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Anxiety Sensitivity and Panic-Fear in Pediatric Asthma

by

Linda Rose

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ABSTRACT

This study examined the relationship between asthma panic-fear and anxiety sensitivity in school-age children with asthma. Elevated panic-fear in young patients is known to interfere with the medical management of asthma. Elevated anxiety sensitivity is thought to play a major role in the development and maintenance of anxiety disorders, and has been shown to increase subjective distress in situations where breathing is compromised. Data were collected as the children were being treated for acute asthma and also during a follow-up session. Anxiety sensitivity emerged as a better predictor of panic-fear intensity and frequency than were measures of trait anxiety, self-ratings of dyspnea severity, asthma knowledge and self-management, or pulmonary dysfunction. Grouping the children according to anxiety sensitivity status indicated that the high anxiety sensitive subjects were similar to others in the sample in terms of objectively assessed and subjectively perceived severity of asthma during the index. However, they experienced more anxiety and subjective asthma symptomatology, as well as more frequent and intense asthma panic-fear. The results were discussed in terms of their implication for the management of pediatric asthma and for the understanding of the nature of anxiety sensitivity.

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DEDICATION

To my husband and daughter, Gary and Alyssa, with love and gratitude.

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INTRODUCTION

Asthma is a respiratory disorder, characterized by hypersensitivity of the bronchial tree and reversible, episodic attacks of airways obstruction (Kashani, Konig, Sheppard, Wilfley & Morris, 1988; Lemanek, 1990; Yellowlees & Kalucy, 1990). Its symptomatology is heterogeneous and intermittent, with attacks varying in both frequency and severity (Staudenmayer 1982; Lemanek, 1990). Currently, asthma is estimated to affect between 3 and 5 per cent of the population in the western world, and the prevalence rate is believed to be increasing (Brooks, Richards, Bailey, Martin, Windsor & Soong, 1989). It may be the most common chronic disease of childhood (Kashani et al, 1988; Fritz & Overholser, 1989), afflicting between 5 and 15 per cent of children under 15 years old (Lemanek, 1990).

The consequences of asthma can be profound and disabling, and, under some circumstances, fatal (Strunk, Mrazek, Wolfson Fuhrmann & LaBrecque, 1985; Fritz, Rubinstein & Lewiston, 1987). There has been no clear demonstration that children with asthma are at increased risk for psychopathology (Kashani et al, 1988), but nevertheless there is a clear indication that asthma can disrupt the normal course of childhood development (Staudenmayer, 1982; Jurenec, 1988). The child with asthma may miss school, be unable to

engage in some sports or other age-appropriate activities, and may have restricted opportunities for socialization with peers (Staudenmayer, 1982; Jurenec, 1988). These effects tend to be more pronounced when asthma is poorly managed (Rubin, Bauman & Rubin, 1989). In recent years, panic in the context of an asthma attack has been identified as a major impediment to effective management (Fritz & Overholser, 1989; Baron & Marcotte, 1994). Although it has long been recognized as a common response to worsening asthma, factors which may increase the risk for panic have not yet been identified.

The purpose of the present study is to examine whether anxiety sensitivity, the tendency to respond fearfully to symptoms of anxiety because of beliefs surrounding their harmful consequences (Reiss & McNally, 1985), enhances susceptibility to panic in children with asthma. Anxiety sensitivity has been widely researched in the adult population, and is held to be causally related to the development of panic disorder (Taylor, 1995). Anxiety sensitivity in children is less well studied, but does appear to be positively associated with fearfulness as well as with clinical elevations on measures of trait anxiety in younger subjects (Silverman, Fleisig, Rabian & Peterson, 1991; Rabian, Peterson, Richters & Jensen, 1993). It has been suggested that high anxiety sensitive individuals are more vulnerable

to panic because they are inclined to focus on feared inner cues (Shostak & Peterson, 1990), and there are data to support this claim (Holloway & McNally, 1987; Shostak & Peterson, 1990; Asmundsen & Norton, 1994; Rapee & Medoro, 1994). Panic in children with asthma may also reflect a relationship between high anxiety sensitivity and a reduced threshold for noticing symptoms that are a source of threat.

Bronchial Asthma - Pathophysiology, Symptoms and Treatment

The pathogenisis of asthma is thought to include immune system dysfunction (Friedman & Booth-Kewley, 1987) and heightened parasympathetic tone (Miller, 1987; Moran, 1989). Stimuli such as allergens, irritants, cold air, exercise, emotions, and infections are asthma triggers (Isenberg et al, 1992; Weiss, 1994). Exposure to a trigger will first produce an early asthma reaction (EAR), during which mediators are released into the airways and bronchoconstriction develops (Drazen et al., 1987; Weiss, 1994). The bronchial muscles contract, the bronchial tissues swell, excess mucous is secreted, and air exchange in the lung is impeded (Friedman & Booth-Kewley, 1987; Janson-Bjerklie, Kohlman-Carrieri & Hudes, 1986; Isenberg et al, 1992). These physiological changes, which occur within 30 minutes from the initial exposure and can last for up to two hours, lead to increased bronchial hyper-responsiveness (Celano and Geller, 1993) and, ultimately, to the extreme inflammation and bronchoconstriction

that are associated with the late asthma reaction (LAR). Tissue damage in the bronchial tree and the formation of mucous plugs which can completely occlude small airways also typify the LAR (Drazen et al, 1987; Weiss, 1994). The pathogenisis of asthma, in simplified form, is presented in Figure 1.

Wheezing and coughing are signs of acute asthma. Dyspnea, the sensation of difficult or laboured breathing, is its cardinal symptom. Adult patients with dyspnea report sensations of tightness or constriction in the chest. Furthermore, they describe feeling as if being drowned or suffocated, or as though their lungs are closing or filling up (Janson-Bjerklie et al., 1986). Children with asthma tend to use similar descriptors but also report "fighting" to get breath in and out (Kohlman Carrieri, Kieckhefer, Janson-Bjerklie & Souza, 1991).

The physiologic substrate of dyspnea is presumed to include increased inspiratory muscle activation and the stimulation of sensory receptors in the airways, lung, chest wall and diaphragm (Steele & Shaver, 1992), as well as respiratory muscle fatigue (Killian & Campbell, 1985). Studies of asthmatic patients with histamine-induced respiratory distress have indicated that whereas the diaphragmatic muscles become less involved in the breathing process, the inspiratory muscles of the chest remain involved throughout the

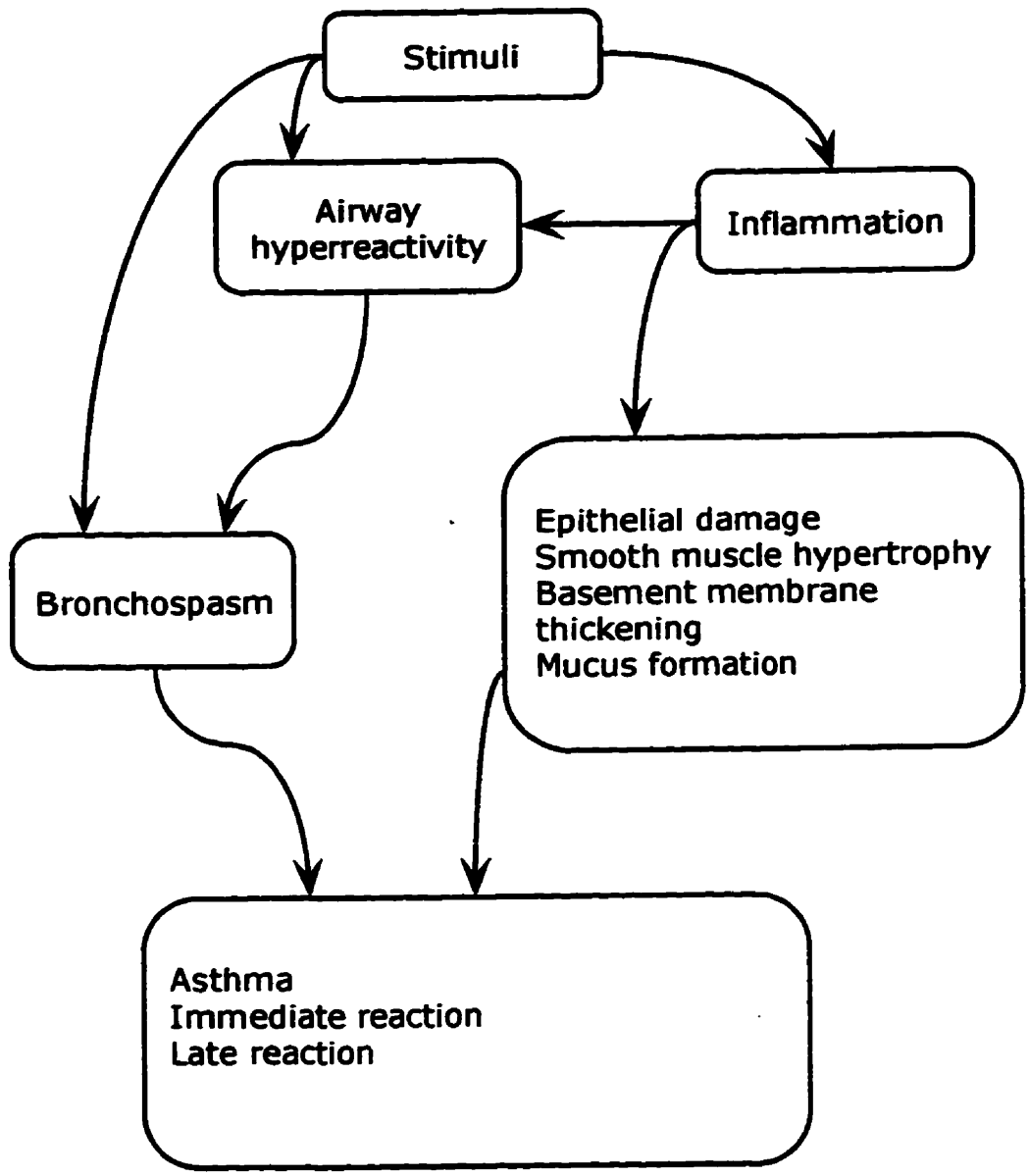

Figure 1.
The Pathogenisis of Asthma
(adapted from Creer and Bender, 1995)

entire respiratory cycle. In addition, the accessory muscles of the chest and neck appear to play a more prominent role in respiration (Martin, Powell, Shore & Engel, 1980; Martin, Shore & Engel, 1983). Clinical observation of patients with naturally occurring bronchoconstriction have also documented an increased involvement of the accessory muscles of the chest and neck during respiration (Gift, 1991). Pronounced respiratory changes, including rapid, shallow or irregular breathing (Gift, 1991; Steele & Shaver, 1992) and thoraco-abdominal asynchrony, a breathing pattern wherein the motion of the ribcage lags behind that of the abdomen (Hillman, Prentice & Finucaine, 1986), have been noted by some researchers; others, however, have reported no observable differences in terms of rate or depth of respiration (Gift & Cahill, 1990).

The major categories of medication used in the treatment of asthma include bronchodilators, sodium cromoglycate, and oral or inhaled corticosteroids. Bronchodilators (such as theophylline or salbutamol) are usually prescribed on an as-needed (PRN) basis (Renne & Creer, 1985), but are also the first line of defense in the emergent care setting (Galant, 1987). They may be delivered via a nebulizer or metered dose inhaler and work to relax the tightness of the bronchial muscles. They have a stimulant effect and high dosages can cause tremors and anxiety. Sodium cromoglycate, an inhaled

agent, inhibits both the EAR and LAR (Naspitz & Tinkelman, 1987). It serves to prevent allergens from triggering bronchospasm, potentiates the effect of bronchodilators, and is effective in approximately 60 per cent of cases. It is prophylactic, and should therefore not be administered during the acute asthma episode. Inhaled corticosteroids, for example beclomethasone, also enhance the efficiency of bronchodilators and can reduce swelling in the bronchial tree. They offer a superior response rate to that of sodium cromoglycate and are designed to be taken daily as a preventive medication (American Institute for Research, 1984). The anti-inflammatory properties of oral corticosteroids make these drugs useful in the short term after a severe asthma exacerbation (Galant, 1987); long term usage tends to be reserved for the 1 to 10 per cent of cases in which there is an inadequate response to inhaled medications (Naspitz & Tinkelman, 1987).

Although modern medication regimes should allow for asthma to be controlled in all but the most severe instances, control remains out of reach for many asthmatics (McNabb, Wilson-Pessano & Jacobs, 1986). Indeed, recent figures suggest that deaths from asthma are on the rise, as is the number of patients requiring inpatient care (Brooks et al., 1989). Non-compliance with physicians' recommendations is quite common in asthma (Lemanek, 1990) and

undoubtedly contributes to poor asthma control. Techniques designed to improve compliance, and thus control, include behavioural contracting, relaxation and biofeedback (Creer, 1991). Psychoeducational programmes which combine information and training with regards to asthma and its management, with family, group, or individual psychotherapy as required, have also been used in the attempt to improve compliance with medication regimes. Outcome research into the efficacy of adjunct therapies has tended to yield unfavourable findings, however little attention has been paid to identifying those patients most likely to benefit from such treatments (Lehrer, Sargunaraj & Hochron, 1992).

Issues in Asthma Management

The literature has identified a number of variables that may be associated with a less than satisfactory response to the medical treatment of asthma. In the 1930's the apparent success of a technique called "parentectomy," which entailed removing the afflicted child from the presumedly "toxic" influence of his or her parents, established family dysfunction as an impediment to the effective management of asthma (Renne & Creer, 1985). These findings were corroborated in the 1950's and 1960's by Purcell and his colleagues. When children were sub-grouped according to whether emotional or physical precipitants were the dominant asthma triggers

the former group, the "steroid dependents" quickly experienced symptom remission when removed from their comparatively more punitive and authoritarian parents (Purcell, Bernstein & Bukantz, 1961; Purcell, 1963; Purcell et al., 1969; cited in Renne & Creer, 1985 and Jurenec, 1988).

With the advent of more rigorous diagnostic criteria and modern medications the success rate of "parentectomy" declined dramatically, and its efficacy as treatment for asthma has now been called into question (Renne & Creer, 1985). Nevertheless, the relationship between family factors and asthma control has continued to attract considerable research interest over the past several decades. Minuchin and his associates (Minuchin, Baker, Rosman, Liebman, Milman & Todd, 1975) hypothesized that family conflict was related to intractable asthma. They argued that the child's condition served a homeostatic function within the family. As family conflict escalated so too did the child's symptomatology. The increased symptomatology then allowed for attention to be deflected away from the source of conflict and towards the asthmatic child, thus restoring family equilibrium. Minuchin and his co-workers noted that these so-called "psychosomatic families" were more rigid and enmeshed than were healthy families. They further noted that family therapy was effective

in altering family structure and improving asthma control (Minuchin et al., 1975).

