, and six other smaller ones with eigenvalues greater than one. The subsequent extraction of two forced factors led to a solution that was highly consistent with Siegert et al.'s (1995). These two factors were labelled "relationship anxiety" and "avoidance" in keeping with the meta-analytical findings of Brennan et al. (1998) in the broader attachment literature. "Relationship anxiety" accounted for approximately 20% of the scale's variance and contained items referring to worry about love not being reciprocated in relationships (e.g. Item 23, "I often worry about being abandoned"). It was composed of items 4, 8, 9, 11, 14, 16, 18, 21, 23, 25, and 28, and had high internal reliability (*Alpha* = .86). "Avoidance" accounted for approximately 18% of the total variance and consisted of items referring to the need for independence and distance versus a desire for intimacy. An assessment of its internal reliability determined good reliability (*Alpha* = .76) following removal of items 10 and 27. The avoidance scale consisted of items 1, 2, 3, 5, 7, 12, 13, 17, 19, 20, 24, 26, 29, 30. Items 6, 15, and 22 did not load highly on either factor and were deleted from further analyses. Given that the 2-factor solution extracted for the data of the present study

was comparable with Siegert et al's but able to account for more variance (37.3% vs. 29%), it was decided to use the present study's factor solution for subsequent analyses.

Square root transformations were applied to the separation anxiety (SASI) and anxiety sensitivity (ASI) scores in response to indications that these variables departed significantly from normality. The transformations were performed prior to the variables being entered into statistical analyses. The 'relationship anxiety' factor of the Relationship Scales Questionnaire did not depart significantly from normality. For the remaining variables (RSQ- avoidance, all PBI and all STAXI scales), transformations did not significantly improve them with regard to an assumption of normality and, as such, it was decided to proceed with the original scores. An *alpha* level of .05 was used for all statistical tests.

The following results chapter is divided into 5 sections. Section 1 presents the mean distribution of each variable in relation to the normative mean data available for the individual measures. Section 2 provides an overview of the relationships found between the dependent and independent variables. The subsequent three sections individually address the hypotheses stated earlier. Specifically, section 3 assesses the homogeneity of the self-reported panic disorder group with respect to childhood separation anxiety. Section 4 uses a simple logistic regression procedure to assess the relationship between panic classification, perceptions of early parental behaviour, attachment style, and childhood separation anxiety. Finally section 5 reports on the relationship between panic frequency, attachment and emotion regulation (i.e., anger expression) using a series of linear regression equations.

## 5.1 Variable Distributions and Normative Data

### 5.1.1 Self-reported Panic Disorder Classifications

Of a total of 242 participants, 22% ( $n = 52$ ) were classified as having panic disorder (i.e., 'panic') based on their reports of their experiences with panic disorder symptomatology, and according to *DSM-IV* diagnostic criteria. An additional 19% ( $n = 45$ ) were classified as having subclinical panic disorder symptomatology (i.e., 'subpanic'). That is, these participants reported either 1) having had panic attacks with fewer than the *DSM-IV* required four symptoms (i.e., limited-symptom attacks), or 2) they reported clinical panic attacks but no anticipatory anxiety, or 3) they reported no experience with panic attacks but extensive agoraphobia avoidance. The remaining 60% of participants ( $n = 145$ ) reported no panic or agoraphobic symptoms at all and were classified as 'no panic'. All in all, a total of 36% of the sample ( $n = 88$ ) reported having experienced a panic attack in their lifetime (both full-blown and limited symptom attacks included). Of these, 45% ( $n = 40$ ) reported recent experiences with panic attacks (i.e., at least one attack in the month prior to completing the survey). These findings compare well with previous reports that about one third of university students report a lifetime history of panic on self-report measures of panic history (e.g., Norton, Dorward, & Cox, 1986; Watt & Stewart, 2000). Panic frequency (i.e., the number of panic attacks in the month prior to completing the survey) for individuals who reported having had recurrent panic attacks in their lifetime, ranged between 0 and 50 ( $M = 1.81$ ).

### 5.1.2 Anxiety Sensitivity Index

The ASI provides a measure of an individual's fear of anxiety symptoms and sensations in the adult population. A high level of anxiety sensitivity is considered to

be a risk factor for anxiety disorders in general, and panic disorder in particular. Furthermore, scores on the ASI have been shown to significantly discriminate between people with panic disorder, people with other anxiety disorders, and healthy controls.

The mean scores of participants on the ASI and SASI are reported according to panic disorder classification and presented in Table 1 below. Although the total scores for these two scales were subjected to a square root transformation prior to entering them into further analyses, the untransformed data are presented for the ASI only, in order to facilitate comparison with the normative means published in the literature. Normative data for the SASI are already reported by the authors in transformed form (Silove et al., 1993a).

As shown in Table 1, anxiety sensitivity increased substantially across panic classification with the panic group demonstrating the highest scores, and the 'no panic' group the lowest. In order to examine the strength of the differences between mean ASI scores across panic classification, a one-way ANOVA was conducted with ASI scores as the dependent variable, and panic classification as the independent variable. The results of the ANOVA revealed that the mean ASI scores were significantly different across groups ( $F_{(2, 238)} = 35.16, p < .001$ ). Scheffe's post hoc analysis indicated that these significant differences occurred across all levels of panic classification (for panic and subpanic groups,  $M \text{ diff} = 8.80, SE = 2.03, p < .001$ ; for subpanic and no panic groups,  $M \text{ diff} = 5.47, SE = 1.71, p < .01$ ). These findings provide support for the proposal that people who suffer from panic attacks have a greater fear of the symptoms of anxiety than those who panic infrequently, and infrequent panickers in turn have a greater fear of anxiety symptoms than people without panic.

The average ASI score for the whole sample was not significantly different ( $t_{(240)} = 1.19, p > .05$ ) from the normative figures reported in the ASI manual for non-clinical college students (i.e.,  $M = 19.01, SD = 9.11$ : Peterson & Reiss, 1992). Moreover, the mean ASI score for the panic group was not significantly different from either Cox, Endler, and Swinson's (1991) finding of a mean ASI score of 30.1 for 38 college students who suffered from panic attacks, or Otto, Pollack, Sachs, and Rosenbaum's (1992) finding of a mean ASI score of 30.5 ( $SD = 11.2$ ) for 50 panic disorder patients ( $t_{(51)} = -.25, p > .05$ ). As such, the published normative ASI means for panic sufferers and panic disorder patients provide evidence of convergent validity that the self-reported panic disorder classification used in this study was significantly able to differentiate between panickers, subclinical panickers and non-panickers, and that participants classified into the panic group have significant levels of panic disorder symptomatology.

Table 1

*Means and standard deviations for the Anxiety Sensitivity Index (ASI) and Separation Anxiety Symptom Inventory (SASI) by panic disorder classification.*

Measures		Panic	Subclinical Panic	No Panic	Total
ASI	<i>M</i>	30.08	21.27	15.81	19.88
	<i>SD</i>	12.36	9.98	8.90	11.43
	<i>n</i>	52	44	145	241
SASI	<i>M</i>	3.75	3.67	3.07	3.33
	<i>SD</i>	1.20	1.24	1.11	1.19
	<i>n</i>	52	45	144	241

### 5.1.3 Separation Anxiety Symptom Inventory

The SASI provides a measure of the extent that a given individual experienced separation anxiety symptoms during his/her childhood. Childhood separation anxiety is a risk factor for the development of adult anxiety disorders such as panic disorder.

As presented in Table 1, separation anxiety symptoms, as measured by the SASI, increased across panic classification, with the panic group reporting the highest levels of childhood separation anxiety and the no panic group reporting the lowest. A one-way ANOVA was conducted on the mean SASI scores, with SASI scores entered as the dependent variable and panic classification as the independent variable, in order to determine the strength of the apparent differences in separation anxiety symptoms between panic groups. The results of the ANOVA indicated that there were significant differences between panic groups ( $F_{(2, 238)} = 8.96, p < .001$ ). Scheffe's post hoc analysis indicated that there was no significant difference between the panic and subpanic groups, but significant differences between the two panic groups and the no panic group (for panic and no panic groups,  $M \text{ diff} = .68, SE = .19, p < .01$ ; for subpanic and no panic groups,  $M \text{ diff} = .60, SE = .20, p < .05$ ). These findings provide support for the proposal that high levels of separation anxiety in childhood puts an individual at risk for the development of panic symptomatology. Nevertheless, it provides only limited support for the proposition that high levels of childhood separation anxiety specifically predispose an individual to the development of panic disorder in later life, as individuals with self-reported panic disorder experienced similar levels of separation anxiety to individuals with subclinical panic despite more severe levels of current psychopathology.

On the whole, participants' mean SASI scores were significantly higher than Silove et al.'s (1993a) normative mean of 3.1 ( $SD = 1.1$ ;  $t_{(240)} = 3.01$ ,  $p < .01$ ) for their nonclinical sample, suggesting that the participants in this study had higher than average levels of childhood separation anxiety. This finding probably reflected the fact that females represented approximately 80% of the sample for this study and only about 40% in Silove et al.'s study. Females have been found to return higher scores on the SASI than males (Silove et al., 1993a). The mean score for the panic group was comparable with the mean SASI score of Silove et al. (1993b) sample of 79 patients from an anxiety clinic ( $M = 3.7$ ;  $t_{(51)} = .31$ ,  $p > .05$ ).

#### 5.1.4 Parental Bonding Instrument

The PBI assesses an individual's perceptions of his/her mother's and father's parenting behaviours. Specifically, it provides a measure of the individual's memories of his/her parents' behaviour along three dimensions assessing the degree of parental warmth or caring, overprotectiveness, and authoritarianism. Mean scores were generated separately for mother and father for the three subscales of the Parental Bonding Instrument across panic disorder classification (refer to Table 2 below).

As shown in Table 2, fathers were generally perceived as less caring, less overprotective, but comparably authoritarian as mothers. Furthermore, the mean scores outlined in Table 2 indicate a trend for the panic group to perceive both parents as less warm, more overprotective, and more authoritarian than the subclinical panic group, who in turn view their parents as less warm, more overprotective and more authoritarian than the panic-free group. Perceptions of paternal authoritarianism defy this trend slightly with the subpanic group offering the lowest score. In order to test the strength of the observed differences in parental perceptions across panic disorder classifications, a Multivariate Analysis of Variance (MANOVA) was conducted with

the PBI subscales entered collectively as the dependent variables and panic classification entered as the independent variable.

Table 2

*Means and standard deviations for the subscales of the parental bonding instrument (PBI) by panic disorder classification, reported separately for mother and father.*

Parent	PBI subscale		Panic	Subclinical Panic	No panic	Total
Mother	Warmth	<i>M</i>	19.08	22.07	23.31	22.16
		<i>SD</i>	9.01	7.25	6.88	7.61
		<i>n</i>	52	45	143	240
	Overprotection	<i>M</i>	7.25	6.22	6.03	6.33
		<i>SD</i>	5.32	4.69	4.63	4.80
		<i>n</i>	52	45	143	240
	Authoritarianism	<i>M</i>	6.23	5.51	5.36	5.58
		<i>SD</i>	3.92	3.29	3.40	3.50
		<i>n</i>	52	45	143	240
Father	Warmth	<i>M</i>	16.78	18.33	19.36	18.62
		<i>SD</i>	8.77	8.91	8.25	8.52
		<i>n</i>	49	45	138	232
	Overprotection	<i>M</i>	6.39	5.38	5.13	5.44
		<i>SD</i>	5.15	3.47	4.27	4.34
		<i>n</i>	49	45	138	232
	Authoritarianism	<i>M</i>	6.43	5.62	5.75	5.87
		<i>SD</i>	4.29	3.46	3.74	3.80
		<i>n</i>	49	45	138	232

The results of the MANOVA indicated that there was a significant main effect for panic ( $Roy's = .066, F_{(6, 223)} = 2.47, p < .05$ ). Scheffe's post hoc analysis indicated that there was a significant difference between mean levels of reported maternal warmth for those persons classified as having panic versus the no panic group ( $M_{diff} = -4.45, SE = 1.25, p < .01$ ). There were no significant differences in reported maternal warmth between panic and subpanic, or between subpanic and no panic means. Furthermore, univariate analysis revealed that none of the other maternal or paternal factors demonstrated significant differences between the panic groups. These findings provide support for the proposition that maternal parenting styles have a greater impact on later mental health than perceptions of paternal parenting styles. Moreover, the trends in the data provide support for the proposition that low levels of maternal care and high levels of overprotection and restriction may predispose some people to the development of psychopathology in general and panic disorder in particular. The fact that only the maternal warmth factor significantly differentiated between panic and no panic groups is consistent with the panic disorder literature that found low maternal care to be the strongest predictor of later psychopathology, whereas maternal overprotection was more variable across studies in its ability to predict panic disorder. No normative data are available for this questionnaire based on the three factors as used for this study.

#### 5.1.5 *Relationship Scales Questionnaire*

The RSQ provides a measure of an individual's attachment style along two dimensions – relationship anxiety and avoidance. 'Avoidance' refers to the extent that persons profess to value independence and distance over intimacy and closeness. 'Relationship anxiety' refers to the extent that persons worry about abandonment, or about love not being reciprocated in their relationships. Low avoidance and low

relationship anxiety are indications of a secure attachment. All the other high/low combinations (e.g., high avoidance, low anxiety) represent insecure attachments.

As shown in Table 3 below, the desire for independence and distance (i.e., ‘avoidance’) and anxiety about relationships (i.e., ‘relationship anxiety’) tended to increase across panic classifications, with the no panic group indicating the lowest levels of both. The strengths of the observed differences in relationship anxiety and avoidance across panic groups were assessed using a MANOVA, with the RSQ subscales entered as the dependent variables, and panic classification entered as the independent variable. The results indicated that there was a significant main effect for panic classification on attachment variables ( $Pillai's = .105$ ,  $F_{(4, 478)} = 6.63$ ,  $p < .001$ ). Both avoidance ( $F_{(2, 239)} = 6.83$ ,  $p = .001$ ) and relationship anxiety ( $F_{(2, 239)} = 10.47$ ,  $p < .001$ ) demonstrated significant differences across classifications according to univariate analysis. Furthermore, Scheffe’s post hoc analysis identified that, on avoidance, the panic and subpanic groups were not significantly different from each other, but they *were* significantly different from the panic-free group ( $M \text{ diff} = 5.15$ ,  $SE = 1.59$ ,  $p < .01$  and  $M \text{ diff} = 4.25$ ,  $SE = 1.68$ ,  $p < .05$  respectively). For relationship anxiety, the panic group was significantly different from no panic group ( $M \text{ diff} = 6.29$ ,  $SE = 1.38$ ,  $p < .001$ ). However, there were no significant differences on relationship anxiety between those classified with panic versus those classified with subpanic, nor between the subpanic and no panic groups. These findings provide support for the proposition that individuals with panic disorder are more likely to have an insecure attachment style than those without panic disorder. Specifically, this means that people with panic disorder show higher levels of anxiety than people without panic symptomatology about being abandoned or sufficiently loved in

relationships, and as such they may avoid intimacy for fear of their anxieties being realised.

Table 3

*Means and standard deviations for the factors of the Relationship Scales Questionnaire (RSQ), and Anger Expression scales of the State-Trait Anger Expression Inventory (STAXI) by panic disorder classification.*

Measures	Subscales		Panic	Subclinical Panic	No panic	Total
RSQ	Avoidance	<i>M</i>	51.38	50.91	45.52	47.76
		<i>SD</i>	11.40	9.82	11.00	11.19
		<i>n</i>	52	44	146	242
	Relationship Anxiety	<i>M</i>	34.75	31.20	28.36	30.25
		<i>SD</i>	9.27	7.50	8.55	8.88
		<i>n</i>	52	44	146	242
STAXI	Anger In	<i>M</i>	19.17	17.44	16.89	17.48
		<i>SD</i>	4.86	3.80	4.38	4.46
		<i>n</i>	52	45	145	242
	Anger Out	<i>M</i>	16.81	16.11	15.81	16.08
		<i>SD</i>	4.46	3.32	3.71	3.82
		<i>n</i>	52	45	145	242
	Anger Control	<i>M</i>	22.33	22.11	23.03	22.71
		<i>SD</i>	4.87	4.40	4.51	4.57
		<i>n</i>	52	45	145	242

### 5.1.6 *Anger-Expression scales of the State-Trait Anger Expression Inventory*

The anger expression scales of the STAXI (AX-STAXI) provide a measure of the style in which a person expresses anger. Specifically, the AX-STAXI assesses the tendency of an individual to suppress anger (i.e., experience it inwardly; ‘anger in’), to express anger (‘anger out’), and to control their experience of anger (‘anger control’).

As outlined in Table 3 above, the tendency to both express and suppress anger was found to increase across panic classification, with the panic disorder group evidencing the highest scores for both of these subscales. The experience of controlling anger, however, did not follow this trend of increasing with increasing levels of pathology. In order to determine the strength of the observed differences in anger expression between panic groups, a MANOVA was conducted with the AX-STAXI scales entered as dependent variables, and panic classification acting as the independent variable. The results indicated a significant main effect for anger expression ( $Pillai's = .054, F_{(6, 476)} = 2.21, p < .05$ ). Univariate analysis and Scheffe's post hoc analysis found that there was a significant difference between mean levels of anger suppression for those persons classified as panic disordered versus the panic-free group ( $M\ diff = 2.28, SE = .71, p < .01$ ). There were no significant differences however on reported levels of anger suppression between the panic and subpanic groups, nor the subpanic and panic-free groups. In addition, no significant differences were found between panic groups for the expression of anger (i.e., anger out) nor for its control. These findings provide support for the proposal that individuals with panic disorder have difficulties with the regulation of anger, so that

they may experience more anger, and suppress their feelings of anger significantly more often than people without any panic symptomatology.

The mean scores for anger out and anger control for the whole sample were comparable with the Spielberger's (1988) reported normative data ( $M = 15.66$ ,  $SD = 4.00$ , and  $M = 22.48$ ,  $SD = 5.00$  respectively) for undergraduate college students ( $t_{(241)} = 1.70$ ,  $p < .05$ , and  $t_{(241)} = .77$ ,  $p > .05$  respectively). For anger in, the mean score of the participants in this study was significantly higher than the normative mean of 16.72 ( $SD = 4.18$ ;  $t_{(241)} = 2.66$ ,  $p < .01$ ), suggesting that these participants may have had higher than average levels of anger suppression.

## 5.2 *Relationships between the Anxiety Sensitivity Index and the independent variables*

As a preliminary step to the hypotheses testing, a Pearson's correlation analysis was conducted using all the independent variables measured for the study in order to examine for the potential confounding effects of high inter-correlations between them. In addition, the Anxiety Sensitivity Index was included in the analysis as an index of the study's dependent variable -panic classification, as the ASI represents a continuous variable that differentiated well between the panic groups and is significantly associated with vulnerability for panic attacks. The ASI was included in order to assess the relationships between the study's dependent and independent variables. The correlations matrix is presented below in Table 4.

As shown in Table 4, the Anxiety Sensitivity Index (ASI) was found to have significant and positive low to moderate correlations with most of the independent variables, with maternal warmth (PBI) providing the only negative correlation of those that were significant. The only nonsignificant correlation was between the ASI and the maternal overprotection subscale of the Parental Bonding Instrument (PBI).

These relationships indicate that individuals with high anxiety sensitivity also tended to have had high separation anxiety symptomatology in childhood, and to have perceived their mothers as being relatively cool and insensitive and somewhat authoritarian. Furthermore, individuals with high ASI tended to report higher levels of desire for independence in their contemporary close relationships, and simultaneously experienced considerable anxiety about abandonment in these relationships. Finally, individuals high on anxiety sensitivity also tended to suppress their feelings of anger, and those people who professed experiences with panic attacks, and had high ASI, tended to have more panic attacks per month than those with low ASI. These findings are consistent with the proposition that individuals who have high ASI and are thus more highly predisposed to developing panic disorder are more likely to a) have experienced separation anxiety in childhood, b) to perceive their parents as unsupportive, c) to have an insecure attachment style, and d) to have problems with emotion regulation, particularly with respect to anger and anxiety (as reflected by panic frequency).

As seen in Table 4, about half of the significant correlations between the independent variables were relatively weak, with the other half being of moderate strength. The fact that there were no strong correlations provides support for the proposal that the independent variables were not convergent with one another, and thus each should be capable of assessing unique aspects of the variance between the panic disorder classifications.

