

## Psychological Aspects of Asthma

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Asthma can be affected by stress, anxiety, sadness, and suggestion, as well as by environmental irritants or allergens, exercise, and infection. It also is associated with an elevated prevalence of anxiety and depressive disorders. Asthma and these psychological states and traits may mutually potentiate each other through direct psychophysiological mediation, nonadherence to medical regimen, exposure to asthma triggers, and inaccuracy of asthma symptom perception. Defensiveness is associated with inaccurate perception of airway resistance and stress-related bronchoconstriction. Asthma education programs that teach about the nature of the disease, medications, and trigger avoidance tend to reduce asthma morbidity. Other promising psychological interventions as adjuncts to medical treatment include training in symptom perception, stress management, hypnosis, yoga, and several biofeedback procedures.

Asthma is a chronic respiratory disease of unclear etiology, characterized by reversible airway obstruction and heightened airway irritability usually accompanied by inflammation of tissues of the airways, mucus congestion, or constriction of airway smooth muscles (Busse & Reed, 1988; National Heart, Lung, and Blood Institute [NHLBI], 1997). Asthma flares are commonly triggered by allergens, airway irritants (frequently tobacco smoke), exercise, cold air, and viral infection (NHLBI, 1997).

### Measurement of Asthma

#### *Methodology for Assessing Presence and Severity of Asthma*

Asthma is usually diagnosed by periodic experience of asthma symptoms (primarily wheezing, nocturnal awakening from asthma, cough, difficulty breathing [*dyspnea*], and chest tightness), episodic decreases and variability in pulmonary function (primarily FEV<sub>1</sub>, FEV<sub>1</sub>/FVC, and peak flow),<sup>1</sup> and reversibility of these symptoms and signs or evidence of bronchoreactivity to methacholine or histamine (American Thoracic Society, 2000; Chai et al., 1975).

Kendall, Marrs-Garcia, Nath, and Sheldrick (1999) made the distinction between improvement and recovery following treat-

ment, assessed, respectively, by tests of statistical and clinical significance. Recovery is usually defined as movement into the normal range, although complete remission is sometimes not possible (Jacobson, Roberts, Berns, & McGlinchey, 1999), and small improvement can be clinically significant (Kazdin, 1999). Such is often the case with asthma. For asthma, there are two types of relevant changes: acute changes during an asthma flare and tonic changes in asthma condition. The clinical significance of a change can be conservatively defined as moving in or out of normal pulmonary function levels or the appearance or disappearance of asthma symptoms (NHLBI, 1997). None of the studies we reviewed used the exact criteria promulgated in NHLBI, primarily because these criteria appeared only recently.

#### *Assessment of Change During a Flare*

For patients' home use, the NHLBI's (1997) guidelines also defined three zones of urgency for clinical intervention, labeled according to the colors of a traffic signal. The green zone indicates normal function for that individual. In the yellow zone the patient is advised to take emergency medication and to contact his or her physician. In the red zone the patient is advised to take large doses of rescue medication and to go to an emergency room. According to these criteria, entering the yellow or red zone would constitute a clinically significant asthma exacerbation. For peak flow values,

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<sup>1</sup> FEV<sub>1</sub> is the volume of air exhaled during the first minute of a forced expiratory maneuver from full vital capacity (FVC). Peak flow is the maximum flow of exhaled air during such a maneuver. Both measures are routinely taken in the clinic using a spirometer. Inexpensive plastic devices can be used for home assessment of peak flow. Recent improvements in computer technology have led to the appearance of home assessment devices that can record a wide variety of pulmonary function measures and detect poor technique or submaximal effort (Burge et al., 1999; Izbickei, Abboud, Jordan, Perruchoud, & Bolliger, 1999), but no behavioral studies have yet appeared using them.

normal functioning is considered to be either 80% expected (according to norms for height, weight, age, sex, and ethnicity) or 80% of "personal best" (the highest values from twice-daily home peak flow values achieved in the past 2–3 weeks), along with absence of wheezing, chest tightness, or dyspnea (NHLBI, 1997). An improvement in FEV<sub>1</sub> of  $\geq 12\%$  and 200 mL is considered to be significant under ordinary circumstances, but a change to normal levels, although not obtainable by all patients, is the goal of treatment for an asthma flare and can be used as a conservative measure of clinically significant improvement.