Rigid families are those in which there are inflexible rules of conduct; enmeshed families are characterized by parental over-involvement and over-protectiveness. Both rigidity and enmeshment have been documented in more recent investigations of children with asthma and their families. Gustaffson, Kjellman, Ludvigsson and Cederbland (1987) observed that families of children with asthma, as compared to those of healthy children or children with diabetes, were more likely to be rated as rigid or enmeshed. In this same study family functioning and peak expiratory flow rate, a measure of airways obstruction, were inversely correlated, suggesting greater respiratory distress in those asthmatic children who lived in a more disturbed family environment. DiBlasio, Molinan, Peri and Taverna (1990) compared decision-making processes in families of children with mild asthma versus families where no chronic disease was present. They found that rigid or enmeshed interactions were more prevalent in the families of asthmatic children.

Although family dynamics were not specifically examined, a study conducted by Carson and Schauer (1992) also yielded results suggesting that enmeshed interaction patterns characterize at least some parents and children with asthma. They noted that some

mothers of children with asthma were more over-indulgent and over-protective, and therefore more enmeshed with their offspring, than were mothers of children without a chronic disease. Others, however, appeared more rejecting. Finally, Baron and his colleagues (Baron, Veilleux & Lamarre, 1992) documented a relationship between family enmeshment or rigidity and anxiety in children with asthma. Highly anxious children, who were more often prescribed oral steroid medications than were their less anxious counterparts, tended to come from rigid and enmeshed families. Low anxious children, who were frequently non-compliant with medication regimes, were equally as likely to be found in rigid and enmeshed or chaotic and disengaged families.

The literature, then, does support the notion that family factors can influence the medical management of asthma. Individual differences, however, are also held to be important in this regard. The ability to detect alterations in pulmonary functioning would seem to be a necessary prerequisite for good asthma control (Fritz, Klein & Overholser, 1990; Barnes, 1992), in that subjective assessments of dyspnea tend to be interpreted by patients and physicians alike as a marker of disease severity (Rubinfeld & Pain, 1976; Peiffer, Marsac & Lockhart, 1989). Yet it is well-known that many patients with asthma either overestimate or underestimate the degree to which airways are

occluded (Rubinfeld & Pain, 1976; Burdon, 1982; Barnes 1992). Hence there is often little correspondence between subjective and objective assessments of airways obstruction (Gift, 1990; Gift & Cahill, 1991). Researchers have examined the perception of bronchoconstriction in asthma in the natural setting as well as by such means as metacholine challenge. They have hitherto been unable to differentiate "good" and "poor" perceivers of asthma on the basis of disease or demographic characteristics such as bronchial hyper-reactivity, age or gender (Rubinfeld & Pain, 1976; Peiffer, Marsac & Lockhart, 1989; Fritz, Klein & Overholser, 1990; Kendricks, Higgs, Whitfield & Lazlo, 1993).

Dyspnea, the central symptom of acute asthma, is a complex psychobiological phenomenon and, as such, is influenced by affective and motivational factors (Steele & Shaver, 1992). Although negative emotional states such as panic, depression or anger are known to accompany dyspnea (Kohlman-Carrieri et al., 1991; Steele & Shaver, 1992), psychologic variables have attracted surprisingly little research attention in the area of the perception of bronchoconstriction in asthma. The limited evidence that exists, however, suggests that the under-reporting of asthma symptoms may be linked to a repressive defense style. Repressors show signs of autonomic arousal in stressful circumstances but report little or no anxiety (Steiner Higgs, Fritz,

Lazlo & Harvey, 1987). Repressive tendencies have been observed in children with asthma (Fritz, Spiroto & Yeung, 1994) as well as in adult patients (Steiner et al., 1987). Overestimation, on the other hand, seems to be associated with high anxiety and a hypersensitivity to changes in arousal level (Heim, Blaser & Waidelich, 1972; Steiner et al., 1987). Repression and high anxiety have both been linked to the mismanagement of asthma. In the case of repression, there is reason to believe that this may put patients at risk for death from asthma (Yellowlees & Ruffin, 1989). As for high anxiety, it too may be predictive of asthma mortality (Mascia et al., 1989). In addition, it may be related to the overuse of PRN medications (Yellowlees & Kalucy, 1990), and to asthma exacerbations of sufficient severity to warrant hospitalization (van der Schoot & Kaptein, 1990).

Panic-Fear in Asthma

On the surface, family dysfunction and the perception of bronchoconstriction in asthma appear to be unrelated topics. Yet both of these lines of research point to a connection between asthma and anxiety. In the case of the former, a direct association between family dynamics and anxiety levels has been demonstrated (Baron et al., 1992). In the latter, inappropriately low or high levels of anxiety have been shown to influence the subjective appraisal of asthma symptomatology (Heim et al., 1972; Steiner et al., 1987). The

connection between anxiety and respiratory symptom reporting has been corroborated by other sources. The results of two recent population-based studies suggest that anxiety may make ventilatory changes more salient. For example, a survey conducted by Dales, Spitzer, Schechter and Suissa (1988) revealed that anxiety was strongly and positively related to dyspnea intensity and several other symptoms of respiratory distress. The results could not be explained in terms of differences in pulmonary functioning. Similar findings were reported in an independent investigation which sampled patients with asthma as well as healthy subjects (Janson, Bjornsson, Hetta & Boman, 1994).

Anxiety has now come to be viewed as the key variable in the literature on psychologic factors in asthma (Maes & Schlosser, 1988). Indeed, as breathing is central to our physical survival, anxiety could be thought of as a "normal" response to the struggle for breath that typifies acute asthma (Yellowlees & Kalucy, 1990). The literature, however, clearly documents anxiety in excess of "normal" levels in many asthma patients. Asthma panic-fear, which is presumed to reflect the patient's level of anxiety with regards to his or her condition, was first described by Kinsman and his associates. In the course of developing a scale to assess subjective asthma symptomatology, the Asthma Symptom Checklist (ASC: Kinsman,

Luparello, O'Banion & Spector, 1973), the investigators found that 42 per cent of adult patients reported feeling nervous and jittery, worried about the attack, scared, and helpless during most or all acute asthma episodes.

Subsequent work with the ASC has identified panic-fear as an important dimension of the asthma experience. Adult patients with high, moderate, or low levels of panic-fear have been differentiated in terms of medical prognosis (Dirks, Jones & Kinsman, 1977), frequency of requests for PRN medications (Dahlem, Kinsman & Horton, 1977), and likelihood of hospitalization (Staudenmayer, Kinsman, Dirks, Spector & Wangaard, 1979). More recently, panic-fear has been found to be inversely related to asthma knowledge and self-management skills (Brooks et al., 1989) and positively correlated with dyspnea intensity (Janson-Bjerklie et al., 1986; Gift, 1990; Carr, Lehrer & Hochron, 1992). The observed differences in medical outcomes and asthma symptomatology in patients with varying levels of panic-fear seem to be independent of objective measures of pulmonary functioning (Smoller, Pollack, Otto, Rosenbaum & Kradin, 1995).

Research examining panic-fear in children has yielded findings consistent with those obtained in studies using adult subjects. For example, there is reason to believe that panic-fear influences the

medical management of pediatric asthma. Children with elevated levels of asthma panic-fear, as compared to those with moderate or low levels, are more often prescribed continuous steroid medication (Fritz & Overholser, 1989) and are given such drugs at higher dosages (Baron et al.,1992). As is true for adults, the observed differences in medication regime are independent of level of pulmonary functioning and therefore do not appear to be a function of disease severity (Baron et al, 1992). Panic-fear in young patients, furthermore, is related to the number of hospitalizations in the previous year (Fritz & Overholser, 1989) and to psychosocial adjustment and family dysfunction (Baron et al., 1992). Children's panic-fear symptoms are similar to those of adults, but in addition they report feeling alone, lonely, unhappy, left out, and being worried about themselves and about asthma; they do not report being nervous and jittery (Fritz & Overholser, 1989).

Panic-fear's ability to impact, in a negative manner, upon treatment regimes, medical outcomes, behavioural adjustment, and family functioning is quite well-documented. Nevertheless, the panic-fear phenomenon remains poorly understood. Specific vulnerability factors related to its development have yet to be identified. Moreover, it remains to be established whether panic-fear represents a temporary state, influenced perhaps by variables such as disease

severity, or a more enduring tendency to respond anxiously to certain physiological changes (Fritz & Overholser, 1989). Early work conducted by the Kinsman group of researchers indicated that panic-fear may be both state-like and trait-like (Dirks, Fross & Evans, 1977; Dirks, Kinsman & Jones, 1977). Although Dirks et al.'s (1977) hypothesized "Panic-Fear personality" failed to generate much research interest in other quarters, a recent investigation has suggested a more prominent role for subjectively perceived and objectively measured disease severity in asthma-specific versus generalized panic-fear (Carr, Lehrer & Hochron, 1995).

Panic-Fear and Panic Disorder

In their quest to better comprehend the nature of the panic-fear phenomenon, some investigators have turned to the panic disorder literature. Although this line of research is in its infancy, preliminary results suggest that variables implicated in the pathogenesis of panic disorder may also have explanatory value with regards to asthma panic-fear (Carr et al., 1992; Carr, Lehrer, Rausch & Hochron, 1994; Carr et al., 1995). DSM-IV (American Psychiatric Association, 1994) defines a panic attack as "a discrete period of intense fear or discomfort, in which four (or more) of the following symptoms developed abruptly and reached a peak within 10 minutes." The symptoms specified include palpitations, sweating, trembling or

shaking, shortness of breath, choking feelings, chest pain, nausea or abdominal distress, dizziness, derealization, paresthesias, chills or hot flushes, and fears of losing control, going crazy or dying. In order for the criteria for a diagnosis of panic disorder to be met there must be a history of recurrent, unexpected panic attacks that are not due to a chemical substance, explainable by a medical condition, or better accounted for by another mental disorder. In addition, at least one of the panic attacks must have been followed by persistent concern about having another attack, catastrophic concerns about the consequences of the attack, and significant behavioural change.

Several of the symptoms of a panic attack overlap with symptoms of pulmonary conditions such as asthma (Smoller et al., 1995), moreover dyspnea has been described as the core somatic symptom of panic disorder (Carr et al., 1992). This symptom overlap has given rise to a great deal of speculation about the role of respiratory physiology in panic disorder (Smoller et al., 1995) and the nature of psychiatric morbidity in patients with obstructive lung disease (Porzelius, Vest & Nochomovitz, 1992; Carr et al., 1994). Such speculations have been fuelled by the finding that pulmonary patients with a history of panic display marked tendencies to catastrophize about physical sensations (Porzelius et al., 1992), as well as by the high comorbidity rates between panic disorder and

pulmonary dysfunction (Spinhoven, Ros, Westgeist, & Van Der Does, 1994). For example, panic disorder prevalence rates of 12 to 24 percent have been documented in the asthmatic population (Yellowlees et al, 1988; Yellowlees and Ruffin 1989); in the general population the range is 1 to 6 per cent (Carr et al, 1992). Although some authors have attributed higher prevalence rates to selection bias (Peski-Oosterbaan, Spinhoven, Van Der Does, Willems & Sterk, 1996), there does appear to be a more general consensus on the issue of a relationship between catastrophic cognitions and an enhanced sensitivity to respiratory changes in some patients with asthma as well as in patients with panic disorder.

Respiration and Panic - Three Conceptual Models

Respiration features prominently in a number of conceptual models of panic disorder. In two of these, the false suffocation alarm and the hyperventilation models, respiratory anomalies assume a central role. Under the terms of Klein's (1992) false suffocation alarm model, panic disorder patients are presumed to have a highly sensitive "suffocation monitor." This alleged mechanism is held to be of evolutionary significance in that it could have served to alert our ancestors to dangerous increases in CO_2 levels, such as may have occurred during cave-ins.

Experimental evidence in support of the false suffocation alarm model is drawn from CO_2 challenge studies, which have demonstrated that panic disorder patients experience panic and ventilatory changes when forced to inhale high concentrations of this agent. Clinical confirmation is drawn from congenital central hypoventilation syndrome, a condition also known as Ondine's curse. Individuals afflicted with this rare disorder fail to increase respiratory drive in the face of falling oxygen levels, nor do they suffer from dyspnea or smothering sensations as CO_2 rises. Their marked lack of distress, both respiratory and affective, in life-threatening circumstances is interpreted by proponents of the model as indicative of an abnormally insensitive suffocation monitor (Klein, 1992; Smoller, et al., 1995).

The false suffocation alarm theory of panic implies that a heightened sensitivity to rising CO_2 levels should be manifested through such mechanisms as a reduced breath-holding capacity. Studies of breath-holding duration, however, have failed to find the expected negative correlation with anxiety and panic (Eke & McNally, 1996). The argument that the extreme concentrations of CO_2 used in challenge studies are irrelevant to spontaneous panic and that even at these concentrations the panic rate among panic disorder patients is considerably less than 100 per cent poses further difficulties for this model (Smoller et al., 1995).

Whereas the false suffocation alarm model accentuates the panicogenic effects of increased CO_2, the hyperventilation model stresses the role of reduced CO_2 in the panic cycle. It assumes that individuals who are vulnerable to panic are chronic hyperventilators. Their abnormally high rate of respiration increases under stress to the point where CO_2 is blown off from the lungs so quickly that its arterial partial pressure drops sharply. The resulting hypocapnia produces a corresponding rise in blood pH which, in turn, causes the symptoms of dyspnea, dizziness, derealization as well as a sense of impending doom (Barlow, 1988; Ley, 1989; Smoller et al., 1995; Gardner, 1996).

The hyperventilation model has as its basis a venerable body of evidence linking this distinct ventilatory pattern with anxiety, as well as more contemporary data suggesting that panic disorder patients are particularly sensitive to its effects (Smoller et al., 1995). One variant of the model, Ley's dyspnea-fear theory (Ley, 1989), acknowledges that catastrophic cognitions are an integral part of the panic experience but assumes that these are a consequence of hyperventilation-induced cerebral hypoxia. The theory further assumes that panic is preceded by severe and perceivedly uncontrollable dyspnea (Ley, 1989). In support of the theory Ley

(1989) has noted that dyspnea is the only frequently reported and intense symptom that regularly precedes the onset of panic.

The hyperventilation model is well able to account for the symptomatic presentation of panic disorder, in that the physiological changes that follow periods of breathing in excess of metabolic requirements are known to cause many of the somatic symptoms of panic (Ley, 1989; Smoller et al., 1995; Gardner, 1996). It is less adequate in terms of explaining how hyperventilation challenges can produce symptoms, but not frank panic, in some panic disorder patients or the finding that panic has preceded hyperventilation in CO_2 challenge situations (Smoller et al., 1995). With respect to the dyspnea-fear variant, this has yet to receive an adequate test. Carr, Lehrer and Hochron (1992), in a comparison of panic disorder and asthmatic patients versus normal controls, reported that dyspnea was predictive of panic in the asthmatic but not the panic disorder subjects. These investigators, however, used a symptom checklist designed specifically for asthma patients and, moreover, failed to assess for perceptions of control.