Table 4

## Correlations between Anxiety Sensitivity Index scores and the independent variables for the total sample (N=242)

	ASI	SASI	Mother Warmth	Mother Overprotection	Mother Authoritarianism	Father Warmth	Father Overprotection	Father Authoritarianism	Avoidance	Rship anxiety	Anger In	Anger Out	Anger Control	Panic frequency
ASP	--													
SASP	.48**	--												
Mother Warmth	-.26**	-.08	--											
Mother Overprotection	.12	.16*	-.42**	--										
Mother Authoritarianism	.13*	.03	-.50**	.55**	--									
Father Warmth	-.15*	.01	.39**	-.10	-.08	--								
Father Overprotection	.23**	.15*	-.13*	.42**	.18**	-.27**	--							
Father Authoritarianism	.10	.03	-.18**	.23**	.41**	-.36**	.56**	--						
Avoidance	.39**	.22**	-.34**	.19**	.09	-.31**	.20**	.14*	--					
Relationship anxiety	.46**	.33**	-.26**	.19**	.10	-.21**	.23**	.10	.34**	--				
Anger In	.36**	.21**	-.27**	.19**	.07	-.16*	.14*	.06	.53**	.38**	--			
Anger Out	.04	.05	-.03	.11	.13	.06	.03	-.01	.00	.04	.11	--		
Anger Control	-.12	-.05	.09	-.08	-.12	.03	.00	.04	.02	-.05	.01	-.59**	--	
Panic frequency	.27*	.18	-.19	.19	.14	-.31**	.19	.20	.21	.27*	.30**	.05	-.16	--

\*\* . p &lt; 0.01, two-tailed.

\* . p &lt; 0.05, two-tailed.

a. Anxiety Sensitivity Index scores

b. Separation Anxiety Symptom Inventory scores

c. no. of panic attacks reported in month prior to completing survey; n =88

### 5.3 Hypothesis 1: Heterogeneity

To assess the possible heterogeneity of panic disorder with respect to experiences of childhood separation anxiety, a distribution of total SASI scores by panic disorder classification was conducted. A boxplot of the distribution appears below in Figure 1.

As illustrated in the boxplot in Figure 1, the mean level of childhood separation anxiety increased across panic disorder classifications, with the Panic group demonstrating the highest mean SASI. For each of the groups depicted in Figure 1, a fairly even distribution of SASI scores is apparent with virtually the same spread of scores in the upper and lower quartiles. Despite this similarity, the majority of the scores for the panic group do not range as low as the subclinical panic group's scores, which in turn remains higher than the no panic group. As such, the above figure provides strong evidence that panic disorder is heterogeneous with respect to childhood separation anxiety, as there are at least as many individuals classified with panic disorder, who exhibit low childhood separation anxiety as exhibit high childhood separation anxiety. The same is true for both the subclinical panic and panic-free groups.

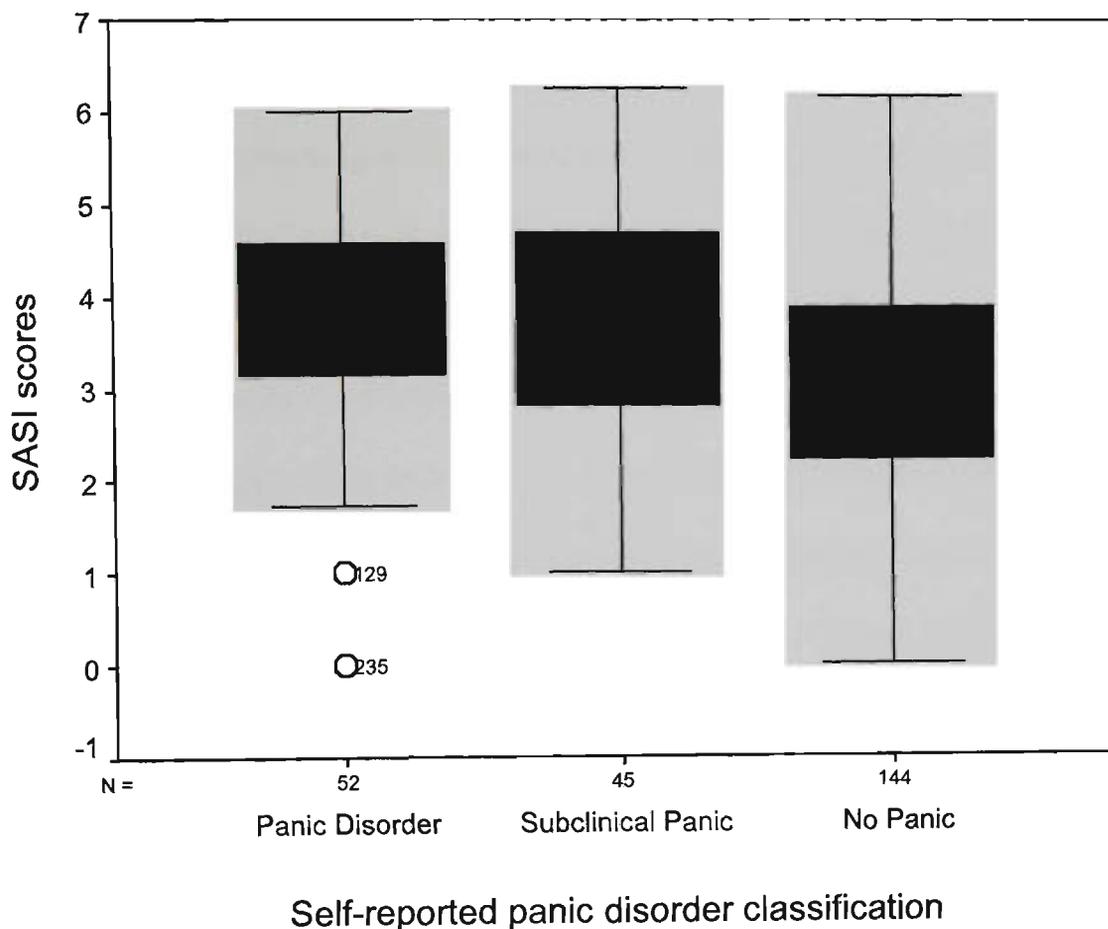


Figure 1. The distribution of total scores on the Separation Anxiety Symptom Inventory (SASI) for each of the self-reported panic classifications.

5.4 Hypothesis 2: *The relation between panic disorder, separation anxiety, parental bonding and attachment.*

In order to determine whether a set of attachment-relevant variables could predict panic disorder classification, a series of logistic regression analyses were conducted. Tabachnick and Fidell (1996) recommend the use of logistic regression for the prediction of group membership from a set of variables, when the predictors are not all normally distributed, as is the case here. Logistic regression enables the

researcher to use goodness-of-fit tests (i.e.,  $\chi^2$  statistic) to choose a model that does the best job of prediction with the fewest number of variables. The simplest (and worst-fitting) model will include only the equation constant and none of the predictors, whereas, assuming all the predictors are significantly related to outcome, the best-fitting model will include the constant, all the predictors, and possibly interactions between the predictors. In line with the hypothesis that a subgroup of people with panic disorder will report histories with childhood separation anxiety, perceptions of their parents as rejecting and overprotective, and an insecure attachment style characteristic of current relationship difficulties, the specific predictors used in the logistic regression equation were scores on the separation anxiety symptoms inventory (SASI), the three subscales of the PBI for mothers only, and the two subscales of the RSQ. The paternal subscales of the PBI were omitted from further analyses as they have a weaker theoretical link with psychopathology than the maternal scales, and furthermore, they were not able to significantly differentiate between panic classifications according to the univariate tests conducted earlier.

A test of the full model with all six predictors against a constant-only model was found to be statistically reliable ( $\chi^2_{(6, N=195)} = 31.57, p < .001$ ), indicating that the predictors as a set reliably distinguished between Panic and No Panic groups. This model was highly successful at predicting classification to the 'no panic' group as it was able to correctly classify 94% of participants in this classification. That is, the vast majority of individuals who had no panic symptomatology were able to be identified as such, purely by assessing their attachment-related histories and current attachment experiences. By contrast, the six predictors used in the full model were able to correctly classify only 31% of individuals in the panic group. That is, only a

subset of people with panic disorder were able to be identified as such on the basis of their attachment-related histories and current attachment experiences. This finding actually provides support for the proposal that panic disorder is a heterogeneous condition with multiple possible developmental pathways. That is, these findings suggest that approximately a third of people with panic disorder report histories of childhood separation anxiety disorder, perceptions of aberrant parenting and an insecure attachment style. Furthermore, these findings suggest that attachment-relevant variables can reliably predict the development of panic disorder for approximately a third of sufferers.

An inspection of the predictors' individual contributions to the full model revealed that only childhood separation anxiety scores (SASI) and relationship anxiety scores reliably predicted panic disorder classification ( $z = 6.62, p = .01$ , and  $z = 4.99, p < .05$  respectively). In order to identify for possible interaction effects between separation anxiety and relationship anxiety in the prediction of panic disorder, a second logistic regression was conducted in which only the SASI scores, the RSQ- Relationship Anxiety subscale and an interaction coefficient between the two variables were entered as predictors. The interaction coefficient did not contribute significantly to the model ( $z = .528, p > .05$ ), indicating that separation anxiety and relationship anxiety each contributed unique predictive power to the classification of panic disordered and panic-free individuals.

Finally, a third logistic regression was conducted using only the separation anxiety and relationship anxiety scores as predictors, in order to determine whether fewer variables would be able to predict panic disorder classification with the same degree of success as the full model described above. In fact, this model, using the SASI and RSQ- relationship anxiety scores, improved the goodness-of-fit over the full

model by approximately 7 points as measured by the -2 Log-likelihood statistic, and reliably predicted panic disorder classification against a constant-only model ( $\chi^2_{(2, N=195)} = 24.75, p < .001$ ). Moreover, this model correctly classified 96% of individuals without panic, and 19% of panic sufferers. These findings indicate that the vast majority of people who have no panic symptomatology can be reliably classified as such on the basis of their history with childhood separation anxiety disorder, and their level of anxiety about abandonment in their current relationships. In addition, these findings support the proposition that a subset of individuals with panic disorder develop the condition along a developmental pathway characterised by childhood separation anxiety and insecurity about their current relationships.

### *5.5 Hypothesis 3: Panic frequency, attachment variables, and emotion regulation as mediator.*

According to Baron and Kenny (1986), a given variable is said to function as a mediator to the extent that it accounts for the relation between the predictor (independent variable) and the criterion (dependent variable). Baron and Kenny recommend using a specific set of regression equations in order to determine the relationships between the predictor, the criterion, and the potential mediating variable. This procedure was used to test this study's hypothesis that emotion regulation, with specific reference to the suppression of anger, mediates the relationship between attachment variables and panic frequency. As such, a series of four regression equations were conducted in which firstly anger suppression (anger in) and then panic frequency were entered as the dependent variables, and the attachment dimensions (i.e., relationship anxiety and avoidance) and anger suppression were entered, at first separately and then together, as the dependent variables (refer to Table 5 below). For

the purposes of this study, panic frequency was assessed according to the number of panic attacks reported by an individual in the month prior to completing the survey.

Table 5

*Regression analyses testing conditions for the mediation of attachment dimensions and panic frequency by anger suppression (N=88).*

Criterion variable	Predictor variable	R <sup>2</sup>	Beta	t value
(a) Anger – in	Avoidance	.324	.456	7.996***
	Relationship Anxiety		.213	3.728***
$F_{(2, 239)} = 57.21, p < .001$				
(b) Panic frequency	Anger – in	.091	.302	2.942**
$F_{(1, 86)} = 8.65, p < .01$				
(c) Panic frequency	Avoidance	.084	.112	1.008
	Relationship Anxiety		.230	2.067*
$F_{(2, 85)} = 3.91, p < .05$				
(d) Panic frequency	Avoidance	.120	.007	.059
	Relationship Anxiety		.181	1.605
	Anger-in		.230	1.848
$F_{(03, 84)} = 3.82, p < .05$				

\*\*\*  $p < .0001$ . \*\*  $p < .001$ . \*  $p < .05$

As shown in Table 5, the four conditions of mediation were successfully met. That is, (a) the attachment dimensions had a significant effect on anger suppression, suggesting that anger suppression could serve as a potential mediator for both avoidance of intimacy and anxiety about relationships; (b) ‘anger in’ significantly

predicted panic frequency, indicating that the suppression of anger does have an effect on the frequency of panic attacks; (c) attachment (with regard to the relationship anxiety dimension but not the avoidance dimension) was significantly related to panic frequency, indicating that anxieties about being insufficiently loved, or abandoned in relationships have a significant effect on the frequency of panic attacks, whereas avoidance of intimacy does not significantly contribute to panic frequency; and (d) when anger in and the attachment dimensions were simultaneously added to the regression equation, the previously significant association between relationship anxiety and panic frequency was no longer significant, indicating that the association between relationship anxiety and panic frequency is significantly mediated by anger suppression. These findings support the proposition that, individuals who feel anxious about the security of their relationship are more likely to have difficulty regulating feelings of anger towards their partner and are thus likely to suppress those angry feelings. As a result of the suppression of anger, the individual is more likely to experience a panic attack.

## Chapter 6: Discussion

This study aimed to explore the importance of attachment and attachment-relevant issues to panic disorder pathogenesis, while addressing an assumption of homogeneity of panic disorder inherent in previous literature. Specifically, it was proposed that individuals with panic disorder would be heterogeneous with respect to a history of childhood separation anxiety. In addition, it was anticipated that a definable subset of panic disorder sufferers would be reliably identifiable on the basis of their histories with childhood separation anxiety, aberrant parenting styles, and present-day insecure attachment styles. All three of the proposed hypotheses proposed were supported by the results of the study, although the second hypothesis regarding the influence of attachment-relevant variables on panic disorder development was only partially supported.

Specifically, the outcomes of the study supported the proposition that panic disorder was heterogeneous with respect to a history of childhood separation anxiety, as panic disorder sufferers were found to vary widely in their reports of childhood separation anxiety. Furthermore, childhood separation anxiety, along with anxiety about abandonment in current relationships (which was one of the assessed dimensions of an insecure attachment style) were reliably able to classify nonpanickers as not meeting panic disorder criteria, whilst correctly and reliably classifying a subset of panickers as meeting panic disorder criteria. By contrast, assessments amongst panic disorder sufferers of the avoidance of intimacy – the second assessed dimension of an insecure attachment style, and perceptions of aberrant parenting did not significantly contribute to the model for panic disorder prediction.

Nevertheless, the finding that individuals with panic disorder could be reliably identified on the basis of two attachment-relevant variables (i.e., separation anxiety and current relationship anxieties) was consistent with the proposal that panic disorder is an end-point disorder reached via multiple pathways, one of which involves anxieties about attachments, in past and present. Finally, for individuals who reported having had panic attacks, current anxieties about abandonment in relationships were significantly associated with panic frequency. This association, moreover, was significantly mediated by the tendency to suppress one's anger. The main findings of this study are discussed in greater detail below.

### *6.1 Hypothesis 1: Heterogeneity*

The outcomes of this study indicated that 22% (n= 52) of participants reported panic symptoms consistent with a lifetime self-reported diagnosis of panic disorder. An additional 19% (n= 45) of participants reported a subclinical panic disorder-related condition, involving limited symptom panic attacks and/or considerable avoidance behaviour. In total, 36% (n= 88) of participants reported having had recurrent panic attacks (either limited-symptom or full-blown) in their lifetime. This figure was consistent with findings from other studies using lifetime self-report measures for panic disorder (e.g., Norton, Dorward, & Cox, 1986; Watt & Stewart, 2000) which have revealed that up to a third of university students report a lifetime history of panic symptoms.

The Anxiety Sensitivity Index (ASI) was chosen as a measure in this study on the basis of its ability to discriminate panic disorder from other clinical populations. Mean scores across groups on the ASI provided concurrent validity for the self-report categories. For example, the mean ASI score for the panic group closely approached the lower end of the clinical range for panic disorder (i.e., 30.5 to 38.2) as reported by

Peterson and Reiss (1992), and was equivalent to the mean ASI score reported by Cox, Endler, and Swinson (1991) for “college students with frequent panic” (i.e.,  $M = 30.1$ ). On the other hand, the mean ASI scores for the subclinical panic and panic-free groups were significantly less than for the panic group, and more consistent with the normative mean ( $M = 19.1$ ) reported by Peterson and Reiss. Further support for the validity of the panic group comes from an examination of the normative ASI figures for people with social phobia, who also experience panic attacks, and report social and relationship anxieties. Nevertheless, the ASI literature (e.g., McNally, 1994; Peterson & Reiss, 1992) has indicated that patients with social phobia can be reliably distinguished from panic disorder patients on the basis of their mean ASI scores. Peterson and Reiss reported that patients with social phobia reported mean ASI scores (ranging from 21.4 to 24.9) that were significantly lower than those reported for panic disorder patients, both in general and in this study in particular. Furthermore, patients with simple phobia report even lower mean ASI scores than those with social phobia (e.g.,  $M = 20.0$ , Peterson & Reiss, 1992). These findings provide further validation that the self-reported panic group defined for the purposes of this study consisted primarily of panic disorder sufferers.

The findings of this study support the concept of heterogeneity of panic disorder aetiology, especially with reference to a developmental pathway involving childhood separation anxiety. Indeed, participants in this study who were classified as having PD exhibited a wide range of experiences of childhood separation anxiety symptoms, with low, moderate and high SASI scores all evident. Thus amongst individuals with PD, there appeared to be at least as many people for whom childhood separation anxiety was not relevant, as there were people for whom childhood separation anxiety was a significant part of their history. Furthermore, the logistic

regression analysis indicated that close to 20% of people classified with PD could be reliably identified according to a combination of childhood separation anxiety history, and anxiety over intimacy/ abandonment in current relationships.

As referred to earlier, Manicavasagar and colleagues also found heterogeneity with respect to separation anxiety amongst panic disorder patients. Indeed, for many of the participants in their research, the separation issues they experienced were so central to their psychopathology as to lead Manicavasagar et al. to propose that these patients might be better characterised as having adult separation anxiety as their primary diagnosis, with panic disorder as a secondary complication. This description may well be appropriate for the subset of participants identified in this study, whose panic disorder symptomatology was strongly associated with a history of severe childhood separation anxiety symptoms, and present-day anxieties about abandonment in relationships. These findings have important implications for the ways we approach the treatment of panic disorder in clinical settings. While only a minority, albeit a large minority, of panic disordered participants in this study (i.e., 20%) reported significant levels of separation anxiety, the work of Manicavasagar et al. suggests that the subset of sufferers who experience separation-related panic disorder in clinical samples, versus community samples, of PD patients might be as high as 75%. Given these substantial figures, it is essential that treatment programs properly assess panic disorder patients in order that the underlying psychopathology of these patients can be adequately addressed.

The findings of this study strongly support the idea of heterogeneity amongst panic disorder sufferers in that, a proportion of panic disorder cases, but certainly not all of them, were found to develop in association with significant levels of unresolved childhood separation anxiety. This means that, the remaining participants who were

classified as having panic disorder developed the condition by a pathway that was independent of attachment-related problems such as separation anxiety. Indeed, it might well be possible that this large group of nonattachment-related panic disorder could be further classified into subgroups. For example, some panic sufferers might have arrived at a diagnosis through frightening experiences with restricted breathing such as asthma or pulmonary disease, whereas others might present with a neurophysiological dysfunction.

It is possible that the separation anxiety pathway to panic psychopathology has not emerged convincingly or consistently in past literature because it has been obscured by the presence of heterogeneity in panic aetiology. That is, whereas clinicians who work therapeutically with PD patients have been repeatedly struck by the profound difficulties that these patients have with issues of separation and dependence, researchers have insisted that, unless systematic testing found these issues to be common to all or most PD patients, they could not be considered theoretically or statistically significant for panic disorder development. On the one hand, this is a fair approach to clinical research given that clinicians are subject to biases, both with respect to his/her own beliefs about the development of psychopathology, but more significantly, as a result of the fact that clinicians predominantly work with the most severe and complex of psychiatric cases, rather than a representative mix of a patient population. However, although clinical impressions cannot be accepted at face value as relevant to all patients, they should not be easily dismissed, as clinicians are in a position to appreciate the subtleties and full complexities of a person's psychopathology. The proposition of this study that PD is heterogeneous –that is, made up of subsets of patients whose pathology developed along qualitatively different paths – makes sense of the inconsistencies

reported by clinicians and researchers, indicating that the conclusions of both views have merit. That is, it is possible that the most complex cases of PD as seen by clinicians may be overrepresented by cases of separation-relevant panic disorder, as these patients may be more motivated (or encouraged by others) to seek help as a result of their interpersonal difficulties, and possibly their greater levels of agoraphobic avoidance. However, the findings of this study and others investigating the relationship between separation anxiety and panic disorder indicate that this group of patients (i.e., those with separation-relevant PD) is not representative of the PD population as a whole but actually equates to a distinct subset of the condition. As such, the finding of this study that PD is not homogeneous indicates that we need to modify our research approach, so that we are in a position to identify the features of the condition that best characterise the various possible pathways of panic disorder pathogenesis.

## 6.2 *Hypothesis 2: Attachment and Panic Disorder*

The importance of attachment-related variables in panic disorder pathogenesis was highlighted by the findings from this study. Specifically, this study examined the influence of separation anxiety, perceptions of early parental behaviours, and current attachment style in panic disorder. As mentioned, logistic regression found that nonpanickers and a subgroup of panickers (approximately 20%) could be reliably identified by a combination of childhood separation anxiety symptoms and current anxieties about intimacy and/or abandonment in relationships. Overall, these findings meant that, for the participants of this study, a given individual exhibiting significant childhood separation anxiety, and significant relationship anxiety may or may not have also had panic disorder. By contrast, the probability of an individual with non-

significant levels of both childhood separation anxiety and relationship anxiety also having panic disorder was very, very low.