#### *Assessment of Tonic Change in Asthma Condition*

Although the severity of disease status is usually considered to be a trait measure, assessed prior to treatment, it is possible to use change in current status of asthma severity as a measure of change. NHLBI's (1997) guidelines defined a four-step classification system of asthma severity: mild intermittent, mild persistent, moderate, and severe. The level is based on a complex index of two domains: symptoms and pulmonary function. Disease severity is determined by the domain with the greater severity. One could consider clinically significant a change of at least one category in severity.

#### *Adjustment for Medication*

Where a psychological intervention is proposed as a substitute for asthma medication or as a method for safely reducing medication consumption, medication use must be factored in as part of assessment of asthma severity. Because daily dosage of asthma "controller" medication is specifically tied to asthma severity (NHLBI, 1997), medication consumption can be used in psychological studies as a third dimension for assessing asthma severity, particularly in studies where behavioral strategies may be proposed as substitutes for medication (e.g., in studies of biofeedback, yoga, or training in avoidance of asthma triggers). However, medication usage can be affected by idiosyncratic prescribing practices of physicians, some of whom may not rigorously follow NHLBI guidelines, and also by patients' level of compliance. This may not necessarily render impossible the task of scoring asthma severity, because total severity can be scored as the highest severity level of the three dimensions (symptoms, pulmonary function, and medication). Thus, an undermedicated patient will have more severe symptoms or worsened pulmonary function, which would increase the overall severity score. An *overmedicated* patient, however, may score as more severe than his or her actual asthma, because of an overly high score on the medication dimension. This can be a significant problem for asthma researchers, because many asthma specialists deliberately overmedicate symptomatic asthma patients at the beginning of treatment and then gradually titrate medication downward. This is particularly the case after a severe asthma flare, when asthma is usually treated with high doses of oral steroids for several weeks, which are only gradually titrated downward, even when symptoms disappear immediately. Thus, a high level of asthma medication is not always a sign of more severe current asthma condition.

For these reasons, we recommend that in such studies the patient's medicine gradually be reduced to the minimum required to ensure stability (defined as optimal pulmonary function without

any asthma symptoms) before a psychological intervention is applied. The effect of the intervention can then be assessed by the ability to titrate the medication further downward without reappearance of symptoms or impaired pulmonary function. However, this strategy may be impossible for studies examining behavioral triggers of asthma. Also, in psychological studies of self-care behavior, medication use may be a legitimate dependent variable, so adjusting for it would obviously be inappropriate.

#### *Quality of Life*

Measures of asthma quality of life also can be useful for assessing changes in asthma. Current reviews of various asthma quality of life instruments are maintained on the Web site of the American Thoracic Society's Behavioral Science Assembly ([www.thoracic.org](http://www.thoracic.org)). Although these measures are mostly not specifically tied to NHLBI's categories for disease severity, quality of life (severity of symptoms and impairment in daily function) does contribute to NHLBI's criteria.

#### *Asthma and Vocal Cord Dysfunction*

Asthma is often confused with paradoxical vocal cord dysfunction, in which the vocal cords constrict during inhalation rather than exhalation. In such cases spirometric measures of flow during inhalation are usually more impaired than those during exhalation (Newman & Dubester, 1994; Newman, Mason, & Schmalning, 1995). There appears to be a major psychological contribution to this problem (Gavin, Wamboldt, Brugman, Roesler, & Wamboldt, 1998; F. S. Wamboldt, 1998).