Barlow (1988), in a thorough review of the panic provocation literature, grouped the procedures used by researchers into three broad categories. In addition to respiratory based techniques such as hyperventilation or CO_2 inhalation, investigators have also employed

pharmacological agents and behavioural strategies to produce panic. As the physiological processes invoked by many of these methods are quite diverse and, in some cases, incompatible Barlow (1988) concluded that there is no single biological pathway to panic. If biology alone is insufficient to account for panic, then it necessarily follows that other factors must also come into play. In addition to acknowledging that the respiratory system may be implicated in the pathogenesis of panic, cognitive models accord equal weight to the psychologic substrate of panic disorder. Moreover, somatic events other than respiratory changes, for example an accelerated heart rate, are recognized as able to trigger the panic cycle (Smoller et al., 1995).

The major premise adopted by supporters of a cognitive model of panic disorder is that the panic response arises from a catastrophic misinterpretation of bodily symptoms. According to cognitive theorists, panic disorder patients interpret anxiety related somatic sensations such as dyspnea as signifying a physiologically or psychologically dangerous outcome (Beck & Emery, 1985; Clark, 1986). These catastrophic thoughts give rise to further anxiety, which then exacerbates the original physical symptoms. Panic results when this positive feedback loop spirals out of control (Lilienfield,

Turner & Jacob, 1993; Smoller et al., 1995). A schematic representation of the cognitive model of panic is shown in Figure 2.

The basic assumptions of cognitive theorists are substantiated by research findings demonstrating that panic disorder patients, when compared to control subjects, have similar physiologic reactions in challenge situations but differ in the manner in which these are perceived (Holt & Andrews, 1988; Smoller, 1995). Additional support is derived from studies showing that provocation is less likely to produce panic when panic disorder patients are given information designed to reduce catastrophic thinking (Smoller et al., 1995). For example, it has been demonstrated that increasing panic disorder patients' perceptions of control (Sanderson, Rapee & Barlow, 1989) or forewarning them about the symptoms likely to be elicited (Rapee, Mattick & Murrell, 1986) reduces the prospect that panic will occur in response to CO_2 inhalation. The proven efficacy of cognitive-behavioural therapy in treating panic disorder would also seem to lend credence to cognitive models of panic (Smoller et al., 1995).

Cognitive explanations have been criticized on the grounds of their apparent inability to account for nocturnal panics. Research findings which imply that catastrophic thinking does not inevitably precede or accompany panic attacks have also been interpreted as inconsistent with cognitive theory (Lilienfeld et al., 1993; Smoller et

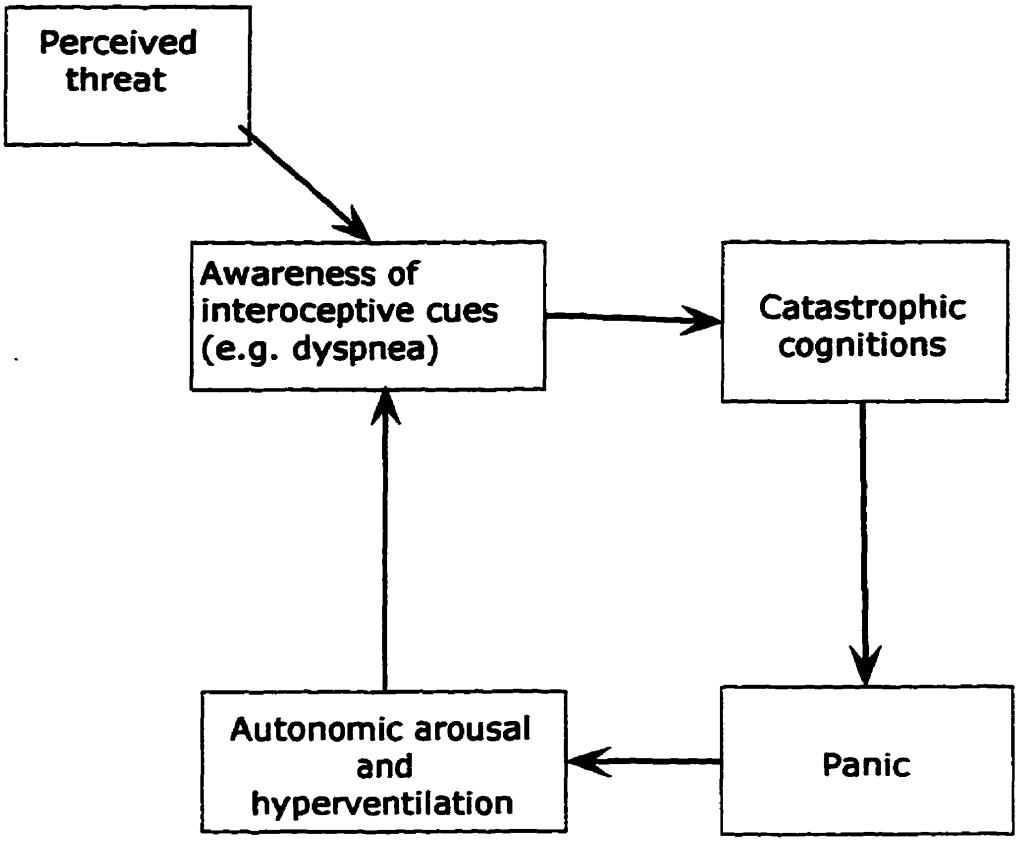

Figure 2.
Cognitive Model of Panic
(from Smoller et al., 1995)

al., 1995). Cognitive theorists, however, contend that catastrophic thoughts may be so fleeting as not to enter into conscious awareness (Lilienfeld et al., 1993). They further argue that some panic disorder patients may be so hypersensitive to somatic events that normal physiologic changes occurring during sleep, such as decreases in heart or respiration rate, are sufficient to trigger the panic cycle (Barlow, 1988).

Anxiety Sensitivity and Panic

In recent years the concept of anxiety sensitivity has been the focus of much debate among those who favour cognitive models of panic (Lilienfeld et al., 1995; Taylor, 1995). Anxiety sensitivity, which refers both to the fear of the physiologic or psychologic symptoms of anxiety and the belief that such symptoms portend harmful consequences, is presumed to be a stable personality trait that is causally related to the development of panic disorder (Reiss & McNally, 1985; Reiss, 1991). It is thought to be the product of biological factors and learning history. This latter may include, but is not restricted to, prior exposure to frightening experiences such as panic attacks. Other plausible routes likely involve social learning experiences (Reiss, Peterson, Gursky & McNally, 1986; Donnell & McNally, 1990), for example repeated warnings to a child about the dangers of becoming "over-excited." Similar constructs, such as "fear

of fear" (Goldstein & Chambless, 1978), differ from anxiety sensitivity in that they are held to arise solely through the process of interoceptive conditioning and further, to be a consequence rather than a cause of panic (Silverman et al., 1991).

Anxiety sensitivity is one of several components of Reiss' (1991) expectancy theory of anxiety, fear and panic. A related component, anxiety expectancy, refers to the expectation that anxiety will occur in a given situation. According to expectancy theory, both anxiety expectancy and anxiety sensitivity are necessary prerequisites of anxiety. Anxiety sensitivity, however, is presumed to take temporal precedence over anxiety expectancy. To illustrate this point, an individual may fear dyspnea because he or she believes that any discomfort in the chest region signifies that a heart attack is imminent (anxiety sensitivity). He or she would therefore expect to feel anxious in any situation that could trigger the feared symptom (anxiety expectancy). In addition to taking temporal precedence in the chain of events leading to anxiety, anxiety sensitivity is also thought to serve as an amplication factor, in that elevated levels of this individual difference variable increase the propensity to attend to, and perhaps exaggerate, internal cues (Shostak & Peterson, 1990). Thus this perceptual style intensifies the aversiveness of the anxiety

experience, and sets in motion the positive feedback loop that culminates in panic (Taylor, 1995).

Anxiety Sensitivity and Trait Anxiety

Whereas trait anxiety is the general tendency to respond anxiously to a large number of stressors, anxiety sensitivity is a specialized tendency to respond anxiously to symptoms of arousal (Lilienfeld et al., 1993; Taylor, 1995). One implication of this distinction is that individuals may demonstrate elevated levels of trait anxiety but show no corresponding increase in anxiety sensitivity. Patients with generalized anxiety disorder appear to be characterized by this pattern; those with panic disorder obtain high scores on measures of both constructs (McNally, 1989). Some authors (e.g. McNally, 1989; Taylor, 1995) have interpreted such findings as supportive of the nosological significance of anxiety sensitivity; others, however, have questioned the extent to which trait anxiety and anxiety sensitivity are conceptually distinguishable (e.g. Lilienfeld, Jacob & Turner, 1989; Lilienfeld et al, 1993).

Investigations which have included measures of trait anxiety and anxiety sensitivity have found that such instruments are only modestly correlated (McNally, 1989; Reiss, 1991; Taylor, 1993), and factorially distinct (Taylor, 1993). Moreover, as discussed in greater detail below, anxiety sensitivity has been shown to be a better

predictor of panic-related phenomena than is trait anxiety. Current views of anxiety sensitivity acknowledge an overlap with trait anxiety in that the two variables are deemed to be hierarchically organized (Lilienfeld et al, 1993; Taylor, 1995). Although the exact nature of the organizational structure is yet to be determined, confirmatory factor analysis has lent preliminary support to a model proposed by Lilienfeld and his colleagues (Lillienfeld et al., 1993). The model, which is shown in Figure 3, draws on the work of Reiss (1991) and Telch, Shermis and Lucas (1989), amongst others, in assuming that anxiety sensitivity is but one of a number of lower-order factors nested within a higher-order dimension of trait anxiety. The model is compatible with extant anxiety sensitivity findings in that it presumes shared variance with the higher-order factor as well as unique variance that is unrelated to trait anxiety. It thus allows for measures of anxiety sensitivity to augment the information provided by more global measures of trait anxiety (Lilienfeld et al., 1993).

Anxiety Sensitivity in Clinical and Non-Clinical Panic

Consistent with its definition as the fear of anxiety related sensations, Reiss and his colleagues, in the context of validating a scale to measure anxiety sensitivity, the Anxiety Sensitivity Index (ASI: Reiss et al., 1986) found that subjects high in anxiety sensitivity were more fearful when discussing anxiety symptoms than

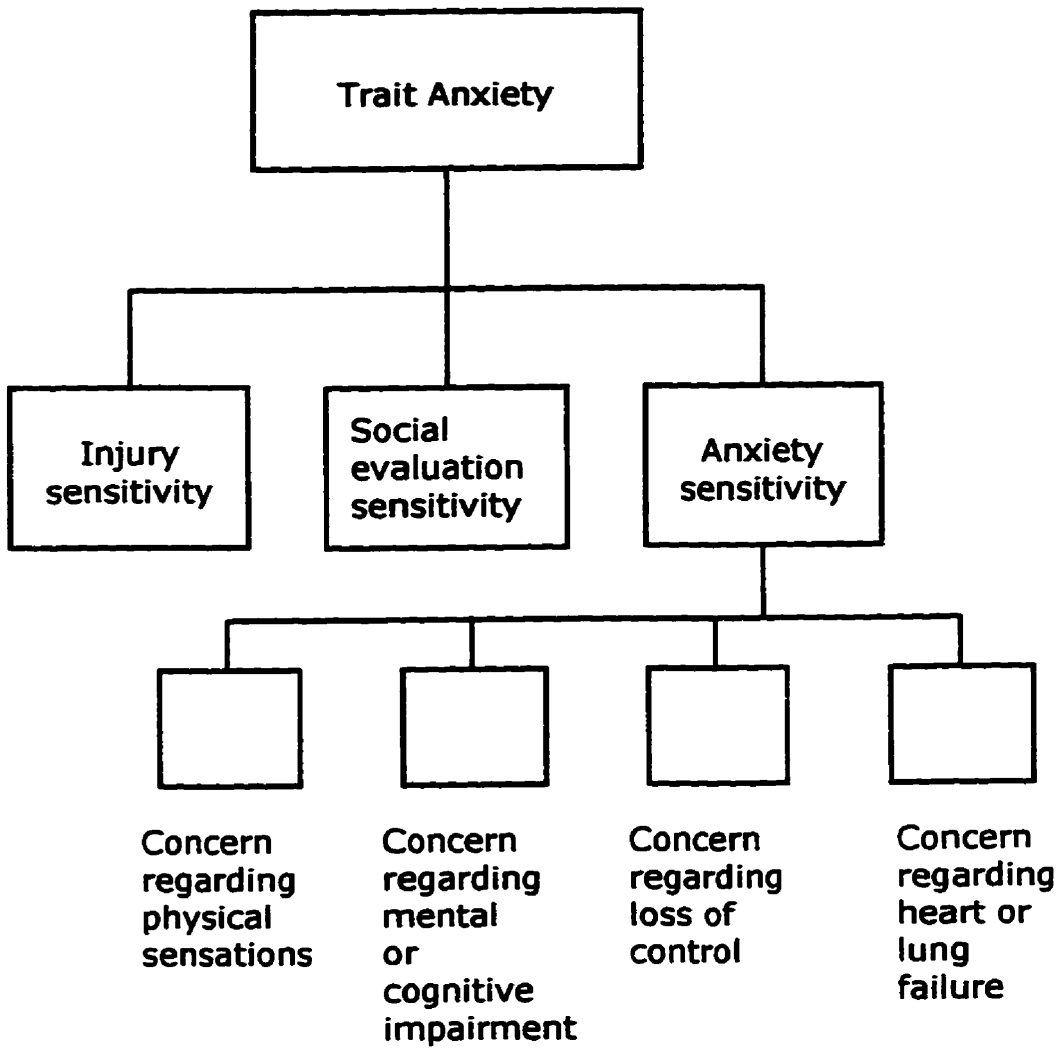

Figure 3.
Hierarchical Model of the Relationship between Trait Anxiety and Anxiety Sensitivity
(adapted from Lilienfeld et al., 1993)

were low anxiety sensitive subjects, even when levels of trait anxiety were held constant (Reiss et al., 1986). In additional work examining the relationship between anxiety sensitivity and fear responding, the ASI was proven to be more predictive of scores on a fear survey than either measures of trait anxiety or anxiety frequency (Reiss et al., 1986). In order to avert criticisms that the superior predictive power of the ASI reflected nothing more than a general tendency for diverse fears to correlate, Reiss, Peterson and Gursky (1988) attempted to determine whether scores on the ASI and an Injury Sensitivity Index (ISI) made independent contributions to the prediction of a measure of dissimilar fears they designed specifically for their study. The results indicated that the ASI predicted variance in the dependent measure beyond that which was attributable to the ISI, trait anxiety and anxiety frequency.

A link between anxiety sensitivity and panic disorder has been documented in a number of studies. For example, it has been found that panic disorder patients with agoraphobia, as compared to other anxiety disordered patients or normal controls, demonstrate elevated levels of anxiety sensitivity (Reiss et al., 1986). Similar results have been reported by McNally & Lorenz (1987), who further observed that scores on the ASI were more predictive of general fearfulness among this patient population than were those on a measure of trait anxiety.

In a like vein, Carr and his associates (Carr et al., 1994), noted that ASI scores differentiated between panic disorder patients with or without asthma and their non-panicking counterparts.