As mentioned, one of the attachment-relevant variables hypothesised to be associated with panic disorder development was participants' retrospective perceptions of parental warmth, overprotectiveness, and authoritarianism as assessed by the Parental Bonding Inventory (PBI). As anticipated, participants who had been classified as panic disordered generally viewed both their parents as less warm, more overprotective and more authoritarian than panic-free participants. Only perceptions of maternal warmth however were able to significantly differentiate between the panic and panic-free groups. Even so, neither the maternal warmth factor nor the other two maternal parenting styles (i.e., overprotectiveness and authoritarianism) improved the model for panic disorder prediction over and above predictions based on separation anxiety and relationship anxiety scores. Indeed, given Bowlby's emphasis on the importance of the parental contribution to the development of severe separation anxiety in the child, one would have expected separation anxiety in particular to be more closely related to parenting styles – especially with respect to maternal parenting styles given that it is typically the mother who plays the role of primary caregiver. In fact, childhood separation anxiety and maternal overprotection were only weakly, albeit significantly, correlated. Furthermore, maternal warmth and authoritarianism were not significantly correlated with separation anxiety at all. It is interesting to note however that, whereas separation anxiety and maternal warmth exhibited no significant correlations, relationship anxiety and maternal warmth were significantly, albeit weakly correlated. Furthermore, as for separation anxiety, relationship anxiety and maternal overprotection were significantly but weakly correlated, whereas maternal authoritarianism was not significantly correlated at all. Therefore, it should

be noted that early parental behaviours, particularly in the form of maternal overprotection, were indeed significantly, although not strongly, associated with both the experience of childhood separation anxiety and current relationship anxieties, as Bowlby had predicted.

Nevertheless, the findings that the correlations between early maternal behaviours and subsequent separation and/or relationship anxiety were relatively weak is surprising. Equally unexpected is the finding that early maternal behaviours did not contribute significantly to the prediction of panic amongst individuals with separation-relevant panic disorder. As such, these outcomes may reflect a number of possibilities. Firstly pathological childhood separation anxiety may not be as closely linked to perceived parenting styles as widely believed. Alternatively, the Parental Bonding Inventory may not be sufficiently sensitive or adequate to detect those actual elements of parenting most relevant to the exacerbation of separation anxiety symptoms in the child. Finally, it is possible that statistical redundancy or shared variance between scores on the Separation Anxiety Symptom Inventory (used to assess childhood separation anxiety) and the PBI, and/or scores on the Relationship Scales Questionnaire (used to assess attachment) and the PBI obscured the unique contribution of parental behaviours to the predictive equation.

Attachment style was measured along two dimensions, namely avoidance and relationship anxiety. 'Avoidance' provided information about an individual's preference for closeness and intimacy over distance and independence. 'Relationship anxiety' on the other hand indicated an individual's degree of worry about their close relationships. The content of worries included being alone, being abandoned by a significant other, and not perceiving oneself to be sufficiently valued or loved by a significant other. Individuals high on 'avoidance' would be said to belong to either

the dismissing or fearful adult attachment classifications; whereas those high on relationship anxiety might belong to either the preoccupied or fearful attachment style classifications.

In this study, only relationship anxiety and not avoidance contributed significantly to a reliable model for classifying panic disorder. That relationship anxiety was able to significantly predict panic disorder development, is consistent with the idea that individuals with panic disorder are sensitive to issues of separation and abandonment by loved ones (e.g., Guidano, 1987; Liotti, 1991; Shear, 1996). Nevertheless, this finding applied to only a proportion, albeit a considerable proportion, of panic sufferers, indicating that the proposition that people with panic disorder are sensitive to issues of separation cannot be uniformly applied to the group as a whole. The finding that the avoidance of intimacy did not significantly contribute to the model predicting panic disorder for a proportion of sufferers suggests that individuals who present with separation-related panic disorder do not have a specific preference for either intimacy and closeness or independence and distance. When taken together with the finding that individuals with separation-relevant panic disorder displayed high levels of relationship anxiety, this outcome means that these individuals reported a mixture of preoccupied and fearful attachment styles (which respectively represent preferences for low and high avoidance of intimacy).

The finding of this study that panic sufferers were not significantly characterised by the avoidance of intimacy, is somewhat inconsistent with proposals by such authors as Bowlby (1973) and de Ruiter and van Ijzendoorn (1992). These authors have proposed that an anxious-ambivalent working model of attachment relationships (which corresponds with Bartholomew's (1990) 'preoccupied' attachment classification) specifically predisposes individuals to panic disorder/

agoraphobia. Nevertheless, any inconsistency between this theoretical prediction and the findings of this study may be more a reflection of the limitations of the measures used by this study to assess attachment, than an indication of non-support for a specific link between attachment and panic disorder. In particular, it should be noted that this study assessed attachment dimensions (specifically, relationship anxiety and avoidance), and from these dimensions, inferences have been made about attachment style (whether secure, preoccupied, detached, or fearful). These two concepts are not equivalent. In addition, the two attachment dimensions measured by the Relationship Style Questionnaire may not adequately represent the attachment styles/classifications as perceived by Bowlby or de Ruiter and van Ijzendoorn. Other, more sophisticated instruments may have more success in capturing and representing the attachment styles.

Indeed, Strodl and Noller (2003) found support for the proposition of a specific link between a preoccupied attachment style and the development of panic/agoraphobia using a more sophisticated assessment instrument with respect to its ability to assess multiple attachment dimensions. Specifically, while Bartholomew's Relationship Style Questionnaire assesses attachment style along two dimensions, such that a preoccupied attachment style is proposed to reflect a high level of desire for intimacy and a high level of anxiety about relationships; Feeney, Noller, and Hanrahan's (1994) Attachment Style Questionnaire (ASQ) assesses five dimensions – four of which could be considered as insecure and one as secure. The secure dimension is labelled confidence. The insecure dimensions are referred to as discomfort with closeness (theoretically equivalent to 'avoidance'), need for approval, preoccupation with relationships (theoretically equivalent to 'relationship anxiety'), and relationships as secondary. Using the ASQ, Strodl and Noller (2003) found that,

as they had predicted, only the preoccupation with relationships scale, and not the other three insecure dimensions of the ASQ, was uniquely associated with agoraphobic behaviour. Thus, the findings of this study serve to highlight the limitations of Bartholomew's bidimensional assessment tool, whilst providing tentative support for the proposition that particular aspects of insecure attachment styles (i.e., anxieties about abandonment and being sufficiently valued in relationships) may be specifically associated with panic disorder psychopathology.

It is interesting to note that the interaction coefficient of the childhood separation anxiety measure and the relationship anxiety measure did not contribute significantly to the predictive model. Essentially this meant that each of the variables contributed uniquely to panic prediction and were not simply measuring the same concept from different perspectives. However, this finding still allows for the possibility that high relationship anxiety in adulthood is an extension of childhood separation anxiety. As such, the results of this study are also consistent with Manicavasagar et al.'s views that individuals who experience significant and functionally disruptive concerns about separation from their significant others would be most appropriately diagnosed with adult separation anxiety disorder.

Manicavasagar et al. have proposed that these individuals, rather than representing a subset of panic disorder cases as suggested by this thesis, actually represent a distinct diagnostic category that is highly comorbid with panic disorder. As this study did not assess individuals specifically for adult separation anxiety disorder, its findings cannot differentiate between these two subtly different conclusions. Nevertheless, both proposals have similar clinical implications with regard to the need for more careful assessment of panic disorder patients in order to assess for the presence of clinically

significant separation issues, combined with the need to address these issues in treatment.

The outcomes of this study indicated that a subset of panic disorder sufferers could be identified on the basis of specific attachment-related variables. These variables included a history of childhood separation anxiety and present-day relationship anxieties. One implication of the finding that a group of PD sufferers can be characterised by separation/ attachment issues, is that panic disorder is an endpoint condition reached via multiple pathways, one of which involves developmental preconditions in the realm of attachment relationships. This is the interpretation favoured by the researcher. Nevertheless, alternative explanations may apply. For example, the individuals identified herein with separation-related panic disorder, could be said to actually suffer from an underlying predispositional sensitivity to anxiety such as behavioural inhibition or neuroticism that might be equally associated with panic disorder development as with separation anxiety and relationship anxiety. That is, in contrast to the proposition of this thesis that panic disorder is likely to be the result of a relatively linear aetiological pathway leading from SA to relationship anxiety and then to panic; separation anxiety, relationship anxiety and panic disorder might be said to all arise in response to a common underlying sensitivity. Whilst, a predispositional-based interpretation is certainly plausible, and cannot be disconfirmed by the present study, research into temperament and attachment (as discussed in Chapter 2) contraindicates such a proposition. That is, attachment research has found that both temperament and parental sensitivity interact to produce an insecure attachment. Thus, in cases where an anxious temperament persists into childhood and beyond, the lack of goodness-of-fit of the infant-mother unit is as likely to be implicated as any genetic predisposition towards anxiety. Indeed, a lack of good

fit between the mother's ability to accommodate and soothe her infant's frustrations/ anxieties has been demonstrated to have ramifications for later interpersonal functioning (e.g., Crockenberg, 1981; Suomi, 1997). As such, the findings of the present study are generally consistent with the proposition that early maternal behaviours play an important role in the subsequent development of childhood separation anxiety, regardless of infant temperament. That is, this study found that retrospective perceptions of maternal warmth significantly differentiated between the self-reported panic disordered and panic-free groups, while the other parenting styles (overprotection and authoritarianism) generally exhibited trends in the expected directions. In all, the results of this study provide a strong case for the need to assess attachment issues, past and present, in all diagnostic interviews for panic disorder.

### 6.3 *Hypothesis 3: Emotion Regulation and Panic Disorder*

The suppression of anger differentiated between groups with and without panic disorder such that panic sufferers suppressed anger more frequently, whereas they expressed and/ or controlled anger at about the same rate. The frequent suppression of anger was also associated with an elevated rate of panic attacks. Interestingly, the sample as a whole demonstrated higher levels of anger suppression than reported by Spielberg's (1988) normative population. This finding was likely to be a relic of the high proportion of women in this study's sample. Although Spielberg found that gender did not have a significant effect on the 'anger in' scores of his normative population, Jack (1991) has found that the non-expression of anger is strongly embedded in cultural norms, especially amongst women. Moreover, anger suppression amongst women was found to be significantly related to psychopathology, especially depression.

It was predicted that anger regulation would be particularly problematic in the context of insecurity about relationships, in which individuals might feel that their relationship was too fragile or unsafe to withstand the expression of anger and discontent. As such, difficulties expressing negative attachment-related emotions such as anger, might predispose the individual to experience panic attacks. Indeed, this study found support for these predictions in that one of the dimensions of attachment style correlated significantly with panic frequency. Moreover, the suppression of anger was found to significantly mediate the association between relationship anxiety and panic frequency. This meant that the more insecure an individual was in his/her attachment relationship (as assessed by the extent of a person's anxieties about abandonment in relationships), the more likely he/she would be to suppress anger, and the more likely he/she would be to experience higher rates of panic.

The outcomes of this study with respect to emotion regulation and attachment provide support for Guidano's model of panic disorder pathogenesis. Guidano (1987) and Liotti (1991) proposed that individuals with panic disorder would be especially sensitive to issues of separation and overprotection, experiencing the former as abandonment and the latter as suffocation. In response they would develop a tendency to overcontrol their relationships, emotions and behaviours in order to minimize their discomfort and/or exposure to separation experiences. As a consequence of this overcontrol it was suggested that panic sufferers would experience negative attachment-related emotions, such as anger and anxiety, even more frequently than others as these emotions would be easily evoked. In addition, Guidano proposed that panic sufferers experienced negative emotions in a primitively somatic way that was dissociated from complex cognitive processes of interpersonal

cause and effect. In response to these bodily sensations, the panic sufferer would react by catastrophically misinterpreting them as a sign of impending illness or disaster, and the increased anxiety levels and concomitant bodily sensations would then feed off each other, escalating into a panic attack. Although Guidano's theory has undergone little empirical testing, the findings of Strodl and Noller (2003) have provided tentative support for his theory of panic disorder pathogenesis. Strodl and Noller found that preoccupation with relationships (equivalent to the 'relationship anxiety' variable used in this study) was significantly associated with agoraphobic behaviour, and this relationship was partly mediated by the extent to which the individual experienced catastrophic cognitions about bodily sensations. Given that the mediating relationship between relationship anxiety and agoraphobic behaviours was only partial, Strodl and Noller concluded that future research needed to examine other potential mediators. As such, the results of the present thesis provide additional support for Guidano's theory of panic disorder pathogenesis. Furthermore, the findings of this study strongly indicate that in addition to the tendency to catastrophise in response to anxiety symptoms, the suppression of anger is an additional and significant mediating influence on the relationship between insecure attachment and panic disorder pathogenesis.

The finding that an insecure attachment, in terms of an elevated level of anxiety about the security of one's relationship, is associated with more anger suppression, which is in turn associated with a greater frequency of panic attacks, is a novel and exciting discovery for the field of panic disorder. These findings highlight and confirm the propositions at the centre of this thesis, that panic disorder is a syndrome which consists of more than its manifest symptomatology of panic attacks and the fear of panic attacks (i.e., anticipatory anxiety). As such, panic disorder

cannot be viewed as a syndrome that can randomly strike otherwise healthy individuals. Instead, panic disorder develops, for a considerable proportion of sufferers, as a consequence of emerging vulnerabilities that arise in response to an individual's history of relationships. In particular, an individual's attachment history ultimately governs the individual's preferences for specific emotion regulation strategies, which may either exacerbate or ameliorate the development of psychopathology.

#### *6.4 Implications*

The findings of this study have significant implications for both theoretical and clinical approaches to panic disorder. The finding that panic disorder is heterogeneous with respect to a history of childhood separation anxiety supports the proposition that panic disorder is characterised by a number of possible developmental pathways. These pathways may be qualitatively very distinct, despite all ending at the same ultimate destination manifested by panic attacks, anticipatory anxiety, and possible avoidance behaviours— that is, panic disorder. For example, this study supports the proposition that panic disorder is heterogeneous with respect to a history of childhood separation anxiety disorder, in that a distinct proportion of panic sufferers clearly demonstrate such histories. Other studies have implicated possible physiological dysfunctions in a proportion of panic disorder patients, or frightening experiences with restricted breathing as occurs with asthma. These may represent separate and alternative pathways for the development of panic disorder. In addition, anxiety sensitivity has also been strongly implicated as a developmental precondition of panic disorder, and further research might be able to elaborate as to whether anxiety sensitivity is a specific marker on the pathway to separation anxiety-relevant panic disorder as Guidano has proposed, or whether it is common to a few

alternative pathways. The importance of clearly establishing the mechanisms for each of the possible developmental pathways to panic disorder relates to the need to find more effective clinical approaches for the disorder.

The finding of this thesis that panic disorder is heterogeneous, along with evidence from longitudinal studies (e.g., Noyes et al., 1990; Pollack & Marzol, 2000) that indicate that panic disorder is often chronic, clearly signifies that our current clinical approaches to panic disorder are inadequate. That is, the application of a uniform psychopharmacologic or cognitive-behavioural treatment to every individual diagnosed with the condition is inappropriate and must be rethought. Instead, the findings of this study suggest that patients with panic disorder must be more carefully assessed in order to ascertain the most appropriate and effective focus of intervention for the particular case at hand.

This thesis has identified that issues of separation and abandonment in relationships may prove to be fruitful clinical foci for individuals with panic disorder who arrived at their condition via a developmental pathway characterised by childhood separation anxiety and current relationship anxieties. In particular, the findings of this research suggest that these individuals may have difficulties expressing anger or discontent in their relationships for fear of abandonment. This fear may relate to psychological abandonment as much as to physical abandonment, in that the panic sufferer may fear the partner's emotional withdrawal in the event of conflict. As a consequence the panic sufferer may try to avoid conflict by suppressing their anger, however this technique is likely to lead to the frequent evocation of anger and anxiety, as the issues which have generated these feelings cannot be resolved by the panic sufferer alone. Indeed the findings of this study indicated that the suppression of anger was significantly associated with an increased frequency of

panic attacks for individuals who were insecure in their relationships. Actually, panic sufferers' avoidance of conflict may well serve to increase their anxieties about separation and abandonment, as the opportunities for the patient to experience interpersonal conflict and thus to know that it can be survived and even resolved will be severely reduced.

It is interesting to note that the proposition that people with panic disorder may avoid interpersonal conflict is consistent with the general avoidant approach to panic attacks that is characteristic of agoraphobic behaviour. Indeed it remains to be seen whether individuals with separation-relevant panic disorder display higher levels of agoraphobic behaviour or symptom severity than other panic disorder subgroups. Further research is required to explore these propositions, and to establish other possible markers of separation-relevant panic disorder.

As such, the findings of this thesis suggest that panic disorder needs to be more carefully assessed in clinical settings in order to identify the subtype with which a particular patient presents. This information should then inform decisions about which of the available treatment interventions would be most appropriate and effective for that particular patient. Whereas medication and cognitive-behavioural treatments have been successful for treating the manifest symptoms of panic disorder in many patients, there may well be a subgroup for whom underlying issues of separation and abandonment may act to maintain their psychopathology despite these first-line treatments. For this panic disorder subgroup, an additional psychotherapeutic focus on relationships and interpersonal conflict may help to further ameliorate or perhaps even cure the condition. Research investigating the effectiveness of such a clinical intervention for this subgroup, over and above the standard treatment program, is required to assess this proposition.

### 6.5 *Limitations*

The findings of this study must be qualified by a few limitations of the research. Firstly, although this thesis has drawn inferences from its findings that a proportion of panic disorder cases arise along a developmental continuum involving childhood separation anxiety disorder and present-day insecure attachment relationships, the cross-sectional and correlational nature of this study does not really allow for conclusions to be drawn about causality. Despite this limitation, the fact that this study's results clearly support associations between attachment and panic disorder, and in light of the theoretical considerations of attachment discussed earlier in this thesis, we believe that it makes sense to propose a causal influence.

Nevertheless, longitudinal studies are necessary to conclusively determine the direction of the associations between attachment, childhood separation anxiety and separation-relevant panic disorder. In addition, a longitudinal study would serve to address the limitations posed by the use of measures in this study that were based on retrospective recall, specifically the Separation Anxiety Symptom Inventory and Parental Bonding Instrument.

A limitation of this study was that it did not include other anxiety or affective disorders for comparative purposes, thus preventing any conclusions being drawn about whether a developmental pathway characterised by separation issues is specifically related to the development of separation-relevant panic disorder or is more generally associated with the development of psychopathology. Nevertheless, the findings of this study indicate that further analysis is indeed warranted. In addition to providing information about causality, longitudinal studies would be able to provide information about the specificity of the link between childhood separation anxiety, current relationship anxieties and separation-relevant panic disorder.

This study was based on a non-clinical convenience sample of university students. Despite the fact that mean scores on the Anxiety Sensitivity Index for the self-reported panic disorder group offered some concurrent validity that this group was indeed characterised by panic disorder sufferers, it would be useful to replicate this study using a clinical sample of panic disorder patients, in order to further substantiate its findings and ensure that they are generalisable to other panic-disordered populations.

### 6.6 *Conclusions*

Attachment theory has been demonstrated to be a useful framework for understanding the ways in which psychopathology can emerge from early relationships that are characterised by a sense of insecurity, conferred to infants by their mother's unpredictable responsiveness and insensitivity to their needs. This early relationship has consequences for individuals in terms of their developing sense of confidence that they can cope with the normal developmental challenges encountered in life, especially with respect to their internal emotional and interpersonal conflicts. That is, attachment theory holds that early relationships provide a template for the internal representations of both the self and significant others, and also for the regulation of emotions, either alone or with the help of others.

The findings of this study supported the proposition that early attachment plays a significant role in the development of psychopathology. Indeed this study found that for some people with panic disorder, the condition was strongly characterised by attachment issues. Specifically, a proportion of panic disorder cases was able to be reliably predicted by the combination of a history of childhood separation anxiety and current relationship anxieties about abandonment and intimacy (which is one aspect of an insecure adult attachment). Furthermore, a person's

internal representations of interpersonal relationships was found to contribute to the etiology or exacerbation of panic disorder psychopathology, such that anxieties about abandonment and intimacy in current relationship were found to be associated with an increased frequency of panic attacks. In addition, anger suppression was found to mediate the association between relationship anxiety and panic. This result supports the idea, consistent with attachment theory, that the manner in which an individual regulates and expresses his/ her emotions in relationships can help to explain how psychopathology emerges from attachment.