### Psychological and Psychophysiological Correlates of Asthma

#### *Stress, the Autonomic Nervous System, and the Exacerbation of Asthma*

Asthma patients tend to show greater bronchoconstriction than healthy controls in response to stress, both in the laboratory (B. D. Miller & Wood, 1994) and in everyday life (Affleck et al., 2000; Ritz, Steptoe, DeWilde, & Costa, 2000; Schmalning, McKnight, & Afari, in press). Stress-induced asthma exacerbation may be mediated by changes in autonomic function. Beta-sympathetic activation produces bronchodilation, whereas alpha-sympathetic activity and parasympathetic activity produce bronchoconstriction (Nadel & Barnes, 1984). Although mildly stressful tasks requiring active coping behaviors, such as mental arithmetic, tend to produce *bronchodilation* among both healthy individuals and those with asthma (Lehrer et al., 1996; Smyth, Stone, Hurewitz, & Kaell, 1999) and although patients with panic disorder, whether or not they have asthma, show lower respiratory resistance than corresponding psychiatrically normal groups (Carr, Lehrer, Hochron, & Jackson, 1996), other patterns of stress are associated with the opposite effects. Passive response to stress or embarrassment appears to trigger clinically significant bronchoconstriction in 20–40% of asthma patients (cf. review by Isenberg, Lehrer, & Hochron, 1992a). Stress-induced sympathetic activation is often followed by parasympathetic rebound after the stress abates (Lehrer, Hochron, et al., 1997; Manto, 1969). This may explain the frequent occurrence of nocturnal asthma symptoms (Ballard, 1999) and, perhaps, the entire phenomenon of stress-induced asthma.

Consistent with the well-known psychophysiological principle of individual response stereotypy (Lacey & Lacey, 1958), Feldman, Lehrer, Hochron, and Schwartz (2002) found that in response to various laboratory tasks, defensive<sup>2</sup> patients with asthma displayed a response pattern involving bronchoconstriction, lower skin conductance levels, and greater respiratory sinus arrhythmia amplitudes: an autonomic response pattern suggestive of increased parasympathetic and decreased sympathetic arousal. Defensive people without asthma tend to show increased sympathetic activity (S. B. Miller, 1993; Shapiro, Goldstein, & Jamner, 1995; Shapiro, Jamner, & Goldstein, 1993).

### *The Immune System and Stress-Related Exacerbation of Asthma*

NHLBI's (1997) guidelines define asthma as an immune system process, but do not include immunologic testing as part of asthma assessment. They note that the role of inflammation in asthma is "still an evolving concept" (NHLBI, 1997, p. 3). There is yet little published research on whether stress can exacerbate asthma directly via immune mechanisms. One recent study (Kang et al., 1997) reported a small increase in Th2 cytokine response profile among asthma patients during examination stress. They also found that IL-5 declined among healthy participants but not asthma patients during examination stress. The authors interpreted this pattern as suggesting a vulnerability to stress-related airway inflammatory reactions among people with asthma. Also, long-term exposure to stress can increase susceptibility to respiratory illnesses (Cohen, Tyrell, & Smith, 1991), which in turn can exacerbate asthma. A detailed review of stress-induced asthma and the current status of research on mediation by autonomic and immune processes has been published by Rietveld, Everaerd, and Creer (2000).