Although the evidence suggests that anxiety sensitivity and panic disorder covary, this does not necessarily imply that the former is causally related to the latter. Donnell and McNally (1990), however, reasoned that the existence of individuals high in anxiety sensitivity but with no history of panic would help render implausible the argument that the fear of anxiety related sensations stems solely from a prior experience of panic. Their comparison of college students with high, moderate, or low scores on the ASI revealed a significantly higher proportion of panickers in the former group. Furthermore, it also established that more than half of the high anxiety sensitive subjects had never had a panic attack. Irrespective of panic status, a significantly greater proportion of high anxiety sensitive subjects had either received psychological treatment or had first-degree relatives with a history of panic.

Spontaneous panics arise in the absence of any obvious trigger. By way of contrast, cued panics occur in the actual or anticipated presence of a feared stimulus (Barlow, 1988). Although spontaneous panics are the hallmark feature of panic disorder, cued panics are also a feature of this condition for some patients (Asmundsen & Norton,

1993). Moreover, spontaneous panics are experienced by patients with anxiety diagnoses other than panic disorder (Barlow, 1988). Donnell and McNally (1990) failed to assess for a history of cued panics. As a consequence, their conclusion that elevated levels of anxiety sensitivity were not necessarily a product of personal experience with panic was criticized as premature (Lilienfeld et al., 1993). Nevertheless, a study in which cued panics were considered yielded results similar to those of Donnell and McNally (1990) in that there were significant differences between the high and medium or low anxiety sensitive groups in terms of panic history as well as a substantial percentage of high anxiety sensitive subjects with no history of panic (Asmundsen & Norton, 1993). Thus it would appear that mechanisms other than direct experience of panic are implicated in the development of anxiety sensitivity.

Provocation studies afford researchers the opportunity to examine physiologic and psychologic determinants of panic. In recent years there have been a number of investigations which have examined the relationship between anxiety sensitivity and experimentally produced panic. Subject samples have primarily been drawn from the non-clinical population, and the procedures used have included voluntary hyperventilation (Holloway & McNally, 1987; Donnell & McNally, 1989; Asmundsen, Norton, Wilson & Sandler,

1994; Rapee & Medoro, 1994;) CO_2 inhalation (Eke & McNally, 1996), intravenous infusions of cholecystokinin tetrapeptide (CCK-4: Koszycki, Cox and Bradwejn, 1993), and stress induction techniques (Shostak & Peterson, 1990). Common to all of these studies is the finding that high anxiety sensitive subjects report more fearful cognitions after challenge than do other subject groups. This finding, moreover, holds when subjects with varying levels of anxiety sensitivity are equated on trait anxiety (Rapee & Medoro, 1994).

Several challenge studies have also documented that subjects with elevated levels of anxiety sensitivity demonstrate a significant increase in self-reported somatic symptoms after the manipulation has been applied (e.g. Holloway & McNally, 1987; Shostak & Peterson, 1990; Koszycki et al., 1993; Asmundsen et al., 1994). For example, Asmundsen et al. (1994) observed that high anxiety sensitive subjects, as compared to those with low anxiety sensitivity, rate post-challenge dyspnea and chest pain as being much more severe. In this same study, an objectively measured physiological response (heart rate) was shown not to vary as a function of anxiety sensitivity level. Similarly, Shostak and Peterson (1990) noted that post-challenge frontal electromyographic activity and systolic blood pressure did not differentiate between their subject groups. In the case of post-challenge anxiety, the results have been less uniform. Three studies

have reported increased anxiety in high anxiety sensitive subjects after voluntary hyperventilation (Holloway & McNally, 1987; Donnel & McNally, 1989) or stress induction (Shostak & Peterson, 1990). Yet there appeared to be no differential increase in anxiety after CCK-4 infusion (Koszycki et al., 1993).

There have also been conflicting results with regards to prior history of panic. Donnell and McNally (1989), for instance, found that those subjects who were most responsive to challenge had high anxiety sensitivity and a history of panic. By way of contrast, Asmundsen and his associates (Asmundsen et al., 1994) found no such interaction. Other noteworthy findings emerging from the provocation literature include the suggestion of a response bias in high anxiety sensitive subjects. In the studies conducted by McNally and his colleagues (Holloway & McNally, 1987; Donnell & McNally, 1989) post-challenge assessments indicated that high anxiety sensitive subjects demonstrated a marked tendency to endorse physical sensations that were unrelated to hyperventilation. Finally, ASI scores appear to be more strongly predictive of somatic, cognitive and affective symptoms following challenge than are scores on measures of trait anxiety (Rapee and Medoro, 1994; Eke & McNally, 1996).

Anxiety Sensitivity and Asthma Panic-Fear

Any firm conclusions regarding the status of anxiety sensitivity as a risk factor for panic disorder must await the results of longitudinal investigations (Lilienfeld et al., 1993). Nevertheless, the weight of the evidence would seem to point to measures of anxiety sensitivity as being more strongly related to fearfulness and panic attacks than are measures of trait anxiety (Taylor, 1995). Anxiety sensitivity, moreover, has been shown to influence the subjective response to a variety of panicogenic agents. There would seem to be adequate reason to suspect that anxiety sensitivity may have some bearing on asthma panic-fear. Firstly, elevated levels of anxiety sensitivity are presumed to be the product of both biology and learning history (Reiss et al, 1986 Donnell & McNally, 1990). In the case of an asthmatic child the basic biological defect, hyper-reactive airways, should provide ample opportunity for fear to be conditioned to asthma and anxiety related sensations such as pulmonary dyspnea. Furthermore, the over-protectiveness that has been shown to characterize the parents of some children with asthma could furnish an environment wherein a child may learn to believe that his or her symptoms are dangerous.

Secondly, there are commonalities between the results of studies on the perception of bronchoconstriction in asthma and those

that have looked at the relationship between anxiety sensitivity and the response to challenge. The increased anxiety and sensitivity to arousal that has been documented in asthma "over-perceivers" appear to be similar in nature to the fearful cognitions and comparatively more intense somatic sensations reported, post-challenge, by high anxiety sensitive subjects. Indeed, it would seem that both of these subject groups display the internal focus described by Shostak and Peterson (1990) as an amplification factor in fear responding.

Finally, research conducted by Carr and his associates has demonstrated a link between anxiety sensitivity and panic in patients with asthma. Two studies have shown that anxiety sensitivity is predictive of panic-fear in asthma patients comorbid for panic disorder (Carr et al., 1994, 1995). A third study involved a comparison between subjects with asthma and no psychiatric diagnosis versus subjects with panic disorder and no respiratory disease (Carr et al., 1992). To recapitulate the findings, there was a strong relationship between dyspnea and panic-fear in asthmatic subjects, but not in panic disorder subjects without asthma. The measures used to assess breathlessness and panic were drawn from a scale intended for use with asthmatics, the ASC (Kinsman et al., 1973). The items, therefore, may not have been wholly consonant with the experience of panic disorder patients who were not asthmatic.

In terms of the implications of these findings with regards to anxiety sensitivity and asthma panic-fear, the former refers to the tendency to respond with fear to symptoms that are perceived as threatening. Thus, one would expect to find very significant correlations between panic-fear and dyspnea in high anxiety sensitive subjects for whom the latter symptom represents an extremely salient source of threat. Support for the speculation that some patients with asthma view dyspnea as dangerous or threatening may be drawn from a study conducted by Janson-Bjerklie, Ferketich, Benner and Becker (1992). These authors performed a content analysis of patients' descriptions of acute asthma episodes. The results revealed the existence of a group of subjects who couched their experiences in life-or-death terms. In subsequent statistical analyses the high "perceived danger" group proved to be significantly more likely to obtain elevated scores on the panic-fear sub-scale of the ASC.

Anxiety Sensitivity and Panic-Fear in Pediatric Asthma

In the present study the relationship between anxiety sensitivity and panic-fear is further explored. Preliminary results have suggested an association between these two variables in adult asthmatic patients comorbid for panic disorder (Carr et al., 1994, 1995); this study aims to extend existing knowledge by examining the relationship between anxiety sensitivity and panic-fear in a group of

subjects for whom a diagnosis of panic disorder is unlikely. The population of interest is 6 to 12-year-old asthmatic children. Diagnoses of panic disorder are rare, but not unknown, in this age group (Klein, Mannuza, Chapman & Fyer, 1994). Panic in the context of an asthma attack, however, is quite common. Parental reports have indicated that as many as 42 per cent of young patients frequently panic in response to a worsening of their condition (Creer, Marion & Creer, 1983).

The panic-fear literature points to a connection between this variable and subjectively perceived dyspnea intensity (Carr et al., 1992, 1995). There is also a suggestion that panic-fear may be associated with objectively measured disease severity (Carr et al., 1995). In addition, there is some indication that panic-fear may be inversely related to knowledge and self-care of asthma (Brooks et al., 1989). Research has yet to determine, however, whether these observed relationships apply to children with asthma as well as to adults.

The current investigation proceeds from the assumption that panic-fear in children with asthma reflects underlying concerns surrounding specific psychophysiologic sensations that have come to be associated with danger or threat. Thus, panic-fear in children with asthma is expected to relate to anxiety sensitivity in much the same

manner as do panic-related experiences in the wider population. The primary objective of the current research is to determine whether anxiety sensitivity is predictive of panic-fear in children with asthma. In the course of meeting this objective the following four questions are addressed:

(1) Is anxiety sensitivity more strongly predictive of panic-fear frequency and intensity than previously identified correlates such as subjectively reported dyspnea, objectively assessed disease severity, or asthma knowledge and self-care behaviour?

(2) Does anxiety sensitivity explain variance in panic-fear over and above that which is attributable to trait anxiety?

(3) Do high anxiety sensitive asthmatic children have more severe asthma exacerbations relative to other children with asthma?

(4) Is the subjective experience of asthma different for children with varying levels of anxiety sensitivity?

Respitrace Recording of Thoraco-Abdominal Asynchrony

A secondary aim of this study is to ascertain whether thoraco-abdominal asynchrony, as assessed by Respiratory Inductance Plethysmography (Respitrace), is a clinically useful index of severity in acute childhood asthma. Indices of acute asthma severity in current use include PEFR and arterial oxygen saturation values. PEFR is problematic because it is effort-dependent and, as such, is influenced

by the patient's own perception of physical status (Geelhoed, Landau & LeSoeuf, 1990). Arterial oxygen saturation measurement, although more reliable than PEFR (Geelhoed et al., 1990), is an indirect means of assessing severity as the value it provides reflects the end result of the physiological processes that underlie acute asthma (Mitchell, 1996, personal communication).

Thoraco-abdominal asynchrony occurs during times of respiratory distress. Under normal circumstances the ribcage and abdomen move in concert during respiration (Davis, Cooper & Mitchell, 1993). When the airways are occluded, however, the outward motion of the ribcage during inspiration may lag behind that of the abdomen. When airway calibre is greatly reduced frank paradoxical motion, with the ribcage moving inwards during inspiration, may be seen (Allen, Wolfson, McDowell & Shaffer, 1990). Thoraco-abdominal asynchrony has been documented in adults with acute asthma as well as in infants with reversible obstructive airways disease, and has been shown to decrease with recovery (Hillman et al., 1986; Allen et al., 1990; Davis et al., 1993). Respitrace recording has proven to be helpful in terms of quantifying the extent of thoraco-abdominal asynchrony in infants between the ages of 3 and 17 months (Allen et al., 1990; Davis et al., 1993), but is yet to be tested on older children with asthma.

METHOD

Subjects

The subjects were a convenience sample of 6 to 12-year-old children who presented at the Emergency Department of the Alberta Children's Hospital for treatment of acute asthma. Each of the subjects was a known asthmatic with a diagnosis of asthma of at least six months' duration. Coexisting heart disease, hypertension, pulmonary conditions other than asthma, and any disorder requiring regular medication or which might impair cooperation with the protocol were grounds for exclusion from the study, as was pseudo-asthma, a condition in which symptoms are triggered by a paradoxical closure of the vocal cord during inspiration, and in which psychogenic factors are strongly implicated. Children who had participated in a previous asthma study conducted on the same site were also ineligible to participate.

Physiological Measures

Arterial Oxygen Saturation: Arterial oxygen saturation levels were monitored by nursing staff using a finger probe attached to a Nallcor Pulse Oximeter (model #N200PB). Research has suggested that children presenting for treatment of acute asthma tend to have oxygen saturation levels in the range of 90 to 97 per cent (Geelhoed et al., 1990).

Heart and Respiration Rate: Heart and respiration rate were assessed by nursing staff by means of one minute counts.

Thoraco-abdominal Asynchrony: The Respitrace system, which allows for a non-invasive continuous measure of respiratory patterns, was used to monitor ribcage and abdominal excursions during breathing. Respitrace consists of coils of insulated wire sewn onto elastic material (respibands) that are worn around the ribcage and abdomen. The plethysmographic signals generated from the respibands were routed through a Dataq Corporation Waveform Scroller board in an IBM PC. The Scroller performed an analogue to digital conversion on each signal and stored the digitized waveforms on a hard disk for later analysis. Volume calibration was not necessary for this study, instead the signals were recorded as an inductance (volts). Inductance measurement allows for a comparison of the extent of ribcage versus abdominal displacement during respiration; of interest in the present study was the calculated phase angle, an index of thoraco-abdominal asynchrony obtained by dividing the difference in time to peak displacement for the ribcage and abdomen by the total respiratory cycle time.

Residual Airways Obstruction: Forced expiratory volume in one second (FEV_1) refers to the maximum volume of air that can be exhaled in one second from full vital capacity. It is a measure of

larger airway flow (Shapiro & Bierman, 1987) and predicted values are based on age, height, weight and sex adjusted norms (Carr et al., 1995). A normal FEV_1 is expected to reach 80 per cent or better of the predicted value (Shapiro & Bierman, 1987).

Psychological Measures

Subjective Asthma Symptomatology: The Children's Asthma Symptom Checklist (CASCL: Fritz & Overholser, 1989), a 47-item instrument suitable for children between the ages of 6 and 18, was used to measure subjective asthma symptomatology. It is presented in Appendix 1. The CASCL is a modification of the ASC (Kinsman et al, 1973) in which the items have been reworded with vocabulary appropriate for children while still retaining the original meaning. Children are required to rate how frequently, on a scale from 1 (never) to 5 (always), each symptom occurs. A factor analysis of the CASCL has yielded a 3 factor solution, Physical Symptoms, Panic-Fear, and Hyperventilation/Irritability. For the purposes of the current study a modified rating format, designed to reflect symptom severity, was presented in conjunction with the panic-fear items at the time of the index episode. The entire CASCL as originally devised was administered at a follow-up session.

Dyspnea: A visual analogue scale (VAS) was used to assess the subjective experience of dyspnea during the index attack. The 16

centimetre vertical scale was anchored at the low end with "not at all breathless" and "as breathless as can be" at the high end. Subjects were asked to mark the point that indicates the amount of dyspnea experienced at that minute; the score was the distance from the low end of the scale to the mark. VAS's are commonly used to assess dyspnea, and are held to be valid, reliable and sensitive measures (Gift, 1989).