However, although psychopathology is often associated with an insecure attachment, an insecure attachment status is not sufficient to explain all psychopathology. As such this study found that panic disorder was heterogeneous with respect to a history of childhood separation anxiety. This means that while a significant minority of panic sufferers in this study (i.e., 20%) reported separation and attachment difficulties, the majority did not. Studies using clinical samples of PD patients (e.g., Manicavasagar, Silove, & Curtis, 1997) suggest that cases of separation-relevant panic disorder might be over-represented in clinical populations such that up to 75% of PD patients might demonstrate clinically significant difficulties with separation and dependence in their interpersonal relationships. Thus, the findings of this study have critical implications for the clinical assessment and treatment of patients with panic disorder. That is, this patient group needs to be carefully assessed for the underlying issues that may have initiated and may be maintaining their pathology, rather than assessed simply on the basis of their manifest panic symptomatology. This assessment would then need to inform the treatment plan such that these issues are addressed in order to achieve optimum long-term intervention outcomes for the patient. Similarly, research efforts should focus on

defining other possible subtypes of panic disorder pathogenesis so that markers for each can be identified and used to inform assessment procedures to ensure maximum efficiency in the diagnostic processes. Research programs could also assess the effectiveness of specific treatment programs for specific subtypes of panic disorder to ensure that patients receive the benefits of the most effective evidence-based treatments. The use of more effective treatment practices for patients with panic disorder would fundamentally serve to improve the ongoing quality of life for these patients, and to ultimately conserve the resources of medical and clinical services in the long-term.

## References

Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/ 4 - 18 and 1991 Profile*. Burlington: University of Vermont.

Ainsworth, M.D.S. (1973). The development of infant - mother attachment. In B.N. Caldwell & H. N. Ricciuti (Eds.), *Review of child development research* (pp. 1 - 94). Chicago: University of Chicago Press.

Ainsworth, M.D., Bell, S.M., & Stayton, D.J. (1972). Individual differences in the development of some attachment behaviors. *Merrill - Palmer Quarterly*, 18(2), 123 - 143.

Ainsworth, M. D. S., Blehar, M.C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation*. Hillsdale, NJ: Erlbaum.

Ainsworth, M. D. S., & Wittig, B. A. (1969). Attachment and Exploratory Behaviour of One - year - olds in a Strange Situation. In B. M. Foss (Ed.), *Determinants of infant behavior* (Vol. 4). London: Methuen.

Alessi, N. E., & Magen, J. (1988). Panic disorder in psychiatrically hospitalized children. *American Journal of Psychiatry*, 145(11), 1450 - 1452.

Alessi, N. E., Robbins, D. R., & Dilsaver, S. C. (1987). Panic and depressive disorders among psychiatrically hospitalized adolescents. *Psychiatry Research*, 20, 275 - 283.

Almond, R. (1997). Psychodynamic therapies. In W. T. Roth (Ed.), *Treating anxiety disorders*. San Francisco, CA: Jossey - Bass.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3<sup>rd</sup> ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3<sup>rd</sup> ed., Rev.). Washington, DC: Author.

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4<sup>th</sup> ed.). Washington, DC: Author.
- Anderson, J. C., Williams, S., McGee, R., & Silva, P. A. (1987). DSM-III disorders in preadolescent children: Prevalence in a large sample from the general population. *Archives of General Psychiatry*, 44, 69 - 76.
- Aronson, T. A., & Logue, C. M. (1987). On the longitudinal course of panic disorder: Developmental history and predictors of phobic complications. *Comprehensive Psychiatry*, 28(4), 344 - 355.
- Aschenbrand, S. G., Kendall, P. C., Webb, A., Safford, S. M., & Flannery - Schroeder, E (2003). Is childhood separation anxiety disorder a predictor of adult panic disorder and agoraphobia? A seven - year longitudinal study. *Journal of the American Academy of Child and Adolescent Psychiatry* 42(12), 1478 - 1485.
- Asmundson, G. J. G., & Stein, M. B. (1994). Triggering the false suffocation alarm in panic disorder patients by using a voluntary breathholding procedure. *American Journal of Psychiatry*, 151, 264 - 266.
- Austin, David W., & Richards, Jeffrey C. (2001). The catastrophic misinterpretation model of panic disorder. *Behaviour, Research and Therapy*, 39(11), 1277 - 1291.
- Ballenger, J. C., Carek, D. J., Steele, J. J., & Cornish - McTighe, D. (1989). Three cases of panic disorder with agoraphobia in children. *American Journal of Psychiatry*, 146, 922 - 924.
- Barlow, D. H. (1986). In defense of panic disorder with agoraphobia and the behavioral treatment of panic: A comment on Kleiner. *Behavior Therapist*, 5, 99 - 100.
- Barlow, D. H. (1988). *Anxiety and its disorders*. New York: Guilford Press

Baron, R. M. and Kenny, D. A. (1986). The moderator - mediator variable distinction in social psychological research: Conceptual, strategic and statistical consideration. *Journal of Personality and Social Psychology*, 51(6), 1173 - 1182.

Bartholomew, K. (1990). Avoidance of intimacy: An attachment perspective. *Journal of Social and Personal Relationships*, 7, 147 - 178.

Bartholomew, K., & Horowitz, L. (1991). Attachment styles among young adults: A test of a four category model. *Journal of Personality and Social Psychology*, 61, 226 - 224.

Battaglia, M., Bago, S., Strambi, L.F., Brambilla, F., Castronovo, C., Vanni, G., & Bellodi, L. (1997). Psychological and behavioral responses to minor stressors in offspring of patients with panic disorder. *Journal of Psychiatric Research*, 31, 365 - 376.

Bell, S. M., & Ainsworth, M. D. (1972). Infant crying and maternal responsiveness. *Child Development*, 43(4), 1171 - 1190.

Belsky, J., Fish, M., & Isabella, R. (1991). Continuity and discontinuity in infant negative and positive emotionality: Family antecedent and attachment consequences. *Developmental Psychology*, 27, 421 - 431.

Belsky, J., & Nezworsky, T. (1988). *Clinical implications of attachment*. Hillsdale, NJ: Erlbaum.

Belsky, J., Spritz, B., & Crnic, K. (1996). Infant attachment security and affective - cognitive information processing at age 3. *Psychological Science*, 7, 111 - 114.

Berg, I. (1976). School phobia in the children of agoraphobic women. *British Journal of Psychiatry*, 128, 86 - 89.

Berg - Nielsen, T. S., Vikan, A., & Dahl, A. A. (2002). Parenting related to child and parental psychopathology: A descriptive review of the literature. *Clinical Child Psychology & Psychiatry*, 7(4), 529 - 552.

Biederman, J., Faraone, S. V., Marris, A., Moore, P., Garcia, J., Ablon, S., Mick, E., Gershon, J., & Kearns, M. E. (1997). Panic disorder and agoraphobia in consecutively referred children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 214 – 223.

Biederman, J., Hirshfeld - Becker, D.R., Rosenbaum, J.F., Herot, C., Friedman, D., Snidman, N., Kagan, J., & Faraone, S.V. (2001). Further evidence of association between behavioral inhibition and social anxiety in children. *American Journal of Psychiatry*, 158, 1673 - 1679.

Biederman, J., Rosenbaum, J.F., Bolduc - Murphy, E.A., Faraone, S.V., Chaloff, J., Hirshfeld, D.R., & Kagan, J. (1993). A 3 - year follow - up of children with and without behavioral inhibition. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 814 - 821.

Biederman, J., Rosenbaum, J.F., Hirshfeld, D.R., Faraone, S.V., Bokluc, E.A., Gersten, M., Meninger, S.R., Kagan, J., Snidman, N., & Reznick, J.S. (1990). Psychiatric correlates of behavioural inhibition in young children of parents with and without psychiatric disorders. *Archives of General Psychiatry*, 47, 21 - 26.

Black, B., & Robbins, D. R. (1990). Case study: Panic disorder in children and adolescents. *Journal of the American Academy of Child and Adolescents Psychiatry*, 29, 36 - 44.

Blais, M.A., Otto, M.W., Zucher, B.G., McNally, R.J., Schmidt, N.B., Fava, M., & Pollack, M.H. (2001). The Anxiety Sensitivity Index: Item Analysis and suggestions for refinement. *Journal of Personality Assessment*, 77, 272 - 294.

Bland, K., & Hallam, R. (1981). Relationship between response to graded exposure and marital satisfaction in agoraphobics. *Behaviour Research & Therapy*, 19, 335 – 338.

Blehar, M.C., Lieberman, A.F., Ainsworth, M.D. (1977). Early face-to-face interaction and its relation to later infant-mother attachment. *Child Development*, 48(1), 182 -194.

Bonn, J. A., Readhead, C. P. A., & Timmons, B. H. (1984). Enhanced adaptive behavioural response in agoraphobic patients pretreated with breathing retraining. *Lancet*, *2*, 665 - 669.

Bouton, M. E., Mineka, S., & Barlow, D. H. (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychology Review*, *108*(1), 4 - 32.

Bowlby, J. (1969). *Attachment and loss, Vol. 1, Attachment*. New York: Basic Books.

Bowlby, J. (1973). *Attachment and loss, Vol. 2, Separation anxiety and anger*. New York: Basic Books.

Bowlby, J. (1980). *Attachment and loss, Vol. 3, Loss*. New York: Basic Books.

Bowlby, J. (1988). Developmental psychiatry comes of age. *American Journal of Psychiatry*, *145*, 1 - 10.

Bowlby, J., Robertson, J., & Rosenbluth, D. (1952). A two-year-old goes to the hospital. *Psychoanalytic Study of the Child*, *7*, 82 - 94.

Brehony, K. A., & Geller, E. S. (1981). Agoraphobia: Appraisal of research and a proposal for an integrated model. In M. Hersen, R. M. Eisler, & P. M. Miller (Eds.), *Progress in behavior modification* (Vol. 12). New York: Academic Press.

Breier, A., Charney, D. S., & Heninger, G. R. (1986). Agoraphobia with panic attacks: Development, diagnostic stability, and course of illness. *Archives of General Psychiatry*, *43*, 1029 - 1036.

Brennan, K. A., Clark, C.L., & Shaver, P.R. (1998). Self - report measurement of adult attachment: An integrative overview. In J.A. Simpson & W.S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 46 - 76). New York: Guilford Press.

Brennan, K. A., & Shaver, P. R. (1995). Dimensions of adult attachment, affect regulation, and romantic relationship functioning. *Personality & Social Psychology Bulletin*, *21*, 267 - 283.

Brennan, K. A., & Shaver, P. R. (1998). Attachment Styles and Personality Disorders: Their Connections to Each Other and to Parental Divorce, Parental Death, and Perceptions of Parental Caregiving. *Journal of Personality*, 66(5), 835 - 878.

Bretherton, I. (1985). Attachment Theory: retrospect and prospect. In I. Bretherton & E. Waters (Eds.), *Growing points of attachment theory and research. Monographs of the Society for Research in Child Development*, 50, 3 - 35.

Bretherton, I. (1991). Roots and growing points of attachment theory. In C. M. Parkes, J. Stevenson - Hinde & P. Marris (Eds.), *Attachment across the life cycle*. London: Routledge.

Brooks, R.B., Baltazar, P. L., & Munjack, D. J.. (1989). Co-occurrence of personality disorders with panic disorder, social phobia, and generalized anxiety disorder: A review of the literature. *Journal of Anxiety Disorders*, 3, 259 - 285.

Brown, G. W., & Harris, T. O. (1993). Aetiology of anxiety and depressive disorders in an inner city population: 1. Early adversity. *Psychological Medicine*, 23, 143 - 154.

Buglass, D., Clarke, J., Henderson, A. S., Kreitman, N., & Presley, A. S. (1977). A study of agoraphobic housewives. *Psychological Medicine*, 7, 73 - 86.

Busch, F.N., Cooper, A.M., Klerman, G.L., Penzer, R.J., Shapiro, T., & Shear, M.K. (1991). Neurophysiological, cognitive - behavioural, and psychoanalytic approaches to Panic Disorder: Toward an integration. *Psychoanalytic Inquiry*, 11, 316 - 332.

Carlson, E. A. (1998). A prospective longitudinal study of attachment disorganization/disorientation. *Child Development*, 69(4), 1107 - 1128.

Carnelley, K. B., Pietromonaco, P.R., & Jaffe, K. (1994). Depression, working models of others, and relationship functioning. *Journal of Personality and Social Psychology*, 66, 127 - 140.

Carter, M. M., Hollon, S. D., Carson, R., & Shelton, R. C. (1995). Effects of a safe person on induced distress following a biological challenge in panic disorder with agoraphobia. *Journal of Abnormal Psychology, 104*(1), 156 - 163.

Carter, M.M., Suchday, S., & Gore, K.L. (2001). The utility of the ASI factors in predicting response to voluntary hyperventilation among nonclinical participants. *Journal of Anxiety Disorders, 15*, 217 - 230.

Cassidy, J. (1988). Child - mother attachment and the self in six - year - olds. *Child Development, 59*, 121 - 134.

Cassidy, J., Kirsch, S. J., Scolton, K. L., & Parke, R.D. (1996). Attachment and representation of peer relationships. *Developmental Psychology, 32*, 892 - 904.

Cavallini, M. C., Perna, G., Caldriola, D., & Bellodi, L. (1999). A segregation study of panic disorder in families of panic patients responsive to the 35% CO<sub>2</sub> challenge. *Biological Psychiatry, 46*, 815 - 820.

Chambless, D. L. (1982). Characteristics of agoraphobics. In D. Chambless & A. J. Goldstein (Eds.), *Agoraphobia: Multiple perspectives on theory and treatment*. New York: Wiley Interscience.

Chambless, D. L., Gillis, M. M., Tran, G. Q., & Steketee, G. S. (1996). Parental Bonding Reports of Clients with Obsessive - Compulsive Disorder and Agoraphobia. *Clinical Psychology & Psychotherapy, 3*(2), 77 - 85.

Chambless, D. L., Hunter, K., & Jackson, A. (1982). Social anxiety and assertiveness: A comparison of the correlations in phobic and college student samples. *Behaviour Research & Therapy, 20*(4), 403 - 404.

Chambless, D. L., & Mason, J. (1986). Sex, sex - role stereotyping and agoraphobia. *Behaviour Research and Therapy, 24*, 231 - 235.

Chambless, D. L., Renneberg, B., Goldstein, A., & Gracely, E.J. (1992). MCMI - diagnosed personality disorders among agoraphobic outpatients: Prevalence

and relationship to severity and treatment outcome. *Journal of Anxiety Disorders*, 6, 193 - 211.

Charney, D.S., Woods, S.W., Nagy, L.M., Southwick, S.M., Krystral, J.H., & Heninger, G.R. (1990). Noradrenergic function in panic disorder. *Journal of Clinical Psychiatry*, 51, 5 - 11.

Clark, D. M. (1986). A cognitive approach to panic. *Behaviour Research and Therapy*, 24, 461 – 470.

Clark, D. M. (1988). A cognitive model of panic attacks. In S. Rachman & J. D. Maser (Eds.), *Panic: Psychological perspectives* (pp. 71 – 89). Hillside, NJ: Erlbaum.

Clark, D. M. (1996). Panic disorder: From theory to therapy. In P.M. Salkovskis (Ed.). *Frontiers of cognitive therapy* (pp. 318 – 344). New York: Guilford Press.

Clark, D. M., Salkovskis, P. M., & Anastasiades, P. (1990, November). Cognitive mediation of lactate induced panic. In R. M. Rapee (Chair), *Experimental investigations of panic disorder*. Symposium conducted at the meeting of the Association for Advancement of Behavior Therapy, San Francisco.

Clark, D. M., Salkovskis, P. M., & Chalkley, A. J. (1985). Respiratory control as a treatment for panic attacks. *Journal of Behavior Therapy & Experimental Psychiatry*, 16(1), 23-30.

Clarke, J. C., & Wardman, W. (1985). *Agoraphobia: A clinical and personal account*. Sydney, Australia: Pergamon Press.

Clum, G. A., & Knowles, S. L. (1991). Why do some people with panic disorders become avoidant?: A review. *Clinical Psychology Review*, 11, 295 - 313.

Collins, N. L., Cooper, M. L., Albino, A., & Allard, L. (2002). Psychosocial vulnerability from adolescence to adulthood: A prospective study of attachment style

differences in relationship functioning and partner choice. *Journal of Personality*, 70(6), 965 - 1008.

Collins, N. L., & Read, S. J. (1990). Adult attachment, working models and relationship quality in dating couples. *Journal of Personality and Social Psychology*, 58(4), 644 - 663.

Cooper, M. L., Shaver, P. R., & Collins, N. L. (1998). Attachment styles, emotion regulation and adjustment in adolescence. *Journal of Personality and Social Psychology*, 74(5), 1380-1397.

Cox B. J., Borger, S. C., & Enns, M. W. (1999). Anxiety sensitivity and emotional disorders: Psychometric studies and their theoretical implications. In S. Taylor (Ed.), *Anxiety sensitivity: Theory, research, and treatment of the fear of anxiety*. Mahwah, New Jersey: Lawrence Erlbaum Associates.

Cox, B. J., Endler, N. S., Norton, G. R., & Swinson, R. P. (1991). Anxiety sensitivity and nonclinical panic attacks. *Behaviour Research & Therapy*, 29(4), 367 - 369.

Cox, B. J., Endler, N. S., & Swinson, R. P. (1991). Anxiety sensitivity and nonclinical panic attacks. *Behaviour Research & Therapy*, 29, 367 – 369.

Cox, B. J., Enns, M. W., & Clara, I. P. (2000). The Parental Bonding Instrument: Confirmatory evidence for a three - factor model in a psychiatric clinical sample and in the National Comorbidity Survey. *Social Psychiatry and Psychiatric Epidemiology*, 35(8), 353 - 357.

Craske, M. G. (1997). Fear and anxiety in children and adolescents. *Bulletin of the Menninger Clinic*, 61(2A), A4 - A34.

Crockenberg, S. B. (1981). Infant irritability, mother responsiveness and social support influences on the security of infant - mother attachment. *Child Development*, 52, 857 - 865.

- Crowe, R. R., Noyes, R., Pauls, D. L., & Slymen, D. (1983). A family study of panic disorder. *Archives of General Psychiatry*, *40*, 1065 - 1069.
- Crowell, J. A., Fraley, R. C., & Shaver, P. R. (1999). Measurement of Individual Differences in Adolescent and Adult Attachment. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research and clinical applications*. New York: Guilford Press.
- Dadds, M.R., Rosenthal Gaffney, L., Kenardy, J., Oei, T.P.S., & Evans, L. (1993). An exploration of the relationship between expression of hostility and the anxiety disorders. *Journal of Psychiatric Research*, *27*(1), 17 - 26.
- Dadds, M. R., & Roth, J. H. (2001). Family processes in the development of anxiety problems. In M. W. Vasey & M. R. Dadds (Eds.), *The developmental psychopathology of anxiety*. New York: Oxford University Press.
- de Ruiter, C., & van Ijzendoorn, M. (1992). Agoraphobia and anxious - ambivalent attachment: An integrity review. *Journal of Anxiety Disorders*, *6*, 365 - 381.
- Deutsch, H. (1929). The genesis of agoraphobia. *International Journal of Psycho - Analysis* *10*, 51 - 69.
- Dick, C. L., Bland, R. C., & Newman, S. C. (1994). Panic disorder. *Acta Psychiatrica Scandinavica*, *89*(376), 45 - 53.
- DiLalla, L.F., Kagan, J., & Reznick, J.S. (1994). Genetic etiology of behavioral inhibition among 2 - year - old children. *Infant Behavioral Development*, *17*, 405 - 412.
- Donnel, C. D., & McNally, R. J. (1990). Anxiety sensitivity and panic attacks in a nonclinical population. *Behaviour Research & Therapy*, *27*, 352 - 332.
- Dozier, M., Chase Stovall, K., & Albus, K. E. (1999). Attachment and psychopathology in adulthood. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of*

*attachment: Theory, research and clinical applications* (pp. 497 - 519). New York: Guilford Press.

Eagle, M. (1995). The developmental perspective of attachment and psychoanalytic theory. In S. Goldberg, R. Muir & J. Kerr (Eds.), *Attachment theory: Social, developmental and clinical perspectives*. NJ: Analytic Press.

Eaton, W. W., Dryman, A., & Weissman, M. M. (1991). Panic and phobia. In L. N. Robins & D. A. Regier (Eds.), *Psychiatric disorders in America* (pp. 155 - 179). New York: Free Press

Eaton, W. W., Kessler, R. C., Wittchen, H. U., & Magee, W. J. (1994). Panic and panic disorder in the United States. *American Journal of Psychiatry*, *151*(3), 413 - 420.

Ehlers, A. (1993). Somatic symptoms and panic attacks: A retrospective study of learning experiences. *Behaviour Research & Therapy*, *31*(3), 269 - 278.

Ehlers, A. (1995). A 1 - year prospective study of panic attacks: Clinical course and factors associated with maintenance. *Journal of Abnormal Psychology*, *104*, 164 - 172.

Emmelkamp, P. M. G. (1980). Agoraphobics' interpersonal problems: Their role in the effects of exposure *in vivo* therapy. *Archives of General Psychiatry*, *37*, 1303 - 1306.

Emmelkamp, P. M. G., & Bouman, T. K. (1991). Psychological approaches to the difficult patient. In J. R. Walker, G. R. Norton, & C. A. Ross (Eds.), *Panic disorders and agoraphobia* (pp. 398 - 430). Belmont: Brooks/ Cole.

Erickson, M. F., Sroufe, L. A., & Egeland, B. (1985). The relationship of quality of attachment and behaviour problems in preschool in a high risk sample. In I. Bretherton & E. Waters (Eds.), *Growing points in attachment theory and research. Monographs of the Society for Research in Child Development*, *50*(1 - 2, Serial No. 209), 147 - 186.