### *Asthma and Psychological Disorders: Anxiety and Depression*

Patients with asthma, especially children, appear particularly likely to suffer from psychological problems, particularly anxiety disorders (Bussing, Burket, & Kelleher, 1996; Vila et al., 1999; M. Z. Wamboldt, Schmitz, & Mrazek, 1998). Persons with asthma and comorbid psychiatric disorders have more impaired functioning in both emotional and physical arenas than persons with either disease alone, with poorer control of asthma (Afari, Schmalung, Barnhart, & Buchwald, 2001; Siddique et al., 2000) and greater health care utilization (ten Brinke, Ouwerkerk, Zwinderman, Spinhoven, & Bel, 2001) despite lack of differences in asthma severity (ten Brinke, Ouwerkerk, Bel, & Spinhoven, 2001). This association could occur either through disorganization of self-care behavior or by direct physiological effects of anxiety on the autonomic and immune systems. Elevated anxiety and depression have been found to be positively related to asthma severity in children (Mrazek, 1992) but not in adults (Afari et al., 2001). Those with asthma, especially children, also appear to be more likely than healthy individuals to experience negative emotions without expressing them (Hollaender & Florin, 1983; Silverglade, Tosi, Wise, & D'Costa, 1994). However, empirical data as to whether and how negative emotions precipitate or exacerbate asthma attacks are inconsistent (Lehrer, 1998).

### *Limitations of the Literature on Psychiatric Morbidity and Asthma*

Most studies rely on samples of patients from specialty pulmonology clinics and thus may oversample patients with problematic asthma, including some patients who may be overperceivers of asthma symptoms. Also, they tend to rely on self-report (and often only from a collateral source) of depressive or anxiety symptoms as a proxy for anxiety or mood disorders. In addition, many population-based studies have not adequately assessed asthma *per se* but rather have used measures of atopy, often from questionnaires about symptoms and symptom triggers, without using pulmonary function measures. Although the presence of atopy is associated with asthma, it is not clear to what extent these results can be generalized to individuals with accurately diagnosed asthma, especially because some triggers of asthma may not involve allergies (Pearce, Pekkanen, & Beasley, 1999).

### *Asthma, Panic Symptoms, and Panic Disorder*

Panic disorder appears to be overrepresented among patients with asthma (Carr, Lehrer, & Hochron, 1992; Carr, Lehrer, Rausch, & Hochron, 1994; Karajgi, Rifkin, Doddi, & Kolli, 1990; Shavitt, Gentil, & Mandetta, 1992; Yellowlees, Alpers, Bowden, Bryant, & Ruffin, 1987; Yellowlees, Haynes, Potts, & Ruffin, 1988). Approximately 1 asthma patient in 10 has panic disorder. Also, asthma and other chronic respiratory diseases are three times more common in those with panic disorder than among those with other psychiatric disorders or the general population (Spinhoven, Ros, Westgeest, & van der Does, 1994; Zandbergen et al., 1991). Among co-occurring psychiatric and respiratory disorders, panic disorder tends to be preferentially associated with asthma, whereas depression is found more often in irreversible airway disease (Kinsman, Fernandez, Schocket, Dirks, & Covino, 1983; Kinsman, Luparello, O'Banion, & Spector, 1973; Spinhoven et al., 1994). Furthermore, patients with high levels of generalized panic-fear have been shown to have higher rates of emergency room visits and general asthma morbidity (Nouwen, Freeston, Labbé, & Boulet, 1999). These findings are consistent with earlier work from the National Asthma Center (Dirks, Kinsman, Jones, & Fross, 1978). The causal direction between asthma and panic may be bidirectional.

Panic may elicit or exacerbate asthma symptoms by several pathways. As described above, the psychophysiological stress response that accompanies panic may elicit autonomic and inflammatory responses among people with asthma, and dyspnea and other unpleasant body sensations accompanying asthma may trigger panic. Although the poor correlation between asthma severity and panic symptoms (ten Brinke, Ouwerkerk, Bel, & Spinhoven, 2001) might argue against the latter pathway, there are reasons to believe that both mild and severe asthma symptoms might trigger panic, but by different pathways. Symptoms of mild asthma might more easily be confused with panic symptoms, whereas symptoms of more severe asthma are more recognizable and lead to a clearer path of coping behavior, thus decreasing the panicogenic effect.

<sup>2</sup> Defensiveness is a psychological trait associated with avoidance of threatening stimuli, minimization of negative affect, and impression management (i.e., a tendency to portray oneself in a socially desirable manner).