Self-Management of Asthma: The Asthma Behavioral Assessment Questionnaire (ABAQ: National Heart and Lung Institute, 1984) contains 17 items and children are asked to rate, on a 5-point scale which ranges from never to always, how often they engage in a variety of asthma management strategies. The ABAQ, which has adequate reliability (Cronbach alpha = 0.72), was designed for children aged 9 years and above but has been used successfully with younger age groups (Rubin et al, 1989). The ABAQ is reproduced in Appendix 2.

Knowledge of Asthma: The Parcel Knowledge of Asthma Questionnaire (KAQ: Parcel, Nader & Tiernan, 1980) a 27-item instrument with a true-false response format, is shown in Appendix 3. It is suitable for elementary school children, adolescents and adults. The questionnaire is sensitive to changes in knowledge over time and has

Kuder-Richardson reliabilities of 0.88 for adults and 0.56 for children between the ages of 7 to 18 (Rubin et al, 1989).

Behavioural Adjustment: The Child Behavior Checklist (CBCL: Achenbach & Edelbrock, 1986) requires parents to indicate, on a 3-point scale, whether various social competencies or problem behaviours are true of their child. The checklist is well standardized, with acceptable reliability and validity, and yields scores on two broad band factors, Internalizing and Externalizing, plus several narrow band factors (Sattler, 1988).

Trait Anxiety: The Revised Children's Manifest Anxiety Scale (RCMAS: Reynolds & Richmond, 1978) is a 37-item questionnaire which assesses trait anxiety. Its reliability is well-documented (Lee, Piersel & Unruh, 1989; Perrin & Last, 1992; Rabian et al., 1993). It contains three factor scales, physiological, worry/oversensitivity, and concentration, as well as a lie scale.

Anxiety Sensitivity: The Children's Anxiety Sensitivity Index (CASI: Silverman et al., 1991), an 18-item self-report instrument, is a modification of the ASI (Reiss & McNally, 1985; Reiss et al., 1986) with items reworded so as to be more understandable to children. Children are asked to indicate on a 3-point scale, their level of fear in response to various anxiety symptoms. In a clinical sample of 8 to 15 year-old children the test-retest and inter-item reliabilities were found

to be.0.76 and 0.87 respectively; the figures for a non-clinical sample of 11 to 18 year olds were 0.79 and 0.87 (Silverman et al., 1991). The CASI has been shown to be suitable for use with children aged 6 and up (Rabian et al., 1993), and is reproduced in Appendix 4.

Procedure

Participation took place in two stages. First, subjects were recruited following presentation at the emergency department for treatment of asthma. Permission from the attending physician was sought before any potential subject was approached. Once the child was admitted, but before treatment was started, the investigator approached the parent(s) or guardian(s) and explained the nature and purpose of the study. Parents were required to give formal informed consent prior to their child's participation; children were asked for their assent. Once consent and assent were obtained the children were fitted with the respibands and treatment was begun. Arterial oxygen saturation and heart and respiration rate values were recorded by nursing staff as part of routine admission procedures.

As soon as the first salbutamol mask was started, the child was asked to rate his or her level of dyspnea on the VAS, following which the CASCL items pertaining to panic-fear were administered verbally. In order to minimize stress to the child only pointing responses were required. Also during the first mask, the parent(s) or guardian(s) were

interviewed about the child's asthma. The questions are presented in Appendix 5. They were then asked to rate their child's behaviour on the CBCL. After application of the third salbutamol mask (and prior to reassessment by the attending physician) the respibands were removed and the VAS re-presented. Nursing staff then re-examined arterial oxygen saturation and heart and respiration rate values. Although every effort was made to ensure similar conditions for each child, factors related to medical staff's case loads, preferences with regards to acute asthma care etc., lead to considerable variability in terms of the physiological indices charted, the length of time between intake and reassessment, and the timing of the decision to hospitalize or discharge.

Approximately two weeks after the emergency visit the subjects attended a follow-up session at the hospital, during which time they underwent spirometric assessment. Also at the follow-up the children completed a third VAS as well as the ABAQ, KAQ, RCMAS, CASI and CASCL. In order to ensure that the subjects fully understood each item, all child questionnaires were verbally administered.

RESULTS

Subject Characteristics

Forty-two subjects (25 boys and 17 girls) were recruited between February 1995 and July 1996. An additional five families were approached but declined to participate in the study. Of the children who agreed to take part, thirty-nine returned for the follow-up.

The mean age of the sample was 8.64 years, and age at diagnosis varied from 3 months to 10 years (mean 3.26 years). One subject (2.4%) received no regularly prescribed medication and eight (19%) relied on salbutamol PRN to control asthma. Twenty-six subjects (61.9%) were prescribed both salbutamol and an inhaled steroid and the remaining seven (16.7%) also used oral steroids. Fourteen subjects (33.3%) had nocturnal asthma symptoms, and for 12 (28.6%) strong emotions precipitated attacks. Sixteen subjects (38.1%) were vulnerable to asthma attacks throughout the year and 26 had seasonal asthma. Four of the subjects (9.5%) had received some type of formal asthma education.

In the 12 months leading up to the index attack the average number of days missed from school because of asthma was 9.5 (range 0 to 60 days), and the mean number of attacks requiring emergent care was 2.3 (range 0 to 12). In terms of treatment for the index

attack, the number of salbutamol masks administered to the subjects ranged from one to nine (mean 3.9). 23 subjects (54.8%) were also given Prednisone, an oral steroid. Time from symptom onset to emergency admission ranged from 35 minutes to 48 hours (mean 10.54 hours) and the mean length of stay in the emergent care setting was 3.9 hours. Thirty-one subjects (73.8%) were discharged home and 11 (26.2%) were admitted as inpatients.

Descriptive Statistics

Descriptive statistics for the sample on physiological, self-report, and parent-completed measures are presented in Table 1. At the time of admission the average heart rate for the sample was 118.39, at reassessment the mean heart rate had increased to 132.31. The mean oxygen saturation values obtained during the index attack (intake = 92.48; reassessment = 93.48) were similar to values reported by Geelhoed et al. (1992) in their study of children with acute asthma. Seven children (16.7%) had values in the 85 to 89 per cent range at the time of admission; after treatment the number of subjects with values in this range dropped to four (9.5%). Lower oxygen saturation values post-treatment are predictive of hospital admission, and of return visits to the emergent care setting (Geelhoed et al., 1990). Respiration rate data was available for thirty-three subjects. The mean values at admission and at

Table 1.

Descriptive Statistics for the Sample

Variable	Mean	SD	Range	
			Min	Max
Hr1	118.39	28.13	66.00	250.00
Hrlast	132.34	25.25	66.00	178.00
Sat1	92.48	3.56	85.00	100.00
Satlast	93.47	3.80	80.00	100.00
Rr1	30.36	8.43	16.00	50.00
Rrlast	29.06	7.58	16.00	44.00
VAS1	6.98	5.05	0.00	16.00
VASlast	3.43	3.53	0.00	13.00
VASfollow-up	3.26	4.45	0.00	16.00
P-F intensity	25.92	10.75	15.00	63.00
KAQ	20.90	4.14	9.00	27.00
ABAQ	60.45	1.07	36.00	75.00
CBCLcom	47.50	8.78	30.00	70.00
CBCLprob	53.80	9.96	32.00	76.00
RCMAStotal	47.23	8.72	32.00	74.00
CASI	29.52	5.91	20.00	48.00
CASCL	122.95	17.12	67.00	215.00
P-F frequency	34.34	16.30	13.00	74.00
FEV_1%	89.89	17.85	10.00	117.00

Note: Hr1= heart rate at intake, Hrlast = heart rate at reassessment sat1 = oxygen saturation at intake, satlast = oxygen saturation at reassessment, Rr1 = respiration rate at intake, Rrlast = respiration rate at reassessment, vas1 = self-reported dyspnea at intake, vaslast = self-reported dyspnea at reassessment, meds = number of asthma medications, P-F intensity = Panic-fear intensity, CBCLcom = CBCL competence scale, CBCLprob = CBCL problem scale, RCMAS = Revised Children's Manifest Anxiety Scale, CASI = Children's Anxiety Sensitivity Index, CASCL = Children's Asthma Symptom Checklist, P-F frequency = Panic-fear frequency, FEV_1 % = percent predicted FEV_1 (residual airways obstruction)

reassessment for this group of subjects were 30.36 and 29.06 respectively. The average self-reported dyspnea score at the time of admission suggested a mild to moderate level of discomfort which showed some decline over the course of treatment. The average panic-fear intensity score during the index attack was 25.92 out of a maximum possible score of 75. Three of the subjects (7.2%) obtained scores greater than 45.

At follow-up the average percentage predicted value for FEV_1 was 0.90 which falls within normal limits. Five children (12%) had predicted values which fell below the 0.80 mark. The mean VAS score of 3.26, however, suggested that most children were not experiencing any marked degree of dyspnea. In terms of knowledge of asthma, the average score was 20.90 out of a total score of 27. All of the sample, with the exception of one child who attained a score of 9, had scores of 14 or better on this instrument. On the ABAQ the mean score was 60.45. The maximum value for this instrument is 80.

The average parent-rated behavioural competence and problem scores for the sample were well within normal limits (mean T scores 47.55 and 53.8 respectively). One child (2.4%) received a behavioural competence rating in the clinical range. A further nine children (22.5%) were in the borderline clinical range. As for behavioural problems, five subjects (12%) obtained borderline

clinical ratings and seven (16.8%) had clinical elevations on this measure. One child in the latter group had been diagnosed with attention-deficit/hyperactivity disorder. With respect to trait anxiety, the sample's average T-score on the RCMAS was 47.22, which falls in the non-clinical range. Although four children (9.6%) had clinically elevated scores on this measure, none had a clinically confirmed anxiety disorder diagnosis.

On the CASI the sample mean of 29.53 was somewhat larger than the 26.38 reported by Rabian and his associates (1993) for their non-clinical sample of 6-11 year-old children. Eighteen subjects in the present sample (35%) scored over 30 on this measure. Rabian et al. (1993) reported a mean score of 30.70 in a sample of 6-11 year old anxiety-disordered children (Rabian et al., 1993). The average CASCL score for the total scale was 122.95 out of a maximum possible score of 235. The mean panic-fear frequency score for the sample was 34.34 (maximum score attainable, 75). 27.5 per cent of the sample scored in the 45 to 74 range.

Zero-Order Correlations

The zero-order correlations between the variables are displayed in Tables 2 and 3. The Bonferroni-adjusted alpha level for the correlation coefficients is .001. Table 2 contains the coefficients for the variables assessed during the index attack. Recording difficulties

with the Respitrace equipment resulted in missing data for the thoraco-abdominal asynchrony values for 22 of the subjects. Correlational analyses were run with the data from the subset of subjects for whom scores on this variable were available and no significant coefficients emerged. In terms of other physiological data, heart rate at intake was significantly and negatively correlated with oxygen saturation values at reassessment, r (41) = -0.50, p<.001. Heart rate at reassessment was inversely related to intake and reassessment oxygen saturation values, r (41) = -.54, p<.001; r (41) = -.50, p<.001. Intake oxygen saturation values were also negatively correlated with the number of salbutamol masks given to the child, r (42) = -0.55, p<.001. Respiratory rate values, which were available for 33 subjects, failed to correlate with any other index attack variable.

The zero-order correlations between self-report and parent-report variables are presented in Table 3. Panic-fear intensity, which was assessed during the index attack, was significantly and positively related to panic-fear frequency, r (40) = 0.55, p<.001. Panic-fear frequency, assessed at the follow-up session, was positively correlated with CASCL, RCMAS and CASI scores, r (40) = 0.84, p<.0001; r (40) = 0.52, p<.0001; r (40) = 0.68, p<.0001. CASI

Table 2.

Correlations Among Variables Assessed During the Index Attack

	Hr1	Hr last	Sat1	Sat last	Vas1	Vas last	Rr1	Rr last
Hr1	-	.60*	-.42	-.54*	.18	.40	.18	.28
Hrlast		-	-.54*	-.50*	.24	.22	.30	.28
Sat1			-	.71*	-.15	-.37	-.46	-.41
Satlast				-	-.24	-.41	-.45	-.30
Vas1					-	.49	.14	.31
Vaslast						-	.36	.37
Rr1							-	.76*
Rrlast								-

	Masks	P-Fint
Hr1	.36	-.19
Hrlast	.43	-.05
Sat1	-.55*	-.17
Satlast	-.28	-.04
Vas1	.26	.16
Vaslast	.43	.14
Rr1	.46	-.15
Rrlast	.48	-.03
Masks	-	.12

Note: Hr1 = heart rate at intake, Hrlast = heart rate at reassessment, Sat1 = oxygen saturation at intake, Satlast = oxygen saturation at reassessment, Vas1 = self-reported dyspnea at intake, Vaslast = self-reported dyspnea at reassessment, Rr1 = respiration rate at intake, Rrlast = respiration rate at reassessment, Masks = number of salbutamol masks administered during the index attack, P-Fint = panic-fear intensity.

*p<.001

Table 3.

Correlations Between Self-Report and Parent-Rated Variables

	P-Fint	P-Ffreq	Com	Prob	RCMAS	CASI	CASCL	Meds
P-Fint	-	.55*	-.15	.19	.40	.45	.50*	.00
P-Ffreq		-	-.33	.15	.52*	.65*	.84*	.00
Com			-	-.11	-.40	-.18	-.45	.05
Prob				-	.27	.10	.24	.35
RCMAS					-	.48	.50*	.19
CASI						-	.62*	-.12
CASCL							-	.10
Meds								-

Note: P-Fint = panic-fear intensity, P-Ffreq = panic-fear frequency, Com = CBCL competence scale, Prob = CBCL problem scale, RCMAS = Revised Children's Manifest Anxiety Scale, CASI = Children's Anxiety Sensitivity Index, CASCL = Children's Asthma Symptom Checklist, ABAQ= Asthma Behavioral Assessment Questionnaire, Meds = number of asthma medications.

*$p<.001$

scores were significantly and positively related to full scale CASCL scores. Scores on the RCMAS were also positively correlated with CASCL scores, r (40) = 0.50, p<.0001. The correlation between the CASI and the RCMAS failed to reach significance, but nevertheless is consistent with values obtained in earlier research (see Reiss, 1991) and indicates an overlap in variance of 23 per cent. Percentage predicted FEV_1 the measure of residual airways obstruction did not correlate with any of the other variables.

Age and Gender Effects

In an attempt to correct for possible age effects all regression and Multivariate Analysis of Variance (MANOVA) analyses were run with and without controlling for age. As controlling for age did not alter the results, these analyses are not reported. A one-way MANOVA was conducted in order to investigate the possibility of gender-related differences on self or parent-report data. The alpha level was set at .007 according to Bonferroni criteria. No significant differences emerged therefore all subsequent analyses were collapsed across gender.

Anxiety Sensitivity as a Predictor of Panic-Fear Intensity and Frequency

A series of hierarchical multiple regression analyses were performed in order to examine the relative contribution of the CASI to the prediction of panic-fear intensity and frequency. The hierarchical approach indicates whether entering an additional predictor, or set of predictors, into a regression equation increases its predictive power. It is a conservative test, in that it maximizes the contribution of the variables first entered.