Faravelli, C., Panichi, C., Pallanti, S., Paterniti, S., Grecu, L. M., & Rivelli, S. (1991). Perception of early parenting in panic and agoraphobia. *Acta Psychiatrica Scandinavica*, 84, 6 - 8.

Faravelli, C., Webb, T., Ambonetti, A., Fonnesu, F., & Sessarago, A. (1985). Prevalence of traumatic early life events in 31 agoraphobic patients with panic attacks. *American Journal of Psychiatry*, 142, 1493 - 1494.

Feeney, J. A., & Noller, P. (1996). *Adult attachment*. Thousand Oaks, CA: Sage.

Feeney, J. A., Noller, P., & Callan, V. J. (1994). Attachment style, communication and satisfaction in the early years of marriage. In K. Bartholomew & D. Perlman (Eds.), *Advances in Personal Relationships: Vol 5. Attachment processes in adulthood* (pp. 269 - 308). London: Jessica Kingsley.

Feeney, J. A., Noller, P., & Hanrahan, M. (1994). Assessing adult attachment. In M. B. Sperling, & W. H. Berman (Eds.), *Attachment in adults: Clinical and developmental perspectives* (pp. 128 - 154). New York: Guilford Press.

Feldman, R., Greenbaum, C.W., & Yirmiya, N. (1999). Mother–infant affect synchrony as an antecedent of the emergence of self - control. *Developmental Psychology*, 35, 223–231.

Fisher, L. M., & Wilson, G. T. (1985). A study of the psychology of agoraphobia. *Behaviour Research & Therapy*, 23, 97 – 107.

Fivush, R. (1994). Constructing narrative, emotion and self in parent - child conversations about the past. In U. Neisser & R. Fivush (Eds.), *The remembering self: Construction and accuracy in the self - narrative* (pp 136 - 157). Cambridge, England: Cambridge University Press.

Forgays, D. G, Kirby Forgays, D., & Spielberger, C. D. (1997). Factor structure of the State - Trait Anger Expression Inventory. *Journal of Personality Assessment*, 69(3), 497 - 507.

Fox, N.A., & Calkins, S.D. (1993). Pathways to aggression and social withdrawal: Interactions among temperament, attachment, and regulation. In K.H. Rubin & J.B. Asendorpf (Eds.), *Social withdrawal, inhibition, and shyness in childhood* (pp. 81 – 100). Hillsdale, NJ: Erlbaum.

Fox, N.A., Henderson, H.A., Rubin, K.H., Calkins, S.D., & Schmidt, L.A. (2001). Continuity and discontinuity of behavioral inhibition and exuberance: Psychophysiological and behavioral influences across the first four years of life. *Child Development, 72*, 1 - 21.

Fyer, A. J., & Weissman, M. M. (1999). Genetic linkage study of panic: Clinical methodology and description of pedigrees. *American Journal of Medical Genetics, 88*, 173 - 181.

Garcia Coll, C., Kagan, J., & Reznick, J. S. (1984). Behavioral inhibition in young children. *Child Development, 55*(3), 1005 - 1019.

Garssen, B., Buikhuisen, M., & van Dyck, R. (1996). Hyperventilation and panic attacks. *American Journal of Psychiatry, 153*(4), 513 - 518.

Garssen, B., de Ruiter, C., & Van Dyck, R. (1992). Breathing retraining: A rational placebo? *Clinical Psychology Review, 12*(2), 141 - 153.

Ge, X., Lorenz, F.O., Conger, R. D., Elder, G. H., & Simons, R. L. (1994). Trajectories of stressful life events and depressive symptoms during adolescence. *Developmental Psychology, 30*(4), 467–483.

Gerlsma, C., Emmelkamp, P., & Arrindell, W. (1990). Anxiety, depression, and perception of early parenting: A meta - analysis. *Clinical Psychology Review, 10*, 251 – 277.

Gersten, M. (1989). Behavioral inhibition in the classroom. In J.S. Reznick (Ed.), *Perspectives of behavioral inhibition* (pp. 71 – 91). Chicago: University of Chicago Press.

Gittelman, R., & Klein, D. F. (1985). Childhood separation anxiety and adult agoraphobia. In A. H. Tuma, & J. D. Masser (Eds.), *Anxiety and the anxiety disorders* (pp. 389 - 402). Hillsdale, NJ: Erlbaum.

Gittelman - Klein, R., & Klein, D. F. (1973). School phobia: Diagnostic considerations in the light of imipramine effects. *Journal of Nervous & Mental Disease*, 156(3), 199 - 201.

Goldberg, S. (1995). Introduction. In S. Goldberg, R. Muir & J. Kerr. (Eds.), *Attachment theory: Social, developmental and clinical perspectives*. NJ: Analytic Press.

Goldsmith, H. H., & Alansky, J. A. (1987). Maternal and infant temperamental predictors of attachment: A meta - analytic review. *Journal of Consulting and Clinical Psychology*, 55, 805 - 816.

Goldstein, A. J. & Chambless, D. L. (1978). A reanalysis of agoraphobia. *Behavior Therapy*, 9, 47 - 59.

Gorman, J. M., Cohen, B. S., Liebowitz, M. R., Fyer, A. J., Ross, D., Davies, S. O., & Klein, D. F. (1986). Blood gas changes and hypophosphatemia in lactate - induced panic. *Archives of General Psychiatry*, 43, 1067 - 1071.

Gorman, J. M., Askanazi, J., Liebowitz, M. R., Fyer, A. J., Stein, J., Kinney, J. M., & Klein, D. F. (1984). Response to hyperventilation in a group of patients with panic disorder. *American Journal of Psychiatry*, 141, 857 - 861.

Gorman, J. M., Liebowitz, M. R., Fyer, A. J., & Stein, J. (1989). A neuroanatomical hypothesis for panic disorder. *American Journal of Psychiatry*, 146, 148 - 161.

Gorman, J. M., & Papp, L. A. (1990). Respiratory physiology of panic. In J. C. Ballenger (Ed.), *Neurobiology of panic disorder* (pp. 187 - 203). New York: Wiley - Liss.

Gorman, J. M., Papp, L. A., Coplan, J., Martinez, J., Lennon, R. N., Goetz, R. R., Ross, D., & Klein, D. F. (1994). Anxiogenic effects of CO<sub>2</sub> and hyperventilation in patients with panic disorder. *American Journal of Psychiatry*, *151*, 547 - 553.

Griffin, D. W., & Bartholomew, K. (1994). The metaphysics of measurement: The case of adult attachment. In K. Bartholomew & D. Perlman (Eds.), *Advances in personal relationships: Vol. 5. Attachment processes in adulthood* (pp. 17 - 52). London: Jessica Kingsley.

Grossman, K. E., & Grossman, K. (1991). Attachment quality as an organizer of emotional and behavioral responses in a longitudinal perspective. In C. M. Parkes, J. Stevenson - Hinde & P. Marris. (Eds.), *Attachment across the life cycle* (pp. 93 - 114). London: Routledge.

Grossman, K., Grossman, K., & Schwan, A. (1986). Capturing the wider view of attachment: a re - analysis of Ainsworth's Strange Situation. In C. Elzard & P. Read (Eds.), *Measuring emotions in infants and children*. Cambridge: Cambridge University Press.

Guidano, V. F. (1987). *Complexity of the self*. New York: Guilford Press

Hafner, R. J. (1982). The marital context of the agoraphobic syndrome. In D. Chambless & A. J. Goldstein (Eds.), *Agoraphobia: Multiple perspectives on theory and treatment*. New York: Wiley Interscience.

Hamilton, C. E. (2000). Continuity and discontinuity of attachment from infancy through adolescence. *Child Development*, *71*(3), 690 - 694.

Harris, T. O., Brown, G. W., & Bifulco, A. (1986). Loss of parent in childhood and adult psychiatric disorder: The Walthamstow Study. 1. The role of lack of adequate parental care. *Psychological Medicine*, *16*, 614 - 659.

Harris, T. O., Brown, G. W., & Bifulco, A. T. (1990a). Depression and situational helplessness / mastery in a sample selected to study childhood parental loss. *Journal of Affective Disorders*, *20*, 27 - 41.

Harris, T. O., Brown, G. W., & Bifulco, A. T. (1990b). Loss of parent in childhood and adult psychiatric disorder: A tentative overall model. *Development & Psychopathology*, 2, 311 - 328.

Hayward, C., Killen, J. D., Hammer, L. D., Litt, I. F., Wilson, D. M., Simmonds, B., & Taylor, C. B. (1992). Pubertal stage and panic attack history in sixth- and seventh- grade girls. *American Journal of Psychiatry*, 149, 1239 - 1243.

Hayward, C., Killen, J. D., Kraemer, H. C., Taylor, C. B. (2000). Predictors of panic attacks in adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry*, 39, 207 - 214.

Hayward, C., Killen, J., Kraemer, H., & Taylor, B. (1998). Linking self-reported childhood behavioral inhibition to adolescent social phobia. *Journal of the American Academy of Child & Adolescent Psychiatry*, 37, 1308 - 1316.

Hayward, C., Killen, J. D., & Taylor, C. B. (1989). Panic attacks in young adolescents. *American Journal of Psychiatry*, 146, 1061 - 1062.

Hazan, C., & Shaver, P. R. (1987). Romantic love conceptualized as an attachment process. *Journal of Personality & Social Psychology*, 52, 511 - 524.

Hazan, C., & Shaver, P.R. (1990). Love and work: An attachment – theoretical perspective. *Journal of Personality & Social Psychology*, 59, 270 - 280.

Hibbert, G., & Pilsbury, D. (1989). Hyperventilation: Is it a cause of panic attacks? *British Journal of Psychiatry*, 155, 805 - 809.

Hirshfeld, D. R., Rosenbaum, J. F., Biederman, J., Bolduc, E. A., Faraone, S. V., Reznick, J. S., & Kagan, J. (1992). Stable behavioral inhibition and its association with anxiety disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 31, 103 - 111.

Holloway, W., & McNally, R.J. (1987). Effects of anxiety sensitivity on the response to hyperventilation. *Journal of Abnormal Psychology*, 96, 330 - 334.

Holmes, J. (1993). *John Bowlby and Attachment Theory*. London: Routledge.

- Jack, D.C. (1991). *Silencing the self: Women and depression*. New York: Harper Collins.
- Jacobson, J. L., & Willie, D. R. (1986). The influence of attachment pattern on developmental changes in peer interaction from the toddler to the preschool period. *Child Development, 57*, 338 - 347.
- Jang, K.L., Stein, M.B., Taylor, S., & Livesley, W.J. (1999). Gender differences in the etiology of anxiety sensitivity: A twin study. *Journal of Gender Specific Medicine, 2*, 39 - 44.
- Judd, F. K., Burrows, G. D., & Hay, D. A. (1987). Panic Disorder: Evidence for genetic vulnerability. *Australian & New Zealand Journal of Psychiatry, 21*, 197 - 208.
- Kagan, J. (1982). *Psychological research on the human infant: An evaluative summary*. New York: William T. Grant Foundation.
- Kagan, J. (1994). *Galen's Prophecy*. New York: Basic Books.
- Kagan, J., Reznick, J. S., Clarke, C., Snidman, N., & Garcia - Coll, C. (1984). Behavioral inhibition to the unfamiliar. *Child Development, 55*(6), 2212 - 2225.
- Kagan, J., Reznick, J. S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. *Child Development, 58*(6), 1459 - 1473.
- Kagan, J., Reznick, J. S., & Snidman, N. (1988). Biological basis of childhood shyness. *Science, 240*, 167 - 171.
- Kagan, J., Snidman, N., & Arcus, D. (1998). Childhood derivatives of high and low reactivity in infancy, *Child Development, 69*, 1483 - 1493.
- Katerndahl, D. A., & Realini, J. P. (1993). Lifetime prevalence of panic states. *American Journal of Psychiatry, 150*, 246 - 249.

Kendler, K. S. (1996). Parenting: A genetic - epidemiologic perspective. *American Journal of Psychiatry*, 153(1), 11 - 20.

Kendler, K. S., Heath, A., Martin, N. G., & Eaves, L. J. (1986). Symptoms of anxiety and depression in a volunteer twin population: The etiologic role of genetic and environmental factors. *Archives of General Psychiatry*, 43, 213 – 221.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1992). Childhood parental loss and adult psychopathology in women: A twin study perspective. *Archives of General Psychiatry*, 49(2), 109 - 116.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1993). Panic disorder in women: A population-based twin study. *Psychological Medicine*, 23, 397 - 406.

Kim, Y., Min, S. K., & Yu, B. (2004) Differences in beta-adrenergic receptor sensitivity between women and men with panic disorder. *European Neuropsychopharmacology*, 14(6), 515 - 520.

King, N. J., Gullone, E., Tonge, B. J., & Ollendick, T. H. (1993). Self-reports of panic attacks and manifest anxiety in adolescents. *Behaviour Research & Therapy*, 31, 111 - 116.

King, N. J., Ollendick, T.H., Mattis, S.G., Yang, B., & Tonge, B. (1996). Nonclinical panic attacks in adolescents: Prevalence, symptomatology, and associated features. *Behaviour Change*, 13(3), 171 - 183.

Klein, D. F. (1964). Delineation of drug responsive anxiety syndromes. *Psychopharmacologia*, 5, 397 - 408

Klein, D. F. (1981). Anxiety reconceptualized. In D. F. Klein & J. Rabkin (Eds.), *Anxiety: New research and changing concepts* (pp. 235 - 263). New York: Raven Press.

Klein, R. G., Koplewicz, H. S., & Kanner, A. (1992). Imipramine treatment of children with separation anxiety disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 31*(1), 21 - 28.

Klein, D. F. (1993). False suffocation alarms, spontaneous panics, and related conditions: An integrative hypothesis. *Archives of General Psychiatry, 50*, 306 - 317.

Klein, D. F., Ross, D. C., & Cohen, P. (1987). Panic and avoidance in agoraphobia: Application of path analysis to treatment studies. *Archives of General Psychiatry, 44*(4), 377 - 385.

Klein, D. F., Zitrin, C. M., Woerner, M. G., & Ross, D. C. (1983). Treatment of phobias: II. Behavior therapy and supportive psychotherapy: Are there any specific ingredients? *Archives of General Psychiatry, 40*, 139 - 145.

Kleiner, L., & Marshall, W. L. (1987). The role of interpersonal problems in the development of agoraphobia with panic attacks. *Journal of Anxiety Disorders, 1*, 313 - 323.

Kobak, R.R., & Sceery, A. (1988). Attachment in late adolescence: Working models, affect regulation, and representations of self and others. *Child Development, 59*, 135 - 146.

Korn, M. L., Plutchik, R., & van Praag, H. M. (1997). Panic - associated suicidal and aggressive ideation and behavior. *Journal of Psychiatric Research, 31*(4), 481 - 487.

Lader, M. H., & Mathews, A. M. (1970). Physiological changes during spontaneous panic attacks. *Journal of Psychosomatic Research, 14*, 377 - 382.

Last, C. G., & Strauss, C. C. (1989). Panic disorder in children and adolescents. *Journal of Anxiety Disorders, 3*, 87 - 95.

Laible, D. J., & Thompson, R. A. (1998). Attachment and emotional understanding in preschool children. *Developmental Psychology, 34*, 1038 - 1045.

Lau, J. J., Calamari, J. E., & Waraczynski, M. (1996). Panic attack symptomatology and anxiety sensitivity in adolescents. *Journal of Anxiety Disorders, 10*, 355 - 364.

Lejuez, C. W., Eifert, G. H., Zvolensky, M. J., & Richards, J. B. (2000). Preference between onset predictable and unpredictable administrations of 20% carbon-dioxide-enriched air: Implications for better understanding the etiology and treatment of panic disorder. *Journal of Experimental Psychology: Applied, 6*(4), 349-358.

Leon, C. A., & Leon, A. (1990). Panic disorder and parental bonding. *Psychiatric Annals, 20*, 503 - 508.

Lesser, I.M. (1990). Panic disorder and depression: Co - occurrence and treatment. In J. C. Ballenger (Ed.), *Clinical aspects of panic disorders* (pp. 181 - 191). New York: Wiley - Liss.

Lesser, I. M., Rubin, R. T., Pecknold, J. C., Rifkin, A., Swinson, R. P., Lydiard, R. B., Burrows, G. D., Noyes, R., Jr., & DuPont, R. L., Jr. (1988). Secondary depression in panic disorder and agoraphobia: 1. Frequency, severity and response to treatment. *Archives of General Psychiatry, 45*, 437 - 443.

Lester, B.M., Hoffman, J., & Brazelton, T.B. (1985). The rhythmic structure of mother–infant interaction in term and preterm infants. *Child Development, 56*, 15–27.

Lewis, M., Feiring, C., & Rosenthal, S. (2000). Attachment over Time. *Child Development, 71*(3), 707 - 720.

Ley, R. (1985). Agoraphobia, the panic attack, and the hyperventilation syndrome. *Behaviour Research & Therapy, 23*, 79 - 81.

Ley, R. (1987a). Panic disorder: A hyperventilation interpretation. In L. Michelson & L. M. Ascher (Eds.), *Anxiety and stress disorders: Cognitive–behavioral assessment and treatment* (pp. 191 - 212). New York: Guilford Press.

Ley, R. (1987b). Panic disorder and agoraphobia: Fear of fear or fear of the symptoms produced by hyperventilation? *Journal of Behavior Therapy and Experimental Psychiatry*, 18, 305 - 316.

Ley, R. (1988). Hyperventilation and lactate infusion in the production of panic attacks. *Clinical Psychology Review*, 8, 1 - 18.

Ley, R. (1989). Dyspneic - fear and catastrophic cognitions in hyperventilatory panic attacks. *Behaviour Research and Therapy*, 27, 549 - 554.

Ley, R. (1991). The efficacy of breathing retraining and the centrality of hyperventilation in panic disorder: A reinterpretation of experimental findings. *Behaviour Research & Therapy*, 29(3), 301 - 304.

Ley, R. (1996). Panic attacks: Klein's false suffocation alarm, Taylor and Rachman's data, and Ley's dyspneic - fear theory. *Archives of General Psychiatry*, 53(1), 83 - 85.

Liebowitz, M. R., Gorman, J. M., Fyer, A. J., Levitt, M., Dillon, D., Levy, G., Appleby, I.L., Anderson, S., Palij, M., Davies, S. O., & Klein, D. F. (1985a). Lactate provocation of panic attacks: II. Biochemical and physiological findings. *Archives of General Psychiatry*, 42, 709 - 719.

Liebowitz, M. R., Fyer, A. J., Gorman, J. M., Dillon, D., Davies, S., Stein, J. M., Cohen, B. S., & Klein, D. F. (1985b). Specificity of lactate infusions in social phobia versus panic disorders. *American Journal of Psychiatry*, 142, 947 - 950.

Lilienfeld, S. O., Turner, S. M., & Jacob, R. G. (1989). Comment on Holloway and McNally's (1987) "Effects of anxiety sensitivity on the response to hyperventilation. *Journal of Abnormal Psychology*, 98(1), 100 - 102.

Lilienfeld, S. O., Turner, S. M., & Jacob, R. G. (1993). Anxiety sensitivity: An examination of theoretical and methodological issues. *Advances in Behaviour Research & Therapy*, 15, 147 - 182.

Liotti, G. (1991). Insecure attachment and agoraphobia. In C. M. Parkes, J. Stevenson-Hinde & P. Marris (Eds.), *Attachment across the life cycle* (pp. 216 - 233). London: Routledge.

Lipsitz J.D., Martin, L. Y., Mannuzza, S., Chapman, T. F., Liebowitz, M. R., Klein, D. F., & Fyer, A. J. (1994). Childhood separation anxiety disorder in patients with adult anxiety disorders. *American Journal of Psychiatry*, *151*(6), 927 - 929.

Lizardi, H., & Klein D. N. (2002). Evidence of increased sensitivity using a three - factor version of the Parental Bonding Instrument. *Journal of Nervous & Mental Disease*, *190*(9), 619 - 623.

Lousberg, H., Griez E., & van den Hout, M. A. (1988). Carbon dioxide chemosensitivity in panic disorder. *Acta Psychiatrica Scandinavica*, *77*, 214 - 218.

Macauley, J. L., & Kleinknecht, R. A. (1989). Panic and panic attacks in adolescents. *Journal of Anxiety Disorders*, *3*, 87 - 95.

MacKinnon, A., Henderson, A., & Andrews, G. (1993). Parental 'affectionless control' as an antecedent to adult depression: A risk factor refined. *Psychological Medicine*, *23*, 135 - 141.

Magarin, G. J. (1982). Hyperventilation syndromes: Infrequently recognized common expressions of anxiety and stress. *Medicine*, *61*, 219 - 236.

Main, M., & Cassidy, J. (1988). Categories of response to reunion with the parent at age 6: Predictable from infant attachment classifications and stable over a 1-month period. *Developmental Psychology*, *24*, 415 - 426.

Main, M., Kaplan, N., & Cassidy, J. (1985). Security in infancy, childhood, and adulthood: A move to the level of representation. In I. Bretherton & E. Waters (Eds.), Growing points of attachment theory and research. *Monographs of the Society for Research in Child Development*, *50*(1 - 2, Serial No. 209), 66 - 104.