The frightening nature of severe dyspnea may evoke panic through classical conditioning. Consistent with the possibility that asthma can be a contributing cause of panic disorder are findings that where panic disorder and asthma are comorbid, the respiratory disorder typically precedes the onset of panic disorder (Perna, Bertani, Politi, Colombo, & Bellodi, 1997; Verburg, Griez, Meijer, & Pols, 1995).

Other pathophysiological events may elicit both disorders and, in turn, be elicited by them. Chief among these is hyperventilation, which commonly occurs in panic disorder (Hegel & Ferguson, 1997; Papp, Klein, & Gorman, 1993) and can induce sensations of dyspnea (Chonan, Mulholland, Leitner, Altose, & Cherniack, 1990; Hammo & Weinberger, 1999). Hyperventilation can produce unpleasant body sensations, the fear of which may contribute to panic in susceptible people (Chambless, 1984). Panic-fear is often associated with dyspnea in patients with asthma, and there is some evidence that the sequelae of dyspnea may contribute to panic attacks in at least a subset of panic disorder patients (Carr et al., 1992). Additionally, airway obstruction can lead to hyperventilation. Individuals with asthma tend to show an exaggerated increase in respiratory drive in response to experimentally induced respiratory resistance (Kelsen, Fleegler, & Altose, 1979). Because this response is measured during the first 100 ms of the occluded breath (i.e., before cortical processing of the occlusion can occur; Davenport, Friedman, Thompson, & Franzen, 1986) and before any observable cortical response, it probably is mediated by brain stem reflexes (Chapman, Santiago, & Edelman, 1980). This reflex, nevertheless, appears to contribute to hyperventilation independently of panic, although the hyperventilation symptoms, combined with fear of body sensations in susceptible people, may subsequently induce panic. Hyperventilation also tends to be very common in asthma (Thomas, McKinley, Freeman, & Foy, 2001), and it can cause bronchoconstriction through pathways of cooling and, to a lesser extent, drying of the airways (Gilbert, Fouke, & McFadden, 1988; Kilham, Tooley, & Silverman, 1979; McFadden, Nelson, Skowronski, & Lenner, 1999). The contribution of hyperventilation to panic disorder remains in dispute (Bass, 1997; Garsen, Buikhuizen, & van Dyck, 1996). It is not present in all cases of panic disorder and may be only an epiphenomenon when it does occur, and anxiety can be absent during hyperventilation (Bass & Gardner, 1985) or secondary to it (Lum, 1976). However, we believe that it provides a plausible mechanism for the common co-occurrence of the two disorders and should be a subject for further, more targeted, research. Its contribution as an asthma trigger has been better substantiated.

Another possible bidirectional pathway is shared respiratory dysregulation that may contribute to the pathophysiology of both problems (Smoller, Pollack, Otto, Rosenbaum, & Kradin, 1996). For example, the experience of dyspnea in both disorders may be linked by CO<sub>2</sub> sensitivity. Medullary chemoreceptors and the locus coeruleus may be stimulated by bronchoconstriction in asthma, inducing the expression of an underlying vulnerability to panic (Perna et al., 1997; Svensson, 1987). Repeated stimulation of chemoreceptors may lead to dysfunction of the brain's suffocation alarm system, posited by Klein (1993) to underlie the development of panic disorder. This mechanism may stimulate hyperventilation (Papp et al., 1993), thus exacerbating both panic and asthma.

The relationship between panic disorder and asthma is not a specific one. There is a higher prevalence of nonrespiratory dis-

eases, such as cardiovascular and cerebrovascular disease, among panic disorder patients than among those with other psychiatric disorders or those with no psychiatric disorder (Weissman, Markowitz, Ouellette, Greenwald, & Kahn, 1990). In addition, panic disorder and asthma seem to be independently transmitted in families of those with asthma (Perna et al., 1997). Nevertheless, Baron and Marcotie (1994) reported 20 cases where medical treatment of panic disorder in children with comorbid asthma has led to improvement in asthma, thus suggesting a causal connection.