The first regression analysis examined whether anxiety sensitivity was predictive of panic-fear intensity in the emergent care setting. Trait anxiety, as indexed by scores on the RCMAS, self-reported dyspnea at the the time of the first salbutamol mask, and initial oxygen saturation levels were forced into the equation on the first step. Anxiety sensitivity, as measured by the CASI was allowed to enter on the second step. The results are presented in Table 4. The obtained multiple regression coefficient at the second step was significantly different from zero (F [4,35] = 3.67, p<.05), with the CASI explaining an additional 12 per cent of the variance in the dependent measure, a significant increase in R^2 (F[change] = 5.76, p<.050).

The second analysis was concerned with the relationship between anxiety sensitivity and panic-fear frequency. Trait anxiety,

Table 4.

Hierarchical Multiple Regression: Predictors of Panic-Fear Intensity in the Emergent Care Setting

	Betas	
Variable	Step 1	Step 2
VAS1	.134	.199
RCMAS	.395*	.202
SAT1	.046	.112
CASI		.400*
R	.423	.544
R^2	.180	.300
R^2 adjusted	.111	.215
F	2.626	3.670*
R^2 change	.179	.115*

Note: Sat1 = oxygen saturation at intake, Vas1 = self-reported dyspnea at intake, RCMAS = Revised Children's Manifest Anxiety Scale, CASI = Children's Anxiety Sensitivity Index.

*p<.05

knowledge of asthma, asthma self-management behaviours, and residual airways obstruction were entered on the first step; anxiety sensitivity was free to enter on the second step. The results may be seen in Table 5. At the second step the multiple regression coefficient was significantly different from zero ($F[5,31] = 7.82$, $p=.0001$). The CASI scores produced a significant increase in R^2 of 0.29 (F[change] = 20.18, p =0001) and, when combined with the first set of predictors, explained 57 per cent of the variance in panic-fear frequency scores.

The results of the regression analyses indicated that anxiety sensitivity made a significant independent contribution to the prediction of asthma panic-fear. Scores on the CASI emerged as the chief predictor of panic-fear intensity and frequency. Trait anxiety was no longer predictive of panic-fear once anxiety sensitivity was entered into the equations.

Anxiety Sensitivity and Objective and Subjective Indices of Severity in Acute Asthma

The question of whether high anxiety sensitive children experience more severe asthma exacerbations was examined by means of a series of MANOVA's in which CASI scores were used as a grouping variable. Normative data for the ASI, the original version of the scale, designates individuals scoring above 30 as high

Table 5.

Hierarchical Multiple Regression: Predictors of Panic-Fear Frequency as Reported at Follow-up

	Betas	
Variable	Step 1	Step 2
KAQ	.034	.084
ABAQ	.064	- .117
RCMAS	.513**	.175
FEV_1	.150	.143
CASI		.658***
R	.520	.747
R^2	.270	.559
R^2 adjusted	.179	.486
F	2.963*	7.821***
R^2 change	.270	.289***

Note: KAQ = Knowledge of Asthma Questionnaire, ABAQ = Asthma Behaviour Questionnaire, RCMAS = Revised Children's Manifest Anxiety Scale, CASI = Children's Anxiety Sensitivity Index, FEV_1 = residual airways obstruction.

*p<.05
**p<.01
***p<.001

anxiety sensitive and those scoring below 10 as low anxiety sensitive. Equivalent cut-off points have yet to be established for the CASI. Some investigators have advocated a sample-specific approach to grouping with the ASI which entails classifying subjects with scores greater than one standard deviation above the sample mean as "high," and those who score less than one standard deviation below the sample mean as "low" (Donnell & McNally, 1990; Asmundsen & Norton, 1993; Carr et al., 1994). The sample-specific approach has yet to be adopted for use with the CASI. As its use in the present study would have resulted in groups of widely disparate sizes, the categories "low," "medium," and "high" categories were formed by means of a tertile split.

A repeated measures MANOVA was used to determine whether the three anxiety sensitivity groups differed in terms of oxygen saturation, heart rate, and self-reported dyspnea at intake and reassessment. There was no significant main effect for group, nor was there a significant group by time interaction. With the alpha level set at 0.017 according to Bonferroni criteria, there were significant main effects for time on heart rate ($F[1,35] = 10.79$ $p<.001$ and self-reported dyspnea ($F[1,35] = 19.99$, $p<.0001$), but not oxygen saturation.

Respiration rate was examined separately because of missing data. The repeated measures MANOVA yielded non-significant results. The high anxiety sensitive children, however, demonstrated a slight increase in respiration rate over the course of treatment whereas the other two groups experienced a decline. Group differences on number of masks administered and time from admission to discharge were examined by means of a one-way MANOVA. No significant differences emerged. Group means for all of the dependent measures are displayed in Table 6.

The MANOVA's were replicated using panic-fear intensity as the grouping variable. The only significant results to emerge were for time on heart rate ($F[1,37] = 12.62$, $p<.001$) and self-reported dyspnea ($F[1.37] = 20.96$, $p<.0001$). Respiration rate decreased across time for all three groups. The means for the "low," "medium" and "high" panic-fear groups may be seen in Table 7.

Anxiety Sensitivity, Trait Anxiety, and Subjective Asthma Symptomatology

The final set of analyses addressed the question of whether anxiety sensitivity influences the subjective experience of asthma. The regression analyses established that scores on the CASI made a unique contribution to the prediction of panic-fear intensity and frequency. Zero-order correlations also indicated that CASI scores

Table 6.
Means for the Anxiety Sensitivity Groups on Variables Assessed During the Index Attack

	Low AS		Medium AS		High AS	
Measure	Intake	Reassess	Intake	Reassess	Intake	Reassess
Heart rate	125.31	130.46	118.81	138.36	112.43	126.93
Sat	92.61	92.85	92.90	93.00	92.36	94.64
Vas	7.92	3.43	5.37	3.43	7.59	3.73
Resp rate	31.00	30.199	32.60	28.60	26.73	28.00
	Low AS		Medium AS		High AS	
Masks	4.07		3.58		3.93	
Treatment time	3.81		3.72		4.13	

Note: AS = anxiety sensitivity, Sat = oxygen saturation, Vas = subjectively rated dyspnea, Resp rate = respiratory rate.

Table 7.
Means for the Panic-Fear Intensity Groups on Variables Assessed During the Index Attack

	Low P-F		Medium P-F		High P-F	
Measure	Intake	Reassess	Intake	Reassess	Intake	Reassess
Heart rate	127.14	135.50	119.41	134.75	110.28	127.93
Sat	92.29	92.57	92.58	94.50	92.21	93.31
Vas	6.79	2.98	6.08	3.50	7.77	3.92
Resp rate	33.78	31.67	28.00	26.55	30.00	29.39
	Low P-F		Medium P-F		High P-F	
Masks	4.14		4.14		3.15	
Treatment time	3.71		4.34		3.66	

Note: P-F = panic-fear intensity,Sat = oxygen saturation, Vas = subjectively rated dyspnea, Resp rate = respiratory rate.

were significantly related to subjective asthma symptomatology. In order to determine whether there were significant group differences on these self-report measures, as well as on trait anxiety, a one-way MANOVA was conducted,

The one-way MANOVA with CASI classifications of "low," "medium," and "high" as the independent variable, and RCMAS, CASCL, and panic-fear intensity and frequency scores as the dependent measures revealed a significant difference between the groups according to Wilk's Lambda criterion ($p<.0001$). The significant MANOVA was followed by separate univariate analyses of variance (ANOVA's) for each of the dependent measures. The alpha level was set at $p<.0125$ according to Bonferroni criteria. The results of the ANOVA's indicated that the groups differed significantly on each of the four variables (RCMAS: $F[2,37]= 5.97$, $p<.01$; CASCL: $F[2,37]= 18.78$, $p<.0001$; panic-fear intensity: $F[2,37]= 8.03$, $p<.001$; panic-fear frequency: $F[2,37]= 23.59$, $p<.0001$).

Post hoc analyses were conducted using Tukey's test. The analyses revealed that high AS subjects reported significantly more anxiety and asthma symptomatology than either of the other two groups. In addition, they experienced more intense and more frequent asthma panic-fear. Table 8 lists the ANOVA results and group means for each of the dependent measures.

Table 8.

Means and Analysis of Variance Results

	Low AS	Medium AS	High AS	F	P
Measure	m (s.d)	m (s.d)	m (s.d)	(2,37)	
RCMAS	42.36 (+ 6.42)	46.75[a] (± 6.84)	52.50[b] (± 9.59)	5.97	.006
Panic-fear intensity	19.10[a] (± 3.71)	24.83[a] (± 6.49)	31.36[b] (± 11.71)	8.03	.001
CASCL	102.43[a] (± 12.60)	105.58[a] (± 26.30)	158.36[b] (± 36.15)	18.77	.000
Panic-fear frequency	22.57[a] (± 6.17)	29.42[a] (± 13.06)	50.32[b] (± 12.96)	23.59	.000

Note: Means with different superscripts are significantly different at $p<.05$.

Summary of Results

A majority of children of children in the sample appeared to have asthma of mild to moderate severity. Whereas 56 per cent required both salbutamol PRN and an inhaled steroid to control asthma symptoms, 16.7 per cent also took regularly prescribed oral corticosteroids. In the twelve months preceding the index attack, two thirds of the sample had received emergent care on two or fewer occasions. Over the same period, the identical proportion had missed seven or fewer days of school because of asthma. In terms of treatment for the index attack, the average child received fewer than four salbutamol masks and remained in the emergent care setting for less than four hours. Most (73.8 per cent) were discharged home after treatment; the remaining 26.2 per cent were admitted as inpatients. With respect to asthma knowledge and self-care, few subjects displayed any marked deficits in this area. Clinical range scores on measures of behavioural competence and problems were comparatively rare (2.4 per cent of the sample for the former and 16.8 per cent for the latter). Borderline clinical scores were more common, with 22.5 per cent of the sample attaining scores in this range on the former scale and 12 per cent on the latter. 9.6 per cent of the sample had significantly elevated levels of trait anxiety. As for anxiety sensitivity, the mean score of 29.53 on the CASI was

somewhat higher than the 26.38 that has been reported previously for non-clinical samples. 35 per cent of the sample obtained scores of 30 or greater on this measure.

Correlation analyses revealed that objective indices of pulmonary functioning, whether assessed during the index attack or at follow-up, were not significantly associated with panic-fear frequency or intensity nor with anxiety sensitivity scores. They further suggested a lack of agreement between objective and subjective assessments of asthma severity. They did, however, point to significant relationships between anxiety sensitivity scores and panic-fear frequency and intensity, and self-reported asthma symptomatology. Trait anxiety scores were also positively correlated with panic-fear frequency and with asthma symptomatology.

The regression of panic-fear intensity on subjectively and objectively assessed asthma severity, trait anxiety and CASI scores indicated that the latter was the only significant predictor of the dependent measure. Similarly, CASI scores emerged as the only significant predictor of panic-fear frequency. The results of a series of MANOVA's suggested that, during the index attack, high anxiety sensitive children were indistinguishable from children with low or moderate levels in terms of heart rate, oxygen saturation levels, number of salbutamol masks administered, or time spent in the

emergent care setting. A significant time effect suggested that all of the children followed a similar course of recovery across the treatment period. With respect to respiration rate no significant differences between the groups emerged, however the high anxiety sensitive group evidenced a slight increase on this measure whereas the other two groups showed a decline. The analyses were replicated with the children grouped according to their panic-fear intensity scores. The results followed a similar pattern, however all three panic-fear groups followed the same direction of change in respiration rates. A one-way MANOVA indicated significant differences between the high versus low and medium anxiety sensitive on several of the self-report measures. Subjects with high scores on the CASI, as compared to those with low or moderate scores, were more anxious, endorsed more asthma symptomatology, and experienced more intense and more frequent asthma panic-fear.

DISCUSSION

The primary objective of the present study was to examine the relationship between anxiety sensitivity, the fear of specific psychophysiologic sensations arising from the belief that such sensations represent danger or threat, and asthma panic-fear. Preliminary work has suggested that the former is predictive of the latter in adult asthmatic patients comorbid for panic disorder (Carr et al., 1994); this study aimed to extend existing knowledge by exploring the nature of this association in children with asthma, a population wherein panic-fear is quite common but panic disorder is rare. The specific questions addressed concerned the relative contribution of anxiety sensitivity to the prediction of panic-fear intensity and frequency, and the severity of acute asthma as well as the subjective experience of asthma in children with varying levels of anxiety sensitivity.

To begin with the prediction of panic-fear intensity and frequency, a growing body of literature has indicated that anxiety sensitivity is strongly associated with fearfulness and panic in both clinical and non-clinical samples and, moreover, that it influences the subjective response to panicogenic agents. It was therefore anticipated that anxiety sensitivity would prove to be a powerful predictor of asthma panic-fear. With regards to panic-fear intensity in

the index attack, subjective and objective indices of dyspnea severity and trait anxiety combined explained a non-significant 18 per cent of the variance in this dependent measure. Anxiety sensitivity made a significant contribution to the prediction of panic-fear intensity, and boosted the variance accounted for to 30 per cent. Turning to panic-fear frequency, 25 per cent of the variance in this variable was attributable to the combination of trait anxiety, knowledge of asthma, asthma self-management, and residual airways obstruction. Anxiety sensitivity alone explained a further 30 per cent of the variance in panic-fear frequency.

The present findings are therefore in accord with research which has shown that anxiety sensitivity is an important determinant of the panic experience. They are also in accord with findings indicating that anxiety sensitivity predicts panic better than does trait anxiety. Rapee and Medoro (1994), for example, conducted a series of studies examining the relative contribution of anxiety sensitivity versus trait anxiety to the panic response. All of the studies involved exposing non-clinical samples to a hyperventilation challenge. The results clearly demonstrated that anxiety sensitivity was a better predictor of the response to hyperventilation than was trait anxiety. In interpreting their results, Rapee and Medoro (1994), discussed the distinction between measures of concern about specific threats, such

as the ASI, and measures that encompass a variety of concerns, such as trait anxiety scales. They concluded that measures of the latter type may be less useful than are the former in explaining the response to specific stressors. The ASI and the CASI, as measures of physical threat, would be expected to have greater utility in circumstances where concerns regarding physical harm are likely to be activated. Such circumstances may include biological challenge situations (Rapee & Medoro, 1994) as well as asthma exacerbations.

Rapee and Medoro (1994) construed their results as being compatible with Lilienfeld et al.'s (1993) model of the relationship between anxiety sensitivity and trait anxiety. According to these authors, trait anxiety, as the higher-order factor, should more closely reflect negative affect across a variety of situations. Anxiety sensitivity, as a lower-order factor, should reflect reactivity to a fairly circumscribed set of stressors; those involving physical threat (Rapee & Medoro, 1994). The current study yielded findings that appeared to follow this pattern, and hence may also be taken as evidence in support of Lilienfeld et al.'s (1993) hierarchical model.