Main, M., & Solomon, J. (1986). Discovery of an insecure – disorganized/disoriented attachment pattern. In T.B. Brazelton & M.W. Yogman (Eds.), *Affective development in Infancy* (pp. 95 - 124). Norwood, NJ: Ablex.

Main, M., & Solomon, J. (1990). Procedures for identifying infants as disorganized/disoriented attachment pattern. In M.T. Greenberg, D. Cicchetti & E.M. Cummings (Eds.), *Attachment in the preschool years* (pp. 121 - 160). Chicago: University of Chicago Press.

Main, M., & Weston, D.R. (1981). The quality of the toddler's relationship to mother and to father: Related to conflict behavior and the readiness to establish new relationships. *Child Development*, 52, 932 - 940.

Main, M., & Weston, D.R. (1982). Avoidance of the attachment figure in infancy: descriptions and interpretations. In C.M. Parkes & J. Stevenson - Hinde (Eds.), *The place of attachment in human behavior* (pp.31 - 59). New York: Basic Books.

Maller, R.G., & Reiss, S. (1992). Anxiety sensitivity in 1984 and panic attacks in 1987. *Journal of Anxiety Disorders*, 6 241 - 247.

Manassis, K., Bradley, S., Goldberg, S., Hood, J., & Swinson, R. (1994). Attachment in mothers with anxiety disorders and their children. *Journal of the American Academy of Child & Adolescent Psychiatry*, 33, 1106 - 1113.

Mangelsdorf, S., Gunnar, M. R., Kestenbaum, R., Lang, S., & Andreas, D. (1990). Infant proneness - to - distress temperament, maternal personality, and mother - infant attachment: Associations and goodness of fit. *Child Development*, 61(3), 820 - 831.

Manicavasagar, V., & Silove, D. (1997). Is there an adult form of separation anxiety disorder? A brief clinical report. *Australian & New Zealand Journal of Psychiatry*, 31(2), 299 - 303.

Manicavasagar, V., Silove, D., & Curtis, J. (1997). Separation anxiety in adulthood: A phenomenological investigation. *Comprehensive Psychiatry*, 38(5), 274 - 282.

Manicavasagar, V., Silove, D., & Hadzi - Pavlovic, D. (1998). Subpopulations of early separation anxiety: Relevance to risk of adult anxiety disorders. *Journal of Affective Disorders* 48,181 - 190.

Manicavasagar, V., Silove, D., Wagner, R., & Hadzi - Pavlovic, D. (1999). Parental representations associated with adult separation anxiety and panic disorder - agoraphobia. *Australian & New Zealand Journal of Psychiatry*, 33(3), 422 - 428.

Marcaurelle, R., Belanger, C., Marchand, A., Katerelos, T. E., & Mainguy, N. (2005). Marital predictors of symptom severity in panic disorder with agoraphobia. *Journal of Anxiety Disorders*, 19(2), 211 - 232.

Margraf, J., Taylor, C. B., Ehlers, A., Roth, W. T., & Agras, W. S. (1987). Panic attacks in the natural environment. *Journal of Nervous and Mental Disease* 175(9), 558 - 565.

Markowitz, J. S., Weissman, M. M., Ouellette, R., Lish, J. D., & Klerman, G. L. (1989). Quality of life in panic disorder. *Archives of General Psychiatry*, 46, 984 - 992.

Marks, I. M. (1987). Behavioral aspects of panic disorder. *American Journal of Psychiatry*, 144, 1160 - 1165.

Martin, G., Bergen, H. A., Roeger, L., & Allison, S. (2004). Depression in young adolescents: Investigations using 2 and 3 factor versions of the Parental Bonding Instrument. *Journal of Nervous & Mental Disease*, 192(10), 650 - 657.

Masi, G., Favilla, L., Mucci, M., & Millepiedi, S. (2000). Panic disorder in clinically referred children and adolescents. *Child Psychiatry and Human Development*, 31(2), 139 - 151.

- Matas, Arend, & Sroufe, (1978). Continuity of adaptation in the second year: The relationship between quality of attachment and later competence. *Child Development, 49*, 547 - 556.
- Mathews, A. M., Gelder, M. G., & Johnston, D. W. (1981). *Agoraphobia: Nature and treatment*. New York: Guilford Press.
- Mattis, S. G., & Ollendick, T. H. (1997). Children's cognitive responses to the somatic symptoms of panic. *Journal of Abnormal Child Psychology, 25*, 47 - 57.
- McNally, R. J., & Lorenz, M. (1987). Anxiety sensitivity in agoraphobics. *Journal of Behavior Therapy and Experimental Psychiatry, 18*, 3 - 11.
- McNally, R. J. (1990). Psychological approaches to panic disorder: A review. *Psychological Bulletin, 108*, 403 - 419.
- McNally, R. J. (1994). *Panic disorder: A critical analysis*. New York: Guilford Press.
- McNally, R. J. (1999). Panic and Phobias. In T. Dalgleish & M. J. Power (Eds.), *Handbook of cognition and emotion*. Chichester: John Wiley & Sons.
- McNally, R. J. (2002). Anxiety sensitivity and panic disorder. *Biological Psychiatry, 52* (10), 938 - 946.
- Mennin, D. S., & Heimberg, R. G. (2000). The impact of comorbid mood and personality disorders in the cognitive - behavioral treatment of panic disorder. *Clinical Psychology Review, 20*(3), 339 - 357.
- Mickelson, K. D., Kessler, R. C., & Shaver, P. R. (1997). Adult attachment in a nationally representative sample. *Journal of Personality and Social Psychology, 73*(5), 1092 - 1106.
- Moon, L., P. Meyer, & Grau, J. (1999). *Australia's young people - Their health and wellbeing: The first report on the health of young people aged 12 - 24 years by the Australian Institute of Health and Welfare*. Australia, The Australian Institute of Health and Welfare: 2.

Moreau, D., & Weissman, M. M. (1992). Panic disorder in children and adolescents: A review. *American Journal of Psychiatry*, *149*, 1306 - 1314.

Murphy, E., Brewin, C. R., & Silka, L. (1997). The assessment of parenting using the Parental Bonding Instrument: Two or three factors? *Psychological Medicine*, *27*(2), 333 - 342.

Nachmias, M., Gunnar, M., Mangelsdorf, S., Parritz, R. H., & Buss, K. (1996). Behavioral inhibition and stress reactivity: The moderating role of attachment security. *Child Development*, *67*, 508 - 522.

Nagy, L. M., Krystal, J. H., Charney, D. S., Merikangas, K.R., & Woods, S. W. (1993). Long - term outcome of panic disorder after short - term imipramine and behavioral group treatment: 2.9 year naturalistic follow - up study. *Journal of Clinical Psychopharmacology*, *13*, 16 - 24.

Nagy, L. M., Krystal, J. H., Woods, S. W., & Charney, D. S. (1989). Clinical and medication outcome after short - term alprazolam and behavioral group treatment of panic disorder: 2.5 - year naturalistic follow - up study. *Archives of General Psychiatry*, *46*, 993 - 999.

Nelson, K. (1993). The psychological and social origins of autobiographical memory. *Psychological Science*, *4*, 7 - 14.

Norton, G. R., Dorward, J., & Cox, B. J. (1986). Factors associated with theoretical panic attacks in nonclinical subjects. *Behavior Therapy*, *17*, 239 - 252.

Norton, G. R., Harrison, B., Hauch, J., & Rhodes, L. (1985). Characteristics of people with infrequent panic attacks. *Journal of Abnormal Psychology*, *94*, 216 - 221.

Noyes, R., Reich, J., Christiansen, J., Suelzer, M., Pfohl, B., & Coryell, W. A. (1990). Outcome of Panic Disorder: Relationship to diagnostic subtypes and comorbidity. *Archives of General Psychiatry*, *47*, 809 - 818.

Oei, T. P. S., Wanstall, K., & Evans, L. (1990). Sex differences in panic disorder and agoraphobia. *Journal of Anxiety Disorders*, *4*, 317 - 324.

Oppenheim, D., & Waters, H. A. (1985). Narrative processes and attachment representations: Issues of development and assessment, In E. Waters, B. E. Vaughn, G. Posada, & K. Kondo - Ikemura (Eds.), *Caregiving, cultural, and cognitive perspectives on secure - base behaviour and working models: New growing points of attachment theory and research. Monographs of the Society for Research in Child Development, 60*(2 - 3, Serial No. 244), 197 - 215.

Orvaschel, H. (1994). *Schedule for Affective Disorders and Schizophrenia for School - Age Children - Epidemiologic Version (K - SADS - E)* (5<sup>th</sup> ed.). Fort Lauderdale, Fla: Nova Southeastern University.

Otto, M. W., Pollack, M. H., Sachs, G. S., & Rosenbaum, J. F. (1992). Hypochondriacal concerns, anxiety sensitivity, and panic disorder. *Journal of Anxiety Disorders, 6*(2), 93 - 104.

Pacchierotti, C., Bossini, L., Catrogiovanni, A., Pieraccini, F., Soreca, I., & Castrogiovanni, P. (2002). Attachment and panic disorder. *Psychopathology, 35*(6), 347 - 354.

Pain, M. C. F., Biddle, N., & Tiller, J. W. G. (1988). Panic disorder, the ventilatory response to carbon dioxide and respiratory variables. *Psychosomatic Medicine, 50*, 541 - 548.

Pam, A., Inghilterra, K., & Munson, C. (1994). Agoraphobia: The interface between anxiety and personality disorder. *Bulletin of the Menninger Clinic, 58*(2), 242 - 261.

Papp, L. A., Goetz, R., Cole, R., Klein, D. F., Jordan, F., Liebowitz, M. R., Fyer, A. J., Hollander, E., & Gorman, J. M. (1989). Hypersensitivity to carbon dioxide in panic disorder. *American Journal of Psychiatry, 146*, 779 - 781.

Parker, G. (1979a). Reported parental characteristics of agoraphobics and social phobics. *British Journal of Psychiatry, 135*, 555 - 560.

Parker, G. (1979b). Reported parental characteristics in relation to trait depression and anxiety levels in a non - clinical group. *Australian & New Zealand Journal of Psychiatry*, 13(3), 260 - 264.

Parker, G. (1990). The Parental Bonding Instrument: A decade of research. *Social Psychiatry and Psychiatric Epidemiology*, 25, 281 - 282.

Parker, G., Tupling, H., & Brown, L. B. (1979). A Parental Bonding Instrument. *Journal of Medical Psychology*, 52, 1 - 10.

Patrick, M., Hobson, R. P., Castle, D., & Howard, R. (1994). Personality disorder and the mental representation of early social experience. *Development & Psychopathology*, 6, 375 - 388.

Pearson, J. A., Cohn, D. A., Cowan, P. A., & Cowan, C. P. (1994). Earned- and continuous- security in adult attachment: relation to depressive symptomatology and paring style. *Development & Psychopathology*, 6, 359 - 373.

Peterson, R. A., & Reiss, S. (1993). *Anxiety Sensitivity Index Revised Test Manual*. Worthington, OH, IDS Publishing.

Pettem, O., West, M., Mahoney, A., & Keller, A. (1993). Depression and attachment problems. *Journal of Psychiatry and Neuroscience*, 18, 78 - 81.

Pollack, M.H., Otto, M.W., Sabatino, S., Majcher, D., Worthington, J.J., McArdle, E.T., & Rosenbaum, J.F. (1996). Relationship of childhood anxiety to adult panic disorder: Correlates and influence on course. *American Journal of Psychiatry*, 153, 376 - 381.

Raj, B. A., Corvea, M. H., & Dagon, E. M. (1993). The clinical characteristics of panic disorder in the elderly: A retrospective study. *Journal of Clinical Psychiatry*, 54, 150 - 155.

Rapee, R. M. (1985). Distinctions between panic disorder and generalised anxiety disorder: Clinical presentation. *Australian and New Zealand Journal of Psychiatry*, 19, 227 - 232.

Rapee, R. M. (1997). Potential role of childrearing practices in the development of anxiety and depression. *Clinical Psychology Review, 17*(1), 47 - 67.

Rapee, R.M., Brown, T.A., Antony, M.M., & Barlow, D.H. (1992). Response to hyperventilation and inhalation of 5.5% carbon dioxide - enriched air across the DSM – III - R anxiety disorders. *Journal of Abnormal Psychology, 101*, 538 - 552.

Rapee, R., Mattick, R., & Murell, E. (1986). Cognitive mediation in the affective component of spontaneous panic attacks. *Journal of Behavior Therapy and Experimental Psychiatry, 17*, 245 - 254.

Reed, V., & Wittchen, H - U. (1998). DSM - IV panic attacks and panic disorder in a community sample of adolescents and young adults: How specific are panic attacks? *Journal of Psychiatric Research, 32*(6), 335 - 345.

Reich, J., Noyes, R. Jr., Troughton, E. (1987). Dependent personality disorder associated with phobic avoidance in patients with panic disorder. *American Journal of Psychiatry, 144*, 323 - 326

Reiss, S., & McNally, R. J. (1985). The expectancy model of fear. In S. Reiss & R. R. Bootzin (Eds.), *Theoretical Issues in Behavior Therapy*. New York: Academic Press

Reiss, S. (1992). Expectancy model of fear, anxiety and panic. *Clinical Psychology Review, 11*, 141 - 153.

Reiss, S. (2000). *Who am I: The 16 desires that motivate our actions and determine our personality*. New York: Tarcher/ Putnam.

Reti, I. M., Samuels, J. F., Eaton, W. W., Bienvenu, O. J. III, Costa, P. T. Jr., & Nestadt, G. (2002). Adult antisocial personality traits are associated with experience of low parental care and maternal overprotection. *Acta Psychiatrica Scandinavica, 106*(2), 126 - 133.

Robinson, J. L., Kagan, J., Reznick, J. S., & Corley, R. (1992). The heritability of inhibited and uninhibited behaviour: A twin study. *Developmental Psychology, 28*, 1030 - 1037.

Rodriguez, B. F., Bruce, S. E., Pagano, M. E., Spencer, M. A., & Keller, M. B. (2004). Factor structure and stability of the Anxiety Sensitivity Index in a longitudinal study of anxiety disorder patients. *Behaviour Research & Therapy, 42*(1), 79 - 91.

Rosenbaum, J. F., Biederman, J., Gersten, M., Hirshfeld, D.R., Meminger, S. R., Herman, J.B., Kagan, J., Reznick, S., & Snidman, N. (1988). Behavioral inhibition in children of parents with panic disorder and agoraphobia: A controlled study. *Archives of General Psychiatry, 45*, 463 - 470.

Rosenbaum, J. F., Biederman, J., Hirshfeld - Becker, D.R., Kagan, J., Snidman, N., Friedman, D., Nineberg, A., Gallery, D.J., & Faraone, S.V. (2000). A controlled study of behavioral inhibition in children of parents with panic disorder and depression. *American Journal of Psychiatry, 157*(12), 2002 - 2010.

Rothbard, J.C., & Shaver, P.R. (1994). Continuity of attachment across the life span, In M.B. Sperling & W.H. Berman (Eds.), *Attachment in adults: Clinical and developmental perspectives* (pp. 31 - 71). New York: Guilford Press.

Rutter, M., & Quinton, D. (1977). Psychiatric disorder: Ecological factors and concepts of causation. In H. McGurk (Ed.), *Ecological factors in human development* (pp. 173 - 187). Amsterdam: North Holland.

Rutter, M., & Quinton, D. (1984). Parental psychiatric disorder: effects on children. *Psychological Medicine, 14*, 853 - 880.

Salkovskis, P. M., & Clark, D. M. (1990). Affective responses to hyperventilation: A test of the cognitive model of panic. *Behaviour Research and Therapy, 28*, 51 - 61.

Sander, L. (1988). The event-structure of regulation in the neonate-caregiver system as a biological background of early organization of psychic structure. In A. Goldberg (Ed.), *Frontiers in self psychology* (pp. 64-77). Hillsdale, NJ: Erlbaum.

Sanderson, W. C., Rapee, R. M., & Barlow, D. H. (1989). The influence of an illusion of control on panic attacks induced via inhalation of 5.5% carbon - dioxide - enriched air. *Archives of General Psychiatry*, 46(2), 157 - 167.

Sato, T., Narita, T., Hirano, S., Kusunoki, K., Sakado, K., & Uehara, T. Confirmatory factor analysis of the Parental Bonding Instrument in a Japanese population. *Psychological Medicine*, 29(1), 127 - 133.

Scharfe, E., & Bartholomew, K. (1994). Reliability and stability of adult attachment patterns. *Personal Relationships*, 1, 23 - 43.

Scher, C.D., & Stein, M.B. (2003). Developmental antecedents of anxiety sensitivity. *Journal of Anxiety Disorders*, 17(3), 253 - 269.

Schmidt, N.B., & Lerew, D.R. (2002). Prospective evaluation of perceived control, predictability, and anxiety sensitivity in the pathogenesis of panic. *Journal of Psychopathology & Behavioral Assessment*, 24(4), 207 - 214.

Schmidt, N.B., Lerew, D.R., & Jackson, R.J. (1997). The role of anxiety sensitivity in the pathogenesis of panic: Projective evaluation of spontaneous panic attacks during acute stress. *Journal of Abnormal Psychology*, 106, 355 - 364.

Schmidt, N.B., Lerew, D.R., & Jackson, R.J. (1999). Prospective evaluation of anxiety sensitivity in the pathogenesis of panic: Replication and extension. *Journal of Abnormal Psychology*, 108, 532 - 537.

Schmidt, N.B., Lerew, D.R., & Joiner, T.E. Jr (2000). Prospective evaluation of the etiology of anxiety sensitivity: Test of a scar model. *Behavioural Research Therapy*, 38, 1083 - 1095.

Schmidt, N.B., Storey, J., Greenberg, B.D., Sandiego, H.T., Li, Q., & Murphy, D.L. (2000). Evaluating gene \* psychological risk factor effects in the pathogenesis of anxiety: A new model approach. *Journal of Abnormal Psychology*, 109 (2), 308 - 320.

Schmidt, N. B., Telch M. J., & Jaimez T. L. (1996). Biological challenge of PCO - sub - 2 levels: A test of Klein's (1993) suffocation alarm theory of panic. *Journal of Abnormal Psychology, 105*(3), 446 - 454.

Schore, A.N. (1994). *Affect regulation and the origin of the self: The neurobiology of emotional development*. Mahwah, NJ: Erlbaum.

Schore, A.N. (2001). Effects of a secure attachment relationship on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal, 22*(1-2), 7 - 66.

Schwartz, C.E., Snidman, N., & Kagan, J. (1999). Adolescent social anxiety as an outcome of inhibited temperament in childhood. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*, 1008 - 1015.

Siegert, R.J., Ward, T., & Hudson, S. (1995). The structure of romance: A factor - analytic examination of the Relationship Scales Questionnaire. *New Zealand Journal of Psychology, 24*(1), 13 - 20.

Seligman, M. E. P. (1988). Competing theories of panic. In S. Rachman & J. D. Maser (Eds.), *Panic: Psychological perspectives* (pp. 321 - 329). Hillside, NJ: Erlbaum.

Senchak, M., & Leonard, K. E. (1992). Attachment styles and marital adjustment among newlywed couples. *Journal of Social and Personal Relationships, 9*, 51 - 64.

Servant, D., & Parquet, P. J. (2000). Study of diagnosis and treatment of panic disorder in psychiatry (Abstract). *Encephale, 26*(2), 33 - 37.

Shafar, S. (1976). Aspects of phobic illness: A study of ninety personal cases. *British Journal of Medical Psychology, 49*, 211 - 236.

Shaver, P. R., & Brennan, K. A (1992). Attachment styles and the "Big Five" personality traits: Their connections with each other and with romantic relationship outcomes. *Personality and Social Psychology Bulletin, 18*, 536 - 545.

Shaver, P. R., & Clark, C. (1994). The psychodynamics of adult romantic attachment. In J. M. Masling & R. E Bornstein (Eds.), *Empirical perspectives on object relations theory. Empirical studies of psychoanalytic theories* (Vol. 5, pp. 105 - 156). Washington, DC: American Psychological Association.

Shaver, P., & Hazan, C, (1993). Adult romantic attachment: Theory and evidence. In D. Perlman & W. Jones (Eds.), *Advances in personal relationships* (Vol. 4, pp. 29 - 70). London: Jessica Kingsley.

Shaver, P., Hazan, C., & Bradshaw, D. (1988). Love as attachment: The integration of three behavioural systems. In R. J. Sternberg & M. Barnes (Eds.), *The psychology of love* (pp.68 - 99).New Haven, CT: Yale University Press.

Shear, M. K. (1996). Factors in the etiology and pathogenesis of panic disorder: Revisiting the attachment - separation paradigm. *American Journal of Psychiatry*, 153(7), 125 - 136.

Shear, M. K., Cooper, A. M., Klerman, G. L., Busch, F. N., & Shapiro, T. (1993). A psychodynamic model of panic disorder. *American Journal of Psychiatry*, 150(6), 859 - 866.

Sheikh, J. I., King, R. J., & Taylor, C. B. (1991). Comparative phenomenology of early - onset versus late - onset panic attacks: A pilot survey. *American Journal of Psychiatry*, 148, 1231 - 1233.