### *Asthma and Depression*

The relationship between asthma and depression, although less well established, also may be bidirectional. B. D. Miller and Wood (1997) demonstrated that film-induced sadness can produce bronchoconstriction among children with asthma. Also, the well-known relationship between depression and an attitude of helplessness (Peterson & Seligman, 1984; Seligman, Abramson, Semmel, & von Baeyer, 1984) may create conditions for a passive behavioral response to stress, which appears to be particularly associated with vagal activation (Inamori & Nishimura, 1995; Roozendaal, Koolhaas, & Bohus, 1997). On the other hand, some of the common effects of asthma also can contribute to depression, particularly fatigue, disability, and self-perception as being sick. Bell, Jasnosi, Kagan, and King (1991) found more allergies and a higher rate of asthma among people reporting a greater number of depressive symptoms in a nonclinical sample of 379 college students. There also may be a genetic link between asthma and certain mood disorders (M. Z. Wamboldt, Weintraub, Krafchick, & Wamboldt, 1996; M. Z. Wamboldt et al., 2000).

### *Depression, Anxiety, and Life-Threatening Asthma Attacks*

A high prevalence of denial and anxiety has been found among asthma patients who have experienced a near-fatal attack (Campbell et al., 1995; Martin et al., 1995; Yellowlees et al., 1988; Yellowlees & Ruffin, 1989). Two studies found that children who died from asthma attacks had higher levels of psychosocial problems, including depressive symptoms and family dysfunction (B. D. Miller & Strunk, 1989; Strunk, Mrazek, Fuhrmann, & LaBrecque, 1985), although these latter findings were not replicated in another study (Barboni, Peratoner, Rocco, & Sabadini, 1997). Interpretation of psychological factors in near-death asthma has been limited by retrospective assessment because increases in anxiety or denial may be the result, and not the cause, of these severe asthma exacerbations. Strunk, Nicklas, Milgrom, and Ikle (1999) have recommended that prospective studies be done and a national database implemented for tracking characteristics of patients with fatal or near-fatal attacks.

### *Behavioral Conditioning and Asthma Symptoms*

Although not a newly observed phenomenon, the classical conditioning of respiratory symptoms has been the topic of considerable research in the last decade (see Ley, 1994, for a review). Van den Bergh and colleagues have shown that odors and other stimuli can serve as conditional stimuli for eliciting respiratory symptoms and complaints in healthy individuals (van den Bergh, Kempynck,



van de Woestijne, Baeyens, & Eelen, 1995) as well as among individuals reporting hyperventilation complaints (van den Bergh, Stegen, & van de Woestijne, 1997). Other studies have demonstrated conditioned respiratory responses to fear-relevant images or conditional stimuli associated with stress (Ley, 1994; D. J. Miller & Kotses, 1995; Stegen, De Bruyne, Rasschaert, van de Woestijne, & van den Bergh, 1999) and generalization of odor-conditioned responses to new odors (Devriese et al., 2000).

### Asthma Symptom Perception

The ability to detect changes in the condition of the airways may have important clinical implications for people with asthma. Underestimators may not take prescribed medications and may delay seeking medical attention, which could lead to disastrous consequences, whereas overperceivers may take excessive medication, experience side effects, and overuse health care resources. People tend to rely on their subjective perceptions of symptoms more than they do on objective findings to guide medication consumption (Apter et al., 1997; Priel, Heimer, Rabinowitz, & Hendler, 1994), and numerous studies have found major discrepancies between perception of respiratory symptoms and actual airway obstruction, caused either by asthma or by external resistive loads (Kendrick, Higgs, Whitfield, & Laszlo, 1993; Nguyen, Wilson, & German, 1996; Rietveld, Prins, & Kolk, 1996; Rushford, Tiller, & Pain, 1998). Rietveld, Kolk, Prins, and Colland (1997) showed that listening to false sounds of wheezing after exercise increases report of breathlessness among children with asthma, independent of actual pulmonary function.