To turn to the question of attack severity, the results of a series of MANOVA's yielded no indication of worse asthma in high anxiety sensitive children. Oxygen saturation and heart rate values were similar in the high, moderate and low anxiety sensitive groups.

Furthermore, the groups did not differ in terms of the number of salbutamol masks they received nor in duration of treatment. A significant time effect suggested a comparable course of recovery across the three groups. For all subjects, the response to treatment was demonstrated through significant increases in heart rate and decreases in self-reported dyspnea. Oxygen saturation values did increase, but not to a significant extent. Although there were no significant differences between the groups on respiration rate the direction of change varied, with the high anxiety sensitive children evidencing a small increase on this measure and the low and moderate groups a slight decline. When the MANOVA's were replicated using panic-fear intensity as the grouping variable similar results emerged. The high panic-fear group, however, did not show an increase in respiration rate across time.

There have been relatively few studies conducted in the emergent care setting which have examined both physiologic and psychologic responses to acute asthma. Gift (1991) studied adult asthmatics undergoing emergent care for the purpose of identifying variables associated with severe dyspnea. She found that her subjects had decreased oxygen saturation values and increased respiration rates pre-treatment as compared to post-treatment. She also observed higher scores on measures of state anxiety, depression

and on the ASC upon intake versus at reassessment. Gift (1991) did not assess anxiey sensitivity nor did she group patients according to their panic-fear scores. Rather, she examined differences between subjects obtaining high and low full-scale ASC scores. She reported that the groups were indistinguishable from one another in terms of oxygen saturation levels, respiratory rate, accessory muscle use, peak expiratory flow rate, pulse rate, state anxiety and depression.

Neither the current investigation nor that of Gift (1991) provided evidence to suggest a physiologic basis for the increased asthma symptom reporting apparent in some subjects. This lack of physiological differences between patients reporting heightened fearfulness or greater symptomatology is broadly consistent with results from the panic provocation literature. A number of challenge studies have suggested that panickers and non-panickers have the identical physiologic response to provocation agents. Panickers can, however, be differentiated by means of their tendency to interpret provocation-induced somatic sensations in a catastrophic manner (Smoller et al., 1996), a propensity that some authors attribute to enhanced anxiety sensitivity (Reiss, 1991). Catastrophic cognitions with regards to respiratory symptoms have been documented in pulmonary patients, including those with asthma, who report a history of panic attacks (Porzelius et al., 1987). Moreover, asthma patients

who panic have higher anxiety sensitivity but no greater pulmonary dysfunction than non-panicking asthmatics (Carr et al., 1994). Thus panic in response to worsening asthma may have little to do with the degree of bronchoconstriction present, but may rather be a function of factors such as anxiety sensitivity that can influence symptom interpretation.

The final research question concerned the subjective experience of asthma in children with varying levels of anxiety sensitivity. In addition to experiencing more frequent and more intense asthma panic-fear, high anxiety sensitive children also endorsed more symptoms reflecting physical distress, hyperventilation and irritability than did the other children. These results are compatible with findings in the panic provocation literature which indicate that subjects with increased anxiety sensitivity report more intense somatic sensations and affective symptoms, post-challenge, than do subjects with moderate or low amounts of this individual difference variable (Holloway & McNally, 1987; Shostak & Peterson, 1990; Koszycki et al., 1993; Asmundsen & Norton, 1994; Rapee & Medoro, 1994).

The present results also appear to correspond with the notion that anxiety sensitivity may serve to amplify the response to stress (Shostak & Peterson, 1990; Taylor 1995). This study yielded no

evidence to suggest worse asthma in the high anxiety sensitive children. Similarly, challenge studies have failed to document greater physiological reactivity in physically healthy high anxiety sensitive subjects. The observed increase in symptom reporting may therefore indicate heightened self-focused attention in the presence of elevated anxiety sensitivity. Self-focused attention refers to an anxiety driven narrowing of the attentional field (Barlow, 1988) which may serve to decrease the salience of external cues and enhance the awareness of internal states (Shostak & Peterson, 1990; Cioffi, 1991). When specific bodily sensations are associated with negative affect, such as may be the case for individuals who interpret them as sources of danger or threat, attention may be more easily deflected inwards (Cioffi, 1991). Thus high anxiety sensitive patients may have a lower threshold for noticing physical changes. The finding that high anxiety sensitive chronic back pain patients, independent of level of pain severity, experience more subjective distress and take more analgesic medications (Asmundsen & Norton, 1995) would certainly seem to imply that anxiety sensitivity can augment the experience of somatic events.

A secondary aim of this study was to determine whether thoraco-abdominal asynchrony, as measured by Respitrace, was a clinically useful index of severity in children with acute asthma. Due

to recording difficulties, there was missing data for a large proportion of the subjects. The recording difficulties were attributable, in part, to medication effects. Salbutamol tends to increase arousal level and can trigger hyperactive behaviour in children. As the children became more active they were often less amenable to the demands of the protocol. There was also considerable respiband slippage as activity levels increased. In addition, interference from other electrical equipment in the treatment room hampered the recording process. These technical problems, taken together with the lack of significant correlations between the available data and other variables of interest, would appear to argue against the utility of Respitrace assessments with this population in this type of treatment setting.

In sum, this study's findings have yielded results broadly consistent with the anxiety sensitivity variant of the cognitive model of panic. Specifically, the findings have suggested that anxiety sensitivity can mediate the subjective experience of pediatric asthma. High anxiety sensitive children are more likely to experience panic in the context of worsening asthma, and are also more likely to endorse a wide variety of asthma-related symptoms. They do not appear to have more severe asthma than their low or moderately anxiety sensitive counterparts. It would seem then, that anxiety sensitivity influences subjective reactions in situations of physical threat. It

appears not to be associated with other types of concerns, nor does it reflect actual biologic dysfunction. This pattern is compatible with conceptualizations of anxiety sensitivity as a lower-order factor of trait anxiety (Lilienfeld et al., 1993; Rapee & Medoro, 1994) and as a construct that can render specific psychophysiologic sensations more aversive (Shostak & Peterson, 1990).

Although not specifically addressed in the present study, the current results may have some bearing on Fritz & Overholser's (1989) question of whether asthma panic-fear should best be viewed as a transient state, influenced by disease severity, or a more enduring dispositional factor that predisposes an anxious response to certain somatic sensations. Early research involving adults with asthma failed to resolve this issue, as it provided evidence compatible with both positions.

Kinsman and his collaborators, in addition to developing the ASC, also constructed a 15-item scale derived from the MMPI to assess the "Panic-Fear personality" (Dirks et al., 1977). The items selected for inclusion were those that correlated most highly with the ASC panic-fear symptoms (Staudenmayer, Kinsman, Dirks, Spector & Wangaard, 1979). Notwithstanding the considerable overlap between the scales, they were held to capture different aspects of the panic-fear phenomenon. Whereas higher scores on the ASC panic-fear scale

were presumed to reflect a state-like symptom-focused anxiety, higher scores on the MMPI scale were assumed to represent a stable personality trait (Dirks et al., 1977; Staudenmayer et al., 1979).

Subjects with elevations on the ASC panic-fear scale could be distinguished, behaviourally, by means of their heightened attention to asthma symptoms. This hypervigilance was held to be adaptive in so far as it resulted in appropriate steps being taken to counter asthma (Staudenmayer et al., 1979). Yet it could also be maladaptive under some circumstances, as suggested by the observed positive relationship between ASC panic-fear scores and frequency of requests for PRN medications (Dahlem, Kinsman & Horton, 1977). Overuse of PRN's can lead to a state of extreme arousal that may be interpreted as worsening asthma (Yellowlees & Kalucy, 1990), it has also been implicated in some asthma mortalities (Fritz, Rubinstein & Lewiston, 1987).

With respect to the MMPI measure of asthma panic-fear, high scorers have been described as sensitive, dependent, fearful and inclined to over-report asthma symptoms (Dirks et al., 1977). They have further been described as intensely anxious in the acute asthma phase, pessimistic about their ability to manage their condition, and over-reliant on medical personnel for routine asthma care. As was the case for subjects with elevated ASC panic-fear scores, high scorers on

the MMPI scale also appeared to be inclined to overuse PRN's (Kinsman, Dirks & Jones, 1980).

In an examination of the interaction between the ASC and MMPI components of asthma panic-fear Dirks, Fross & Evans (1977) noted that approximately 50 per cent of subjects with significant elevations on the one instrument had a correspondingly high score on the other measure. The same was also true for subjects with extremely low scores. The investigators further discovered that the observed relationship between ASC panic-fear scores and medication regimes disappeared when they removed the variance attributable to MMPI panic-fear scores. They concluded that the ASC subscale was associated with poorer outcomes in asthma only in so far as it was reflective of the underlying personality construct (Dirks, Fross, et al., 1977).

Although the concept of a "Panic-Fear personality" has little contemporary appeal, Dirk et al.'s (1977) results are useful in that they underscore the importance of considering both dispositional and situation-specific influences on asthma panic-fear. In the present study anxiety sensitivity, a dispositional construct, explained 30 per cent of the variance in panic-fear frequency versus 12 per cent for panic-fear intensity during the index attack. Moreover, the two aspects of asthma panic-fear shared a modest 30 per cent overlap in

variance and displayed different patterns of association with other variables assessed in this study. Such findings suggest that panic-fear intensity and frequency may be, to a certain extent, differentially determined. With regards to factors which mediate the panic response in a given attack, these may include contextual variables that can fluctuate widely across time and situations. Factors intrinsic to the child, such as anxiety sensitivity, appear to play a more prominent role in the tendency to experience panic in the context of all, or almost all, acute asthma episodes. To return to Fritz & Overholser's (1989) question, then, it may be that asthma panic-fear in children can be both situation-specific and dispositional.

The present study has yielded results that are, in large part, concordant with existing findings in the areas of asthma panic-fear and anxiety sensitivity and panic. However there are also some marked discrepancies between this investigation and previous studies. Contrary to the results presented by earlier researchers, panic-fear in this sample was not significantly related to subjectively and objectively assessed dyspnea, nor was it associated with asthma knowledge and self-management skills. There are a number of differences, however, between this and previous studies. To begin with sample composition, this study focused on children with asthma whereas others have used adult patients. Hence the possibility exists

that the correlates of panic-fear for the two subject groups are quite dissimilar.

A further source of disparity may lie in the manner in which the estimates of dyspnea intensity were obtained. In Carr et al.'s (1992) investigation, both panic-fear and dyspnea intensity were assessed by means of sub-scales of the ASC. Unlike the ASC, which incorporates five distinct factors, the CASCL has a three-factor solution. Items from the Bronchoconstriction sub-scale of the ASC load on a global Physical Symptoms factor on the CASCL (Fritz & Overholser, 1989). As there is no verbal scale available to assess the subjective experience of dyspnea in children, this study relied upon a VAS. VAS's are held to be valid, reliable and sensitive measures of symptom severity (Gift, 1989), but may be less adequate in terms of capturing subjectively perceived dyspneic sensations than are verbal descriptors. With regards to previous findings suggesting that panic-fear may be related to disease severity, Carr et al., (1995) constructed an index comprised of pulmonary function test results and ASC dyspnea frequency scores. Thus it is difficult to determine to what extent the observed relationship was due to the influence of actual pulmonary impairment versus subjectively experienced dyspnea.

As for asthma knowledge and self-management, few children in the present sample demonstrated any marked deficits in these areas. Although the range of scores for both instruments was quite wide, this was primarily a function of the one or two subjects with exceptionally low scores. This, taken together with the relatively small number of subjects in the sample, may have served to attenuate any relationship with panic-fear. The subjects in this study did seem to be more knowledgeable about asthma than has been the case in other child samples. Where the identical instruments have been used with similar age groups, mean scores have been rather lower. Parcel et al. (1980), for example, reported means on the KAQ ranging from 11.58 to 14.19 in his sample of 5 to 11-year-olds. Rubin, Bauman and Lauby's (1989) sample of 7 to 12-year-olds obtained mean scores of 19.68 on the KAQ and 58.40 on the ABAQ. Mean values for the current sample were 20.90 and 61.06, respectively.

Although one would suspect that increased knowledge should be accompanied by more effective self-care behaviours, the present study gave no indication that this is the case. Scores on the KAQ and the ABAQ failed to correlate. The results of an investigation conducted by Rubin et al. (1989) may help to explain the observed lack of association between the two measures. The authors noted a threshold effect in operation, such that lower levels of knowledge

influenced behaviour but higher levels did not. The ceiling was set at the 70 per cent level. This translates to a raw score of 18.9, which is smaller than the average score for the current sample.

The present results also contradict those of earlier investigations in which asthma panic-fear was found to be related to medication regimes and to psychosocial adjustment (Fritz & Overholser, 1989; Baron et al., 1992). In this study, neither panic-fear intensity nor frequency correlated with number of medications used or with scores on the CBCL. It should be noted, however, that Fritz & Overholser's (1989) positive findings were based on parent-rated panic-fear, and not on child reports. It is possible, then, that a parent's determination of how well a child copes with asthma may influence perceptions concerning overall adjustment as well as the need for medication. Research conducted by Perrin and his associates (Perrin, MacLean & Perrin, 1989) lends some support to this speculation, as it has shown a connection between parental ratings of children's asthma severity and psychological functioning. With regards to Baron et al.'s (1992) investigation, their sample was mainly comprised of severe asthmatics. By way of contrast, a majority of the children in the present study had mild to moderate asthma. Moreover, Baron et al.'s panic-fear ratings were based on a structured interview; they did not

use the CASCL. Thus their criteria for determining the panic-fear groups were likely not the same as those used here.

Overall, this study has suggested greater distress surrounding asthma in high anxiety sensitive children. It may be that these children represent a population doubly at risk. In the long-term, given the weight of evidence connecting anxiety sensitivity with psychological morbidity, they may be vulnerable to the development of panic disorder. Of more immediate concern, however, is the possibility that undetected full-symptom panic attacks could interfere with asthma care. It would seem prudent, then, to advocate the use of some form of adjunct therapy with these at-risk children in order to ameliorate current distress and avert future problems. Baron and Marcotte (1994) have presented an extreme example of the kind of difficulties panic-prone asthmatic children can encounter. They described a 6-year-old boy with 13 hospital admissions for asthma over the course of a three year period. Despite an intensive medication regime he would wake frequently throughout the night with respiratory difficulties. During a further hospital admission it was determined that his nocturnal asthma was, in fact, nocturnal panic. Subsequent treatment with anxiolytic medications and cognitive-behavioural therapy resulted in a significant improvement of his medical condition.

The current research has several limitations that must be acknowledged. Firstly, the number of subjects was quite small, which gives rise to a consequent increase in the probability of committing Type II errors. Secondly, because of the nature of the recruitment procedure the sample was highly select and the results may therefore not be generalizable to the wider population of pediatric asthma patients. Indeed, the higher mean knowledge score of subjects in this study as compared to subjects in earlier investigations does raise the issue of key differences between this and other samples of children with asthma. Thirdly, the use of self-report instruments to assess the principal variables does allow for response bias potentially to inflate the observed relationships (MacCarthy & Brown, 1989). Finally, in terms of increasing our understanding of the origins of panic-fear and anxiety sensitivity, it was, perhaps, an oversight not to have enquired about past experience of status asthmaticus in the child subjects and excessive anxiety in their parents.