Sheikh, J. I., Leskin, G. A., & Klein, D. F. (2002). Gender differences in panic disorder: Findings from the National Comorbidity survey. *American Journal of Psychiatry*, 159(1), 55 - 58.

Shulman, S., Elicker, J., & Sroufe, L. A. (1994). Stages of friendship growth in preadolescence as related to attachment history. *Journal of Social and Personal Relationships*, 11, 341 - 361.

Silove, D. (1986). Perceived parental characteristics and reports of early parental deprivation in agoraphobic patients. *Australian and New Zealand Journal of Psychiatry*, 20(3), 365 - 369.

Silove, D., Harris, M., Morgan, A., Boyce, P., Manicavasagar, V., Hadzi - Pavlovic, D., & Wilhelm, K. (1995). Is early separation anxiety a specific precursor of panic disorder - agoraphobia? A community study. *Psychological Medicine*, 25(2), 405 - 411.

Silove, D., & Manicavasagar, V. (2001). Early separation anxiety and its relationship to adult anxiety disorders. In M. W. Vasey and M. R. Dadds. *The developmental psychopathology of anxiety* (pp.459 - 480). New York: Oxford University Press.

Silove, D., V. Manicavasagar, Curtis, J., & Blaszczynski, A. (1996). Is early separation anxiety a risk factor for adult panic disorder? A critical review. *Comprehensive Psychiatry*, 37(3), 167 - 179.

Silove, D., Manicavasagar, V., O'Connell, D., Blaszczynski, A., Wagner, R., & Henry, J. (1993a). The development of the Separation Anxiety Symptom Inventory (SASI). *Australian & New Zealand Journal of Psychiatry*, 27, 477 - 488.

Silove, D., Manicavasagar, V., O'Connell, D., & Blaszczynski, A. (1993b). Reported early separation anxiety symptoms in patients with panic and generalised anxiety disorders. *Australian and New Zealand Journal of Psychiatry*, 27, 489 - 494.

Silove, D., Parker, G., Hadzi - Pavlovic, D., Manicavasagar, V., & Blaszczynski, A. (1991). Parental representations of patients with panic disorder and generalised anxiety disorder. *British Journal of Psychiatry*, 159, 835 - 841.

Simpson, J. A. (1990). Influence of attachment styles on romantic relationships. *Journal of Personality and Social Psychology*, 59 (5), 971 - 980.

Simpson, J. A., Rholes, W. S., & Nelligan, J. S. (1992). Support - seeking and support - giving within couple members in an anxiety - provoking situation: The role of attachment styles. *Journal of Personality & Social Psychology*, 62, 434 - 446.

Slade, A. (1987). Quality of attachment and early symbolic play. *Developmental Psychology*, 23, 78 - 85.

Snaith, R. P. (1968). A clinical investigation of phobias. *British Journal of Psychiatry*, 114, 673 - 697.

Solyom, L., Silberfeld, M., & Solyom, C. (1976). Maternal overprotection in the etiology of agoraphobia. *Canadian Psychiatric Association Journal*, 21(2), 109 - 113.

Spence, S., Shapiro, D., & Zaidel, E. (1996). The role of the right hemisphere in the physiological and cognitive components of emotional processing. *Psychophysiology*, 33, 112-122.

Spielberger, C. D. (1988). *The Professional Manual for the State - Trait Anger Expression Inventory (Rev. research ed.)*. Odessa, FL: Psychological Assessment Resources.

Sroufe, L.A. (1979). The coherence of individual development. *American Psychologist*, 34, 834 - 841.

Sroufe, L.A. (1983). Infant - caregiver attachment and patterns of adaptation in preschool: The roots of maladaptation and competence. In M. Perlmutter (Ed.), *The Minnesota Symposia on Child Psychology: Vol. 16. Development and policy concerning children with special needs* (pp. 41 - 83). Hillsdale, NJ: Erlbaum.

Sroufe, L. A. (1985). Attachment classification from the perspective of infant - caregiver relationships and infant temperament. *Child Development*, 56, 1 - 14.

Sroufe, L. A. (1990). An organizational perspective on the self. In D. Cicchetti & M. Beeghly (Eds.), *The Self in Transition* (pp. 281 - 307). Chicago: University of Chicago Press.

Sroufe, L. A. (1996). *Emotional development: The organization of emotional life in the early years*. New York: Cambridge University Press.

Sroufe, L. A. (1997). Psychopathology as outcome of development. *Development and Psychology*, 9, 251 - 268.

Sroufe, L.A. Bennett, C., Englund, M., Urban, J., & Shulman, S. (1993). The significance of gender boundaries in preadolescence: Contemporary correlates and antecedents of boundary violation and maintenance. *Child Development, 64*, 455 - 466.

Sroufe, L. A., & Fleeson, J. (1988). The coherence of individual relationships. In R. A. Hinde & J. Stevenson - Hinde (Eds.), *Relationships within families: Mutual influences* (pp. 27 - 47). Oxford: Oxford University Press.

Sroufe, L. A., Fox, N., & Pancake, V. (1983). Attachment and dependency in developmental perspective. *Child development, 54*, 1615 - 1627.

Sroufe, L. A., Carlson, E., & Shulman, S. (1993). Individuals in relationships: Development from infancy through adolescents. In D.C. Funder, R.Parke, C.Tomlinson - Keeseey & K. Widaman (Eds.), *Studying lives though time: Approaches to personality and development*, (pp. 315 - 342). Washington, DC: American Psychological Association.

Stein, M. B., Jang, K. L., Livesley, W. J. (1999). Heritability of anxiety sensitivity: A twin study. *American Journal of Psychiatry, 156*(2), 246 - 251.

Stern, D. N. (1985). *The interpersonal world of the infant*. New York: Basic Books.

Stewart, S. H., Taylor, S., Jang, K. L., Cox, B. J., Watt, M.C., Fedoroff, I. C., & Borger, S. C. (2001). Causal modeling of relations among learning history, anxiety sensitivity, and panic attacks. *Behaviour Research and Therapy, 39*(4), 443 - 456.

Stott, D. H. (1950). *Delinquency and Human Nature*. Dunfermline, Fife: Carnegie UK Trust.

Strodl, E., & Noller, P. (2003). The relationship of adult attachment dimensions to depression and agoraphobia. *Personal Relationships, 10*, 171 - 185.

Suess, G. J., Grossmann, K. E., & Sroufe, L. A. (1992). Effects of infant attachment to mother and father on quality of adaptation in preschool: From dyadic to

individual organisation of self. *International Journal of Behavioral Development*, 15, 43 - 65.

Suomi, S. J. (1997). Early determinants of behaviour: Evidence from primate studies. *British Medical Bulletin*, 53(1), 170 - 184.

Tabachnick, B. G., & Fidell, L. S. (1996). *Using Multivariate Statistics* (3<sup>rd</sup> ed.). New York: HarperCollins

Taylor, S. (1995). Anxiety sensitivity: Theoretical perspectives and recent findings. *Behaviour Research and Therapy*, 33, 243–258.

Taylor, S. (Ed.). (1999). *Anxiety sensitivity: Theory, research and treatment of fear of anxiety*. Mahwah, NJ: Erlbaum Associates.

Taylor, S., Koch, W. J., & McNally, R. J. (1992). How does anxiety sensitivity vary across the anxiety disorders? *Journal of Anxiety Disorders*, 6, 249 - 259.

Taylor, S., & Rachman, S. (1994). Klein's suffocation theory of panic. *Archives of General Psychiatry*, 51, 505 - 506.

Tennant, C. (1988). Parental loss in childhood. *Archives of General Psychiatry*, 45(11), 1045 - 1050.

Tennant, C. (2002). Life events, stress and depression: a review of recent findings. *Australian and New Zealand Journal of Psychiatry*, 36(2), 173.

Telch, M. J., & Harrington, P. J. (1992). The role of anxiety sensitivity and expectedness of arousal in mediating affective responses to 35% CO<sub>2</sub>. Paper presented at the Association for Advancement of Behavior Therapy, Boston, MA.

Telch, M.J, Lucas, J.A., & Nelson, P. (1989). Nonclinical panic in college students: An investigation of prevalence and symptomatology. *Journal of Abnormal Psychology*, 98, 300 - 306.

Telch, M.J, Shermis, M.D., & Lucas, J.A. (1989). Anxiety sensitivity: Unitary personality trait or domain specific appraisals. *Journal of Anxiety Disorders*, 3, 25 - 32

Terhune, W. B. (1949). The phobic syndrome: A study of 86 patients with phobic reactions. *Archives of Neurology and Psychiatry*, 62, 162 - 172.

Teti, D. M., Sakin. J., Kucera, E., Corns, K. M., & Das Eisen, R. (1996). And baby makes four: Predictors of attachment security among preschool –aged firstborns during the transition to siblinghood. *Child Development*, 67, 579 - 596.

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. In N. A. Fox (Ed.), *The development of emotion regulation and dysregulation: Biological and behavioral aspects. Monographs of the Society for Research in Child Development*, 59, 25 - 52.

Thompson, R. A. (1999). Early attachment and later development. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research and clinical applications* (pp 265 - 286). New York: Guilford Press.

Thompson, R. A. (2001). Childhood anxiety disorders from the perspective of emotion regulation and attachment. In M. W. Vasey & M. R. Dadds (Eds.), *The developmental psychopathology of anxiety*. New York: Oxford University Press.

Thompson, R. A., & Calkins, S. (1996). The double - edged sword: Emotion regulation for children at risk. *Development and Psychopathology*, 8, 163 - 182.

Thyer, B. A., & Himle, J. (1985). Temporal relationship between panic attack onset and phobic avoidance in agoraphobia. *Behaviour Research and Therapy*, 23, 607 - 608.

Thyer, B. A., Himle, J., & Fischer, D. (1988). Is parental death a selective precursor to either panic disorder or agoraphobia? A test of the separation anxiety hypothesis. *Journal of Anxiety Disorders*, 2(4), 333 - 338.

- Thyer, B. A., Nesse, R. M., Cameron, O. G., & Curtis, G. C. (1985). Agoraphobia: A test of the separation anxiety hypothesis. *Behaviour Research & Therapy*, 23, 75 – 78.
- Thyer, B. A., Nesse, R. M., Curtis, G. C., & Cameron, O. G. (1986). Panic disorder: A test of the separation anxiety hypothesis. *Behaviour Research & Therapy*, 24, 209 – 211.
- Torgersen, S. (1979). The nature and origin of common phobic fear. *British Journal of Psychiatry*, 134, 343 - 351.
- Torgersen, S. (1983). Genetic factors in anxiety disorders. *Archives of General Psychiatry*, 40, 1085 - 1089.
- Torgersen, S. (1990). Comorbidity of major depression and anxiety disorders in twin pairs. *American Journal of Psychiatry*, 147, 1199 - 1202.
- Tucker, W. I. (1956). Diagnosis and treatment of the phobic reaction. *American Journal of Psychiatry*, 112, 825 - 830.
- Tweed, J. L., Schoenbach, V. J., George, L. K., Blazer, D. G. (1989). The effects of childhood parental death and divorce on six - month history of anxiety disorders. *British Journal of Psychiatry*, 154, 823 - 828.
- Uhde, T. W., Roy - Byrne, P. P., Vittone, B. J., Boulenger, J. - P., & Post, R. M. (1985). Phenomenology and neurobiology of panic disorder. In A. H. Tuma, and J. D. Maser (Eds.), *Anxiety and the Anxiety Disorders*. Hillsdale, NJ: Erlbaum, pp. 557 - 576.
- Urban, J., Carlson, E., Egeland, B., & Sroufe, L. A. Patterns of individual adaptation across childhood. *Development & Psychopathology*, 3(4), 445 - 460.
- van Beek, N., & Griez, E (2003). Anxiety sensitivity in first - degree relatives of patients with panic disorder. *Behaviour Research & Therapy*, 41(8), 949 - 957.

van den Boom, D. (1990). Preventive intervention and the quality of mother-infant interaction and infant exploration in irritable infants. In W. Koops (Ed.), *Developmental psychology behind the dikes* (pp. 249 - 270). Amsterdam: Eburon.

van den Hout, M. A., van der Molen, G. M., Griez, E., & Lousberg, H. (1987). Reduction of CO<sub>2</sub> - induced anxiety in patients with panic attacks after repeated CO<sub>2</sub> exposure. *American Journal of Psychiatry*, *144*, 788 - 791.

van den Heuvel, O. A., van de Wetering, B. J. M., Veltman, D. J., & Pauls, D. L. (2000). Genetic studies of panic disorder: A review. *Journal of Clinical Psychiatry*, *61*(10), 756 - 766.

Vasey, M. W., & Dadds, M. R. (2001). An introduction to the developmental psychopathology of anxiety. In M. W. Vasey & M. R. Dadds (Eds.), *The developmental psychopathology of anxiety*. New York: Oxford University Press.

Vaughn, B. E., & Bost, K. K. (1999). Attachment and temperament: Redundant, independent, or interacting influences on interpersonal adaptation and personality development? In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research and clinical applications* (pp. 198 - 225). New York; Guilford Press.

Waddington, C. H. (1957). *The Strategy of Genes*. Ondon: Allen & Unwin.

Warren, S. L., Gunnar, M. R., Kagan, J., Anders, T. F., Simmens, S. J., Rones, M., Wease, S., Aron, E., Dahl, R. E., & Srouff, L. A. (2003). Maternal panic disorder: Infant temperament, neurophysiology, and parenting behaviors. *Journal of the American Academy of Child and Adolescent Psychiatry*, *42*(7), 814 - 825.

Warren, S.L., Huston, L., Egeland, B., & Sroufe, L. A. (1997). Child and adolescent anxiety disorders and early attachment. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*(5), 637 - 644.

Warren, R., & Zgourides, G. (1988). Panic attacks in high school students: Implications for prevention and intervention. *Phobia Practice and Research Journal*, *1*, 197 - 113.

Waters, E., Merrick, S., Treboux, D., Crowell, J., & Albersheim, L. (2000). Attachment Security in Infancy and Early Adulthood: A Twenty - Year Longitudinal Study. *Child Development, 71*(3), 684 - 689.

Watt, M.C., & Stewart, S.H. (2000). Anxiety sensitivity mediates the relationships between learning experiences and elevated concerns in young adulthood. *Journal of Psychosomatic Research, 49* (2), 107 - 118.

Watt, M. C., & Stewart, S. H. (2003). The role of anxiety sensitivity components in mediating the relationship between childhood exposure to parental dyscontrol and adult anxiety symptoms. *Journal of Psychopathology & Behavioral Assessment , 25*(3), 167 - 176.

Watt, M.C., Stewart, S.H., & Cox, B.J. (1998). A retrospective study of the learning history origins of anxiety sensitivity. *Behaviour Research & Therapy, 36*, 505 - 525.

Webster, A. S. (1953). The development of phobias in married women. *Psychological Monographs, 67*, 267.

Weekes, C. (1978). Simple, effective treatment of agoraphobia. *American Journal of Psychotherapy, 32*, 357 - 369.

Weinfield, N. S., Ogawa, J. R., & Sroufe, L. A. (1997). Early attachment as a pathway to adolescent peer competence. *Journal of Research on Adolescence, 7*, 241 - 265.

Weinfield, N. S., Sroufe, L. A., Egeland, B., & Carlson, E. A. (1999). The nature of individual differences in infant - caregiver attachment. In J. Cassidy & P. R. Shaver. *Handbook of attachment: Theory, research and clinical applications* (pp. 68 - 88). New York: Guilford Press.

Weinfield, N. S., Sroufe, L. A., & Egeland, B. (2000). Attachment from Infancy to Early Adulthood in a High - Risk Sample: Continuity, Discontinuity, and Their Correlates. *Child Development, 71*(3), 695 - 702.

Weissman, M. M., Canino, G. J., Greenwald, S., & Joyce, P. R. (1995). Current rates and symptom profiles of panic disorder in six cross - national studies. *Clinical - Neuropharmacology*; 18(2): S1 - S6.

Weissman, M. M., Leckman, J. F., Merikangas, K. R., Gammon, G. D., & Prusoff, B. A. (1984). Depression and anxiety disorders in parents and children: Results from the Yale Family Study. *Archives of General Psychiatry*, 41, 845 - 852.

Wiborg, I. M., & Dahl, A. A. (1997). The recollection of parental rearing styles in patients with panic disorder. *Acta Psychiatrica Scandinavica*, 96, 58 - 63.

Wittchen, H. U., & Essau, C. A. (1993). Epidemiology of panic disorder: Progress and unresolved issues. *Journal of Psychiatric Research*, 27(1), 47 - 68.

Woods, S. W., Charney, D. S., Goodman, W. K., & Heninger, G. R. (1988). Carbon dioxide - induced anxiety: Behavioral, physiologic, and biochemical effects of carbon dioxide in patients with panic disorders and healthy subjects. *Archives of General Psychiatry*, 45, 43 - 52.

Wolpe, J., & Rowan, V. C. (1988). Panic disorder: A product of classical conditioning. *Behaviour Research and Therapy*, 26, 441 - 450.

World Health Organisation (1996). *Mental health Fact Sheet No. 130*. Geneva, : Author

Yamada, H., Sadato, N., Konishi, Y., Muramoto, S., Kimura, K., Tanaka, M., Yonekura, Y., Ishii, Y., & Itoh, H. (2000). A milestone for normal development of the infantile brain detected by functional MRI. *Neurology*, 55, 218-223.

Zandbergen, J., Pols, H., de Loof, C., & Griez, E. J. L. (1991). Ventilatory response to CO<sub>2</sub> in panic disorder. *Psychiatry Research*, 39, 13 - 19.

Zandbergen, van Aalst, V., de Loof, C., Pols, H., & Griez, E. (1993). No chronic hyperventilation in panic disorder patients. *Psychiatry Research*, 47, 1 - 6.

Zinbarg, R.E., Brown, T.A., Barlow, D.H., & Rapee, R.M. (2001). Anxiety sensitivity, panic, and depressed mood: A reanalysis teasing apart the contributions of

the two levels in the hierarchical structure of the Anxiety Sensitivity Index. *Journal of Abnormal Psychology*, 110, 372 - 377.

Zinbarg, R. E., Mohlman, J., & Hong, N. N. (1999). Dimensions of anxiety sensitivity. In S. Taylor (Ed.), *Anxiety sensitivity: Theory, research, and treatment of the fear of anxiety* (pp. 83–113). Mahwah, NJ: Erlbaum.

Zuroff, D.C., & Fitzpatrick, D.K. (1995). Depressive personality styles: Implications for adult attachment. *Personality and Individual Differences*, 18, 253 - 365.

Zvolensky, M. J., & Eifert, G. H. (2001). A review of psychological factors/processes affecting anxious responding during voluntary hyperventilation and inhalations of carbon - dioxide enriched air. *Clinical Psychology Review*, 21(3), 375 - 400.

Zvolensky, M. J., Eifert, G. H., & Lejuez, C. W. (2000). Assessing the perceived predictability of anxiety - related events: A report on the Perceived Predictability Index. *Journal of Behavior Therapy & Experimental Psychiatry*, 31(3 - 4), 201 - 218.

Zvolensky, M. J., Eifert, G. H., & Lejuez, C. W. (2001). Offset control during recurrent 20% carbon dioxide - enriched air induction: Relation to individual difference variables. *Emotion*, 1 (2), 148 - 165.

Zvolensky, M.J., Feldner, M.T., Eifert, G.H., & Stewart, S.H. (2001). Evaluating differential predictions of emotional reactivity during repeated 20% carbon dioxide - enriched air challenge. *Cognitive Emotion*, 15, 767 - 786.

Zvolensky, M. J., Lejuez, C. W., & Eifert, G. H. (1998). The role of offset control in anxious responding: An experimental test using repeated administrations of 20% carbon dioxide - enriched air. *Behavior Therapy*, 29(2), 193 - 209.

Zvolensky, M. J., Lejuez, C. W., & Eifert, G. H. (2000). Prediction and control: Operational definitions for the experimental analysis of anxiety. *Behaviour Research and Therapy*, 38, 653 - 663.

Appendix A  
Self-Report Survey



## ***demographic information***

This section of the questionnaire asks some basic questions about you and your history. Please answer all questions in the spaces provided, unless directed to move on. Thank you.

### **PART ONE: General demographics**

1. Date of Birth: ..... / ..... / 19.....                      Age: .....years
2. Gender:                       Male                       Female

### **PART TWO: Panic & Agoraphobia**

#### **PANIC**

Please read the following:

A PANIC ATTACK is a discrete period of **very intense fear** or discomfort. It is often accompanied by an inexplicable sense of imminent danger or impending doom, and an urge to escape. A panic attack has a **sudden onset** and builds rapidly to a peak, usually within 10 minutes.