Both children (Kifle, Seng, & Davenport, 1997) and adults with asthma (Kikuchi et al., 1994) who have experienced near-fatal asthma attacks display particularly impaired perception of dyspnea when breathing through external inspiratory resistive loads. Davenport, Cruz, Stecenko, and Kifle (2000) found that approximately half of children with life-threatening asthma fail to emit cortical evoked potentials in response to occlusion of inspiration, in contrast to virtually all other children, with or without asthma, thus suggesting impaired neural processing of inspiratory load information in the former group. Fritz, McQuaid, Spirito, and Klein (1996) showed that better symptom perception predicts less asthma morbidity (e.g., school absences, emergency room visits) among child asthma patients. Kikuchi et al. (1994) hypothesized that the relationship is explained by decreased chemoresponsiveness to hypoxia.

### Assessment of Symptom Perception

Numerous methodological issues are relevant to the study of symptom perception in asthma. External resistive loads have limited external validity as analogues of asthma because of the unique mechanical and sensory changes associated with asthma that cannot be replicated with mechanical loads (Harver & Mahler, 1998). Studies of natural changes in pulmonary function (e.g., estimation of peak flow) may be hampered by lack of variability in fluctuation of airway obstruction. The range of methodologies used in the studies reviewed here illustrates the challenges of defining and measuring perceptual accuracy (see Fritz, Yeung, et al., 1996, for a review of childhood asthma issues). To date, no consensus exists as to which technique (e.g., methacholine–histamine challenge

tests, resistive loads, estimating peak expiratory flow rate [PEFR]) should be used to identify individuals prone to poor symptom perception (NHLBI, 1997).

### Symptom Perception and Defensiveness

The psychological trait of defensiveness may predict underperception of asthma symptoms among adults (Isenberg, Lehrer, & Hochron, 1997; Steiner, Higgs, Fritz, Laszlo, & Harvey, 1987). This impaired perceptual accuracy may be related to higher endogenous opioid levels, that have been found in men with high levels of defensiveness but not women (Jamner & Leigh, 1999). Isenberg et al. (1997) showed that perceptual accuracy increased among defensive adults with asthma after administration of naloxone, an opioid receptor antagonist. On the other hand, *better* symptom perception was found among 10 children with asthma with a defensive coping style (Fritz, McQuaid, et al., 1996). Although the construct validity of defensiveness has not been adequately demonstrated among children (Nassau, Fritz, & McQuaid, 2000), it is nevertheless possible that defensiveness may exert different effects in adults than in children but that, among adults, poor perception of asthma symptoms may act additively to the psychophysiological correlates of defensiveness described above, suggesting that this personality trait may be an important risk factor for asthma, deserving of further research.

### Negative Affect and Symptom Perception

Negative affect bears a complex relationship with symptom perception in asthma, and the type and level of emotional arousal may be important determinants. Spinhoven, van Peski-Oosterbaan, van der Does, Willems, and Sterk (1997) found that patients with asthma reporting greater anxiety during histamine challenge tests, as assessed by the Subjective Units of Distress score (Kaplan, Smith, & Coons, 1995), showed better perception of airway obstruction. These patients did not display elevated levels of trait anxiety. Thus, anxiety that is specifically related to asthma may sensitize the individual to asthma-related body sensations through attentional processes (Arntz, Dreessen, & DeJong, 1994). Although this may promote appropriate symptom recognition and health care behaviors, as described below, negative affect also may lead to overperception of asthma symptoms (Janson, Björnsson, Hetta, & Boman, 1994; Rietveld & Prins, 1998).