In summary, the present study has generated results consistent with a role for anxiety sensitivity in mediating the symptomatic presentation of pediatric asthma. Anxiety sensitivity is strongly related to panic-fear frequency and subjective asthma symptomatology and, to a lesser extent, to panic-fear intensity. The differences in the strength of the observed relationships imply that

panic-fear intensity may be, in part, contextually driven. By way of contrast, panic-fear frequency appears to be largely determined by individual difference variables such as anxiety sensitivity. The results further imply that it is possible to identify, by virtue of elevated scores on the CASI, a subgroup of asthmatic children who may be at risk for adverse medical and psychological outcomes. The results do not support the notion that Respitrace assessment of thoraco-abdominal asynchrony is a clinically useful tool in the acute exacerbations of pediatric asthma.

Future research may further the understanding of the relationship between asthma panic-fear and anxiety sensitivity by examining the origins of the latter variable in the asthma population. Carr et al. (1994), for example, have speculated that increased anxiety sensitivity in asthmatic patients may be a function of past experience with extreme respiratory distress. There is a substantial body of evidence, gathered over many decades, which indicates that fear may be conditioned to somatic sensations. The phenomenon, known as interoceptive conditioning, is remarkably resistant to extinction and likely to produce an exquisite sensitivity to the presence of the feared somatic cues (Barlow, 1988). Thus it is entirely possible that severe asthma may serve to sensitize patients to respiratory changes to such an extent that future attacks provoke

panic. This could be particularly true for children, who may have less well-developed asthma management skills than adults and would therefore be expected to feel more helpless and afraid during a rapidly progressing attack.

The possibility that parental actions may serve to condition fear to asthma symptoms is equally plausible. The highest incidence of asthma occurs during the first few years of life (Burrows, 1987), and parents who are forced to observe their infant or pre-schooler fight to draw breath may become understandably frightened. If parental fears continue unabated they may trigger over-protective behaviours. Parental over-protectiveness, in turn, could ultimately result in a child with maladaptive beliefs and excessive anxiety surrounding asthma. The results of family studies have indeed drawn a connection between an enmeshed, over-protective parenting style, and intractable asthma and panic-fear in children (Minuchin et al., 1979; Gustafsson et al., 1987; Baron et al., 1992).

It is likely that there are multiple pathways to anxiety sensitivity in asthma. Indeed, anxiety sensitivity theorists propose that both biology and the environment play a role in its genesis (Shostak & Peterson, 1990). Obtaining detailed information from patients and parents on past history of status asthmaticus and family attitudes towards asthma should therefore provide some insight into

the origins of anxiety sensitivity in asthmatic patients. Prospective studies which follow children with asthma from the point of diagnosis may also prove helpful in this regard.

Although neither the current study, nor that of Carr et al. (1994), could shed light on the genesis of elevated anxiety sensitivity in the asthma population both did, however, attest to its detrimental effects. Carr et al. (1994) observed enhanced anxiety sensitivity in asthmatic patients comorbid for panic disorder; in the absence of comorbidity, subjects with asthma could not be differentiated from normal controls in terms of their scores on the ASI. By way of contrast, subjects in the present study demonstrated heightened anxiety sensitivity but little in the way of documented psychopathology. Nevertheless, the high anxiety sensitive subjects did appear to be more distressed than were others in the sample. Elevations on the CASI were accompanied by corresponding increases in scores on measures of trait anxiety and subjective asthma symptomatology, as well as by increases in panic-fear intensity and frequency.

If elevated anxiety sensitivity does indeed put children at risk for adverse outcomes, then there is a clear need for research into the efficacy of adjunct therapies in this subject group. Cognitive-behavioural therapy, the treatment of choice for panic disorder

(Smoller et al., 1995), has already proven its worth in the management of intractable asthma (Baron and Marcotte, 1994). It may be helpful with anxiety sensitive asthmatic children if used to target excessive fears and unrealistic beliefs surrounding asthma symptoms. Care must be taken, however, to ensure that children maintain an appropriate level of attention to asthma symptoms. Effective asthma self-management depends upon the ability to identify prodromal symptoms (American Institute for Research, 1984). As the negative emotions that may accompany such bodily signals are known to be an impediment to effective coping (Cioffi, 1991; Bakal, Hesson & Demjen, 1995), it would appear to be vitally important to teach children to recognize and regulate their affective response to asthma symptoms. Involving parents in treatment would also seem to be warranted, as parental fears can easily inflate child anxiety.

In Baron and Marcotte's (1994) case study nocturnal panics were mistaken for nocturnal asthma attacks. The design of the current investigation did not allow for an exploration of the relationship between anxiety sensitivity and nocturnal asthma, however this does appear to be an area that requires further study. According to parental reports, almost one third of the children in the present sample routinely experienced nocturnal asthma. In terms of advancing our understanding of asthma panic-fear, then, it may be

useful to determine whether night-time attacks are more likely to trigger panic and whether high anxiety sensitive children have more frequent nocturnal asthma.

One group of subjects that deserves further investigation is composed of subjects with extremely low scores on measures of anxiety sensitivity. Although the present findings provided no grounds for differentiating between low and moderate anxiety sensitive subjects, previous research has. Shostak and Peterson (1990), in an attempt to understand the differences apparent in their investigation, speculated that low anxiety sensitive individuals may fail to process physiological arousal. Alternatively, they may fail to find it aversive. It is well-known that some asthmatic patients routinely disregard or minimize symptoms, and are consequently at greater risk for fatal or "near-miss" asthma attacks (Steiner et al., 1987; Yellowlees & Kalucy, 1989). It may be that this behaviour reflects the outward manifestation of an underlying low anxiety sensitive disposition in these patients.

In order to attain a more complete understanding of the relationship between anxiety sensitivity and asthma management it may be useful to study a sample of emergent care "repeaters." Repeaters are those adults and children who make numerous visits to the emergency department for treatment of asthma. It is possible

that the relationship between anxiety sensitivity and repeat visits may be curvilnear. High anxiety sensitive asthmatics would likely be repeaters because of their excessive concern surrounding asthma. Low anxiety sensitive subjects may be repeaters because their disregard of symptoms in the early stages of acute asthma could put them at increased risk for frequent, severe exacerbations.

It may also prove fruitful to examine the contribution of other sensitivities to panic-fear in asthma. According to Reiss' (1991) expectancy theory, sensitivities are the beliefs that predispose an individual to fear an anticipated event. In addition to anxiety sensitivity, Reiss describes two other "fundamental fears," injury/illness sensitivity and social evaluation sensitivity. The three fundamental fears, together with their corresponding expectancies, are presumed to be the basis for anxiety, phobias and panic (Reiss, 1991; Taylor, 1995). Whereas anxiety sensitivity has generated considerable research interest, the injury/illness and social evaluation sensitivities have been less well-studied. Injury/illness sensitivity could be of relevance to panic-fear in that individuals who fear incapacitation from asthma would be substantially more likely to become anxious when symptomatic. Moreover, social evaluation sensitivity could have explanatory value under some circumstances for individuals who fear public embarrassment or shame.

Although the present study provided no evidence to suggest significant physiologic differences between patients with varying levels of anxiety sensitivity or asthma panic-fear this is clearly an area that needs additional investigation. Preliminary results from trials of experimental procedures to treat asthma have suggested that techniques which decrease facial muscle tension produce improvement in pulmonary function (Glaus & Kotses, 1983; Loew, Siegfried, Martus, Tritt & Hahn, 1996). Such results may be consistent with a role for increased vagus nerve activity in some patients with asthma. Isenberg, Lehrer and Hochron (1992) have noted that emotionally triggered asthma attacks predominantly affect the upper airways and further, that patients with upper airways obstruction are more responsive to relaxation therapy. As constriction in the upper airways is vagally mediated, and as the vagus nerve communicates with nerves controlling the muscles of the face, these authors have speculated about a potential relationship between vagal activation and psychological factors in asthma. Given that anxiety sensitivity and panic-fear appear to be important determinants of the affective response to asthma, they may be worthwhile considering in future tests of Isenberg et al.'s (1992) vagal hypothesis.

Finally, the small increase in respiratory rate evidenced by the high anxiety sensitivity group is also of interest. This increase may

reflect anxiety driven respiratory changes. It could also represent slower recovery from stress, as observed by Shostak and Peterson (1990) in their high anxiety sensitive subjects. In order to better explore respiratory differences between low, medium and high anxiety sensitive subjects, further work in this area should be conducted in a larger sample that includes patients with severe asthma as well as those with mild or moderate conditions.

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APPENDIX 1.

Children's Asthma Symptom Checklist

The following is a list of things children sometimes feel during asthma attacks. For each item, tell me whether it never, almost never, sometimes, almost always, or always applies to your asthma. Remember, respond to each item of this list in regard to its ability to describe how you feel during an asthma attack.

Rating scale:

1	2	3	4	5
never	almost never	sometimes	almost always	always

1. Cramps
2. Panting, fast breathing
3. Numb, no feeling
4. Sticky, mucous in lungs
5. Cranky
6. Get angry easily
7. Hard to breathe
8. Headache
9. Nervous, jittery
10. Frightened*

11. Uncomfortable
12. Short of breath
13. Heavy feelings in chest
14. Afraid of being alone*
15. Afraid of dying*
16. Unhappy with things
17. Heart pounding
18. Dizzy
19. Worn out
20. Panicky*
21. Weak
22. Pins and needles
23. Hard and fast breathing
24. Don't care about things
25. Feel like you're alone*
26. Wheezy
27. Worried about the attack*
28. Tingly in spots
29. Very angry, mad
30. Chest tightening
31. Tired

32. Scared*

33. Feel helpless*

34. Chest filling up

35. Lonely*

36. Worried*

37. Chest pain

38. Rundown, weak

39. Mad at the world

40. Coughing

41. No energy

42. Unhappy*

43. Worried about myself*

44. Worried about asthma*

45. Worried in general*

46. Feel left out*

47. Breathe quickly

*panic-fear items

APPENDIX 2

Asthma Behavioral Assessment Questionnaire

I'm going to read you some sentences about things children might do if they have asthma. For each sentence I would like you to tell me how often you do it, never, once in a while, about half the time, most of the time or always.

Rating scale:

1	2	3	4	5
never	once in a while	about half the time	most of the time	always

1. I stay away from things that cause breathing problems
2. When having trouble breathing I get away from what's causing it
3. I do breathing exercises when I have trouble breathing
4. I try to make myself relax when I have trouble breathing
5. I try to get my mind off my breathing when I have breathing problems
6. I try something else when I have trouble breathing and the first thing I try doesn't work
7. I seek help from other people at the first sign of breathing problems

8. I let friends talk me into doing things that could cause breathing problems*
9. I stay calm when I am having breathing problems
10. I let adults make me do things that could cause breathing problems*
11. If I have an asthma attack at night I take it easy the next day
12. I tell my friends that I have asthma
13. I stop playing and take it easy when I start to have breathing problems
14. (if you are supposed to take allergy shots) I forget to get allergy shots on schedule
15. I keep asthma medicine handy at home, school, and away from home
16. I take the correct medicine when breathing problems begin
17. I take the correct medicine on my prescribed schedule

*items are reverse scored

APPENDIX 3

Knowledge of Asthma Questionnaire

I'm going to read some sentences. If the sentence is right, say "Yes." If the sentence is wrong, say "No." Even if it is very hard to decide, be sure to say yes or no for every sentence.

If you have asthma

1. You are always sick*
2. You have to stay indoors to play*
3. Your body parts for breathing sometimes do not work right
4. You should not talk about your feelings such as being afraid, angry or worried*

Asthma attacks can happen because

5. You can be allergic to things like dust, pollen or animals
6. You can get infections like colds, flu or sore throat
7. You breathe things like paint fumes, gasoline, smoke or pollution
8. You get upset or laugh too hard

Doing something to keep an asthma attack from happening

9. Is not possible*

10. Might be possible by staying away from things that cause attacks

11. May be helped by doing special breathing exercises

12. Is something only a doctor can do something about*

When I have an asthma attack

13. I have trouble getting air out of my lungs

14. The tiny air tubes inside my lungs open up and become wider*

Taking asthma medication for wheezing

15. Can be used to keep an asthma attack from happening

16. Can be used to stop an asthma attack after it starts

17. Is something children can do to learn to help themselves

18. Is to relax the tightness of the tiny air tubes

If you start to have an asthma attack

19. You might notice coughing before wheezing starts

20. You might notice a tight feeling in your chest before wheezing starts

21. You should only take medicine after you start wheezing*

When you have an asthma attack

22. You can do nothing to try to stop the attack*

23. Your parents must rush you to the hospital before doing anything else

24. You can take medicine to stop the wheezing

25. You can relax by doing breathing exercises

26. You should try not to pay attention to wheezing and hope that it will go away*

27. You should drink lots of liquid like water

*items are reverse scored

APPENDIX 4

Children's Anxiety Sensitivity Index

I'm going to read you a number of statements which children use to describe themselves. For each statement tell me whether it does not describe you at all, describes you some, or describes you a lot.

Rating scale:

1	2	3
not at all	some	a lot

1. I don't want other people to know when I'm afraid*
2. When I cannot keep my mind on my school work I worry that I might be going crazy
3. It scares me when I feel "shaky"
4. It scares me when I feel like I am going to faint
5. It is important for me to stay in control of my feelings*
6. It scares me when my heart beats fast
7. It embarrasses me when my stomach growls (makes noise)
8. It scares me when I feel like I am going to throw up
9. When I notice that my heart is beating fast, I worry that there might be something wrong with me
10. It scares me when I have trouble getting my breath

11. When my stomach hurts, I worry that I might be really sick

12. It scares me when I can't keep my mind on my schoolwork

13. Other kids can tell when I feel shaky

14. Unusual feelings in my body scare me

15. When I am afraid, I worry that I might be crazy

16. It scares me when I feel nervous

17. I don't like to let my feelings show*

18. Funny feelings in my body scare me

*items are reverse scored

APPENDIX 5

Questions for Parents.

1. When was your child diagnosed with asthma (how old was he/she)?
2. Is your child more likely to have an asthma attack at a particular time of the year - for example in the Spring or the Fall?

   Does she/he have asthma attacks at any time throughout the year?
3. Does your child often wake up in the night with an asthma attack?

   (If yes)

   Do night-time attacks occur more often than daytime attacks?
4. Does your child have asthma attacks during or after exercise - for example when taking part in sports, physical education, or while running or playing?
5. Do strong emotions trigger attacks in your child - for example does she/he have attacks when upset, angry, happy or excited?
6. Does your child have any allergies or exczema?

   (If yes)

   What is he/she allergic to ?

In the last twelve months ....

7. How many asthma attacks has your child had?

8. How many times have you brought her/him to the hospital for treatment of asthma?

9. How many days of school has he/she missed (approximately)?