3. Have you ever had a panic attack (as defined above)?  
 Yes                       No (go to next page - Q. 10 – Agoraphobia)
4. A panic attack may be accompanied by any of the following symptoms. Please place a tick in the checkbox beside those symptoms (below) which you have experienced during a typical panic attack.
- |   |   |
|---|---|
| <input type="checkbox"/> heart pounding or racing                             | <input type="checkbox"/> heart palpitations                             |
| <input type="checkbox"/> trembling or shaking                                 | <input type="checkbox"/> sweating                                       |
| <input type="checkbox"/> feeling of choking                                   | <input type="checkbox"/> shortness of breath or smothering              |
| <input type="checkbox"/> nausea or abdominal distress                         | <input type="checkbox"/> chest pain or discomfort                       |
| <input type="checkbox"/> chills or hot flushes                                | <input type="checkbox"/> numbness or tingling sensations                |
| <input type="checkbox"/> fear of dying  | <input type="checkbox"/> fear of losing control or going crazy          |
| <input type="checkbox"/> feelings of unreality or being detached from oneself | <input type="checkbox"/> feeling dizzy, unsteady, light-headed or faint |

5. In what month and year did you have your first panic attack?  
 a) Month:..... b) Year:..... c) How old were you?:.....  
 d) If triggered by particular surrounding circumstances, what were they?.....  
 .....
6. How would you rate the general severity of your panic attacks?  
 Mild       Moderate       Severe       Extreme
7. To what extent have (were) your panic attacks been disruptive of your life activities?  
 Not at all       Moderately       Severely       Extremely
8. Please indicate how many panic attacks you have had:  
 a) in the last week..... b) in the last month.....
9. In the time since your first panic attack, have you at times experienced persistent worry about having further panic attacks?.....

## AGORAPHOBIA

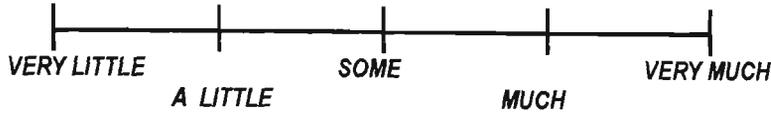
10. To what extent have you avoided situations or places because you became anxious?  
 Never       Rarely       Sometimes       Often       Always
11. To what extent have you avoided situations or places because you were afraid that you might have a panic attack?  
 Never       Rarely       Sometimes       Often       Always
12. To what extent have you made use of the company of a familiar person, in order to feel able to attend situations or places you would have otherwise avoided if you were alone?  
 Never       Rarely       Sometimes       Often       Always

## OTHER

Have you ever received a diagnosis by a psychiatrist or psychologist (for example - depression, generalized anxiety disorder, bipolar disorder, panic disorder)? If more than one, please specify all.....  
 .....

# asi

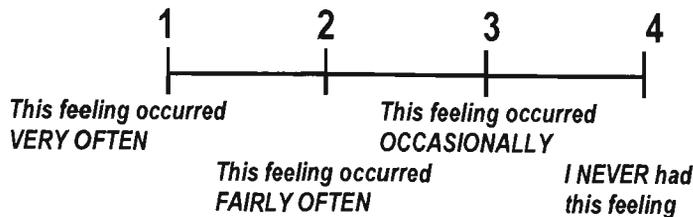
Please indicate to what extent the following statements are true for you, by CIRCLING the number which best represents your answer, where "0" refers to "very little" and "4" refers to "very much". For example, in question 1, if it is very important to you to not appear nervous to others, you would circle "4" (ie. "very much") for the first statement below.



1. It is important to me not to appear nervous ..... 0 1 2 3 4
2. When I cannot keep my mind on task, I worry that I might be going crazy ..... 0 1 2 3 4
3. It scares me when I feel 'shaky' (trembling) ..... 0 1 2 3 4
4. It scares me when I feel faint ..... 0 1 2 3 4
5. It is important to me to stay in control of my emotions ..... 0 1 2 3 4
6. It scares me when my heart beats rapidly ..... 0 1 2 3 4
7. It embarrasses me when my stomach growls ..... 0 1 2 3 4
8. It scares me when I am nauseous ..... 0 1 2 3 4
9. When I notice that my heart is beating rapidly, I worry that I might have a heart attack ..... 0 1 2 3 4
10. It scares me when I become short of breath ..... 0 1 2 3 4
11. When my stomach is upset, I worry that I might be seriously ill .. 0 1 2 3 4
12. It scares me when I am unable to keep my mind on a task ..... 0 1 2 3 4
13. Other people notice when I feel shaky ..... 0 1 2 3 4
14. Unusual body sensations scare me ..... 0 1 2 3 4
15. When I am nervous, I worry that I might be mentally ill ..... 0 1 2 3 4
16. It scares me when I am nervous ..... 0 1 2 3 4

# *sasi*

The following statements refer to fears you might have had in early life. Please CIRCLE the appropriate number for each item, according to your memories before 16 years, where 1 refers to "this feeling occurred very often" and 4 refers to "I never had this feeling". Note that, in contrast to previous pages, "1" now refers to the most frequent category and "4" to the least. Please remember to answer all questions.



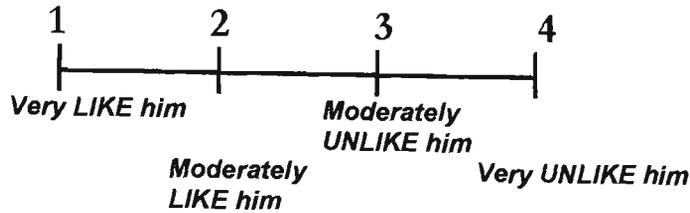
When I was a child...

- |   |   |   |   |   |
|---|---|---|---|---|
| 1. I did not want to go to school. ....   | 1 | 2 | 3 | 4 |
| 2. I feared that one of my parents might come to harm when I was away from home. ....             | 1 | 2 | 3 | 4 |
| 3. I did not want to be left alone at home. ....  | 1 | 2 | 3 | 4 |
| 4. I had physical symptoms like stomach aches, nausea, headaches, before going to school. ....    | 1 | 2 | 3 | 4 |
| 5. I had fears that accidents might happen to members of my family when I was not with them. .... | 1 | 2 | 3 | 4 |
| 6. I was afraid of getting lost when I was in strange places. ....                                | 1 | 2 | 3 | 4 |
| 7. I imagined that monsters or animals might attack me when I was alone at night. ....            | 1 | 2 | 3 | 4 |
| 8. I was very afraid of strangers when I was on my own. ....                                      | 1 | 2 | 3 | 4 |
| 9. I had nightmares about violence towards me or my family. ....                                  | 1 | 2 | 3 | 4 |
| 10. I was very unhappy if I was separated from my family. ....                                    | 1 | 2 | 3 | 4 |
| 11. I was afraid of being harmed or kidnapped when I was alone. ....                              | 1 | 2 | 3 | 4 |
| 12. I daydreamed about being with my family when I was away from home. ....                       | 1 | 2 | 3 | 4 |
| 13. I was afraid to go to sleep alone. ....   | 1 | 2 | 3 | 4 |
| 14. I was very tense before going to school. ....   | 1 | 2 | 3 | 4 |
| 15. I was afraid of the dark. ....  | 1 | 2 | 3 | 4 |



*pbi – father*

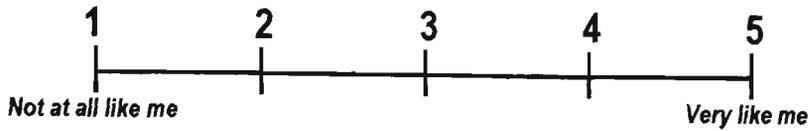
For the following statements, please CIRCLE the number which best represents your **FATHER** (or step-father, if he was the primary male caregiver) as you remember him in your first 16 years of life.



- |   |   |   |   |   |
|---|---|---|---|---|
| 1. Spoke to me with a warm and friendly voice               | 1 | 2 | 3 | 4 |
| 2. Did not help me as much as I needed                      | 1 | 2 | 3 | 4 |
| 3. Let me do those things I liked doing                     | 1 | 2 | 3 | 4 |
| 4. Seemed emotionally cold to me                            | 1 | 2 | 3 | 4 |
| 5. Appeared to understand my problems and worries           | 1 | 2 | 3 | 4 |
| 6. Was affectionate to me                                   | 1 | 2 | 3 | 4 |
| 7. Liked me to make my own decisions                        | 1 | 2 | 3 | 4 |
| 8. Did not want me to grow up                               | 1 | 2 | 3 | 4 |
| 9. Tried to control everything I did                        | 1 | 2 | 3 | 4 |
| 10. Invaded my privacy                                      | 1 | 2 | 3 | 4 |
| 11. Enjoyed talking things over with me                     | 1 | 2 | 3 | 4 |
| 12. Frequently smiled at me                                 | 1 | 2 | 3 | 4 |
| 13. Tended to baby me                                       | 1 | 2 | 3 | 4 |
| 14. Did not seem to understand what I needed or wanted      | 1 | 2 | 3 | 4 |
| 15. Let me decide things for myself                         | 1 | 2 | 3 | 4 |
| 16. Made me feel I wasn't wanted                            | 1 | 2 | 3 | 4 |
| 17. Could make me feel better when I was upset              | 1 | 2 | 3 | 4 |
| 18. Did not talk with me very much                          | 1 | 2 | 3 | 4 |
| 19. Tried to make me dependent on him                       | 1 | 2 | 3 | 4 |
| 20. Felt I could not look after myself unless he was around | 1 | 2 | 3 | 4 |
| 21. Gave me as much freedom as I wanted                     | 1 | 2 | 3 | 4 |
| 22. Let me go out as often as I wanted                      | 1 | 2 | 3 | 4 |
| 23. Was overprotective of me                                | 1 | 2 | 3 | 4 |
| 24. Did not praise me                                       | 1 | 2 | 3 | 4 |
| 25. Let me dress in any way I pleased                       | 1 | 2 | 3 | 4 |

## *relationships*

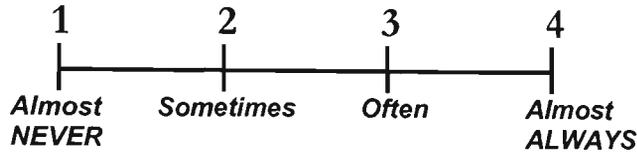
The following page is a questionnaire about relationships. Think about all your relationships, past and present, and respond in terms of how you generally feel in those relationships, where 1 refers to "not at all like me" and 5 is "very like me". Please CIRCLE the number which best represents your answer.



- |  |   |   |   |   |   |
|--|---|---|---|---|---|
| 1. I find it difficult to depend on other people.....  | 1 | 2 | 3 | 4 | 5 |
| 2. It is very important to me to feel independent.....   | 1 | 2 | 3 | 4 | 5 |
| 3. I find it easy to get emotionally close to others.....  | 1 | 2 | 3 | 4 | 5 |
| 4. I want to merge completely with another person.....   | 1 | 2 | 3 | 4 | 5 |
| 5. I worry that I will be hurt if I allow myself to become too close to others.....                | 1 | 2 | 3 | 4 | 5 |
| 6. I am comfortable without close emotional relationships.....                                     | 1 | 2 | 3 | 4 | 5 |
| 7. I am not sure that I can always depend on others to be there when I need them.....              | 1 | 2 | 3 | 4 | 5 |
| 8. I want to be completely emotionally intimate with others.....                                   | 1 | 2 | 3 | 4 | 5 |
| 9. I worry about being alone.....  | 1 | 2 | 3 | 4 | 5 |
| 10. I am comfortable depending on other people.....  | 1 | 2 | 3 | 4 | 5 |
| 11. I often worry that others don't really love me.....  | 1 | 2 | 3 | 4 | 5 |
| 12. I find it difficult to trust others completely.....  | 1 | 2 | 3 | 4 | 5 |
| 13. I worry about others getting too close to me.....  | 1 | 2 | 3 | 4 | 5 |
| 14. I want emotionally close relationships.....  | 1 | 2 | 3 | 4 | 5 |
| 15. I am comfortable having other people depend on me.....   | 1 | 2 | 3 | 4 | 5 |
| 16. I worry that others don't value me as much as I value them.....                                | 1 | 2 | 3 | 4 | 5 |
| 17. People are never there when you need them.....   | 1 | 2 | 3 | 4 | 5 |
| 18. My desire to merge completely, sometimes scares people away.....                               | 1 | 2 | 3 | 4 | 5 |
| 19. It is very important to me to feel self-sufficient.....  | 1 | 2 | 3 | 4 | 5 |
| 20. I am nervous when anyone gets too close to me.....   | 1 | 2 | 3 | 4 | 5 |
| 21. I often worry that friends and/or romantic partners won't stay with me.....                    | 1 | 2 | 3 | 4 | 5 |
| 22. I prefer not to have other people depend on me.....  | 1 | 2 | 3 | 4 | 5 |
| 23. I worry about being abandoned.....   | 1 | 2 | 3 | 4 | 5 |
| 24. I am uncomfortable being close to others.....  | 1 | 2 | 3 | 4 | 5 |
| 25. I find that others are reluctant to get as close as I would like.....                          | 1 | 2 | 3 | 4 | 5 |
| 26. I prefer not to depend on others.....  | 1 | 2 | 3 | 4 | 5 |
| 27. I know that others will be there when I need them.....   | 1 | 2 | 3 | 4 | 5 |
| 28. I worry about having others not accept me.....   | 1 | 2 | 3 | 4 | 5 |
| 29. Friends and/or romantic partners often want me to be closer than I feel comfortable being..... | 1 | 2 | 3 | 4 | 5 |
| 30. I find it relatively easy to get close to others.....  | 1 | 2 | 3 | 4 | 5 |

# emotion

Everyone feels angry, even furious, from time to time, but people differ in the ways that they react when they are angry. A number of statements are listed below which people use to describe their reactions when they feel angry. Please read each statement and then circle the number that indicates how **often** you **generally** react or behave in the manner described when you are feeling angry or furious.



## When Angry or Furious...

- |  |   |   |   |   |
|--|---|---|---|---|
| 1. I control my temper.....  | 1 | 2 | 3 | 4 |
| 2. I express my anger.....   | 1 | 2 | 3 | 4 |
| 3. I keep things in.....   | 1 | 2 | 3 | 4 |
| 4. I am patient with others.....                                     | 1 | 2 | 3 | 4 |
| 5. I pout or sulk.....   | 1 | 2 | 3 | 4 |
| 6. I withdraw from people.....                                       | 1 | 2 | 3 | 4 |
| 7. I make sarcastic remarks to others.....                           | 1 | 2 | 3 | 4 |
| 8. I keep my cool.....   | 1 | 2 | 3 | 4 |
| 9. I do things like slam doors.....                                  | 1 | 2 | 3 | 4 |
| 10. I boil inside, but I don't show it.....                          | 1 | 2 | 3 | 4 |
| 11. I control my behaviour.....                                      | 1 | 2 | 3 | 4 |
| 12. I argue with others.....   | 1 | 2 | 3 | 4 |
| 13. I tend to harbor grudges that I don't tell anyone about.....     | 1 | 2 | 3 | 4 |
| 14. I strike out at whatever infuriates me.....                      | 1 | 2 | 3 | 4 |
| 15. I can stop myself from losing my temper.....                     | 1 | 2 | 3 | 4 |
| 16. I am secretly quite critical of others.....                      | 1 | 2 | 3 | 4 |
| 17. I am angrier than I am willing to admit.....                     | 1 | 2 | 3 | 4 |
| 18. I calm down faster than most people.....                         | 1 | 2 | 3 | 4 |
| 19. I say nasty things.....  | 1 | 2 | 3 | 4 |
| 20. I try to be tolerant and understanding.....                      | 1 | 2 | 3 | 4 |
| 21. I'm irritated a great deal more than people are aware of.....    | 1 | 2 | 3 | 4 |
| 22. I lose my temper.....  | 1 | 2 | 3 | 4 |
| 23. If someone annoys me, I'm apt to tell him or her how I feel..... | 1 | 2 | 3 | 4 |
| 24. I control my angry feelings.....                                 | 1 | 2 | 3 | 4 |

## Appendix B

Faculty of Arts Human Research Ethics Committee Approval

**Memorandum**

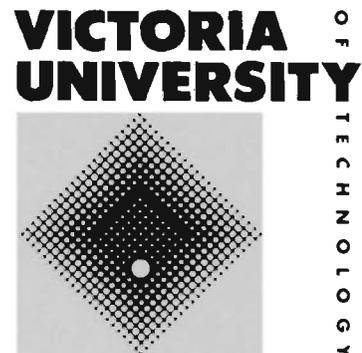
REF: ETH0204

TO: Dr Gerard Kennedy  
Dept of Psychology  
St Albans campus

FROM: A/Professor Ross Williams  
Chair, Faculty of Arts  
Human Research Ethics committee

DATE: 23 February 2000

SUBJECT: ***HRETH.FOA.0003/00 involving human subjects***



---

The Faculty of Arts Human Research Ethics Committee at its meeting on 18 February 2000 considered application for project:

***Towards an Empirical Model for the Development of Anxiety and Panic Disorder***

It was **resolved** to **approve** application HRETH.FOA.0003/00 from 6 March 2000 to 31 December 2001.

  
per **A/Professor Ross Williams**

## Appendix C

Flow chart detailing the classification hierarchy  
used to classify participants according to panic status

Refer to question 3 (“have you ever had a panic attack”) for experiences with panic attacks –

If ‘no’ → refer to avoidance behaviour (questions 10 – 12)

- if mean of 3 items  $\geq 3.67$ , or q. 12  $\geq 4$  → code “SUBPANIC”

- if not → code “NO PANIC”

If present → refer to question 4 for number of panic symptoms endorsed

- if  $< 4$  → code “SUBPANIC”

- if  $\geq 4$  → refer to question 9 for evidence of anticipatory anxiety

- if ‘yes’ → code “PANIC”

- if ‘no’ → refer to avoidance behaviour (questions 10 - 12)

- if mean of 3 items  $\geq 3.67$  or q12  $\geq 4$  → code “PANIC”

- if not → code “SUBPANIC”

*Figure 2.* The classification hierarchy used to determine membership of the ‘panic’, subclinical panic (‘subpanic’) and ‘no panic’ groups.

## Appendix D

### Information Statement for Participants

**Victoria University  
Department of Psychology**

Project Title: TOWARD AN EMPIRICAL MODEL FOR THE DEVELOPMENT OF ANXIETY AND PANIC DISORDER

Experimenter: Debbie Fooks

We would like to invite you to be a part of a study into the development of anxiety problems and panic disorder.

The project will require you to complete a questionnaire booklet that will take approximately 30 minutes to fill out. This booklet is comprised of a number of questionnaires which ask about your experience of anxiety or panic, as well as a number of other features of your history and personal experience that are thought to be linked to the development of anxiety and panic problems. These features include: your memories of your parents, stressful events that may have occurred in your life, your style of relating to others, and the ways that you manage your emotions.

Even if you do not have any problems with anxiety or nervousness, we would be interested in knowing how your experience compares with those who are more prone to nervousness or panic.

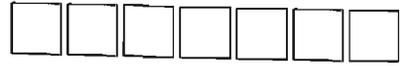
The information gathered in this project will ultimately enable the creation of a model for the development of anxiety and panic disorders which will help us to identify those individuals most at risk of developing these prevalent conditions. We hope that the questionnaire booklet will stimulate interesting self-reflection about the kinds of experiences that contributed to the person you are today. For certain individuals, however, it is possible that some of the questions posed in the booklet might produce uncomfortable memories or feelings. In the unlikely event that you become distressed by these memories or feelings, we would strongly urge you to talk with someone about your experience. The following phone numbers are provided for this purpose. \* Principal Investigator - Dr Gerard Kennedy 9365 2481, \*Student Counselling Service (St Albans campus) 9365 2399, \*VU Psychology Clinic (St Albans campus) 9365 2353.

Please note that all questionnaire booklets will be kept completely confidential. In the event that you would be interested in participating in a follow-up study to this questionnaire, provision is made for you to leave your contact details so that further information can be made available to you. Any identifying information, including contact details, will be kept separate to your questionnaire responses and will remain completely confidential, in that only the investigator and principal investigator will have access to this information.

Participation in this project is completely voluntary and you are free to withdraw your consent at any time. Furthermore, participation in this study is independent of your course, and your consent or withdrawal from the study will not jeopardise you in any way.

Any queries about your participation in this project may be directed to the Principal Investigator, Dr Gerard Kennedy on 9365 2481. If you have any queries or complaints about the way you have been treated, you may contact the Secretary, University Human Research Ethics Committee, Victoria University of Technology, PO Box 14428 MCMC, Melbourne, 8001 (telephone no: 03-9688 4710).

Appendix E  
Participant Consent Form



**Victoria University  
Department of Psychology**

**Consent To Participate In Research**

I have read and understood the information provided, and any questions that I have asked have been answered to my satisfaction.

I certify that I am at least 18 years of age and that I am voluntarily giving my consent to participate in the project entitled:

*TOWARD AN EMPIRICAL MODEL OF ANXIETY AND PANIC DISORDER*

being conducted at Victoria University by: Debbie Fooks.

I understand that I can withdraw from this experiment at any time and that this withdrawal will not jeopardise me in any way.

I have been informed that the information I provide will be kept completely confidential.

SIGNED: ..... DATE: .....

I agree to being contacted about a follow-up to this research in the future. I understand that I am not obliged to participate in any future research but would be interested in receiving more information.

Full name: (print please).....

My postal address is: .....

My phone number is: .....

My email address is: .....

I would prefer to be contacted by:  post  phone  email

SIGNED: ..... DATE: .....