Transient emotional states do not necessarily have an impact on perception of asthma. A study by Apter et al. (1997) found no relationship between perceptual accuracy and mood variables (e.g., combinations of active–passive task orientation and pleasant–unpleasant mood states). Boulet, Cournoyer, Deschesnes, Leblanc, and Nouwen (1994) showed that state anxiety during methacholine challenge tests was not related to symptom perception, although anxiety levels were unusually low in this sample.

Clinical levels of anxiety or depression tend to have a negative impact on asthma symptom perception. Both under- and overperceivers of airflow obstruction appear to have higher overall rates of psychological disorders than normal perceivers (Rushford et al., 1998). Tiller (1990) showed that a small sample of asthma patients with panic disorder were less sensitive to changes in external inspiratory resistive loads than those without. These results are consistent with findings among people with anxiety disorders but

not asthma (Tiller, Pain, & Biddle, 1987). Not all data have been consistent, however. Generalized anxiety (Fritz, McQuaid, et al., 1996) was not found to be related to symptom perception in a sample of children with asthma, although anxiety level in this sample tended to be mild.

### *Symptom Perception and Suggestion*

Luparello, Lyons, Bleeker, and McFadden (1968) developed the method most generally used to evaluate the effects of suggestion on airway function. The individual inhales a relatively inert substance such as saline that she or he is led to believe is a potent bronchoconstrictor or bronchodilator. We have previously reviewed studies showing that this procedure produces a clinically significant effect on pulmonary function among approximately 40% of people with asthma (Isenberg et al., 1992a), although there is evidence that suggestion may have a greater effect on *perception* of bronchial changes than on actual changes (Isenberg, Lehrer, & Hochron, 1992b).

### *Future Research on Symptom Perception*

The mechanism (e.g., biological or cognitive) that mediates altered perception of asthma symptoms in depressive and anxious states remains to be explored, as does the role of symptom perception in patients' medication use, in cases of both under- and overmedication. Additionally, simple and accurate tests for predicting poor perceivers in a clinical setting need to be developed.

## **Treatment Adherence**

NHLBI's (1997) guidelines emphasize the importance of adherence to all aspects of treatment, including medication and environmental control (i.e., avoidance of allergens). However, the complexity of some asthma treatment regimens (the use of several medications at different times for different purposes) often renders them difficult to follow, and adequately teaching asthma self-care requires considerable time, patience, and communication skills on the part of the physician. Indeed, 30–46% of patients with asthma even fail to fill their prescriptions for asthma medication (Kelloway, Wyatt, & Adlis, 1994; Watts, McLennan, Bassham, & El-Saadi, 1997). Nonadherence at a level associated with clinically significant negative effects on disease management, such as taking less than 70% of prescribed doses, occurs among 30–70% of patients with asthma (Rand & Wise, 1994). Adherence to asthma treatment recommendations is lower for controller than rescue medications (Hand & Bradley, 1996).

Poor medication adherence has been directly linked to indices of poor outcome, such as increased use of emergency health care services (Horn, Clark, & Cochrane, 1990; Milgrom et al., 1996; Schmaling, Afari, & Blume, 1998; Smith, Seale, Ley, Mellis, & Shaw, 1994). Adherence with preventive medication does not appear to improve during asthma exacerbations (Mann, Eliasson, Patel, & ZuWallack, 1992). Patients are often reluctant to follow asthma action plans that require them to double their dose of inhaled steroids when indicated, even after they have completed an asthma education program where the importance of this strategy is stressed (van der Palen, Klein, & Rovers, 1997). Even people

experiencing near-fatal attacks demonstrate poor treatment adherence (Boulet, Deschesnes, Turcotte, & Gignac, 1991).

There are many factors that may contribute to poor adherence, such as the financial costs of medication and difficulties with learning how to properly use medications. Poorer adherence with controller versus rescue medications may be explained by lack of immediate results with the former (Kelloway, Wyatt, DeMarco, & Adlis, 2000). Also, the perception of steroids as being threatening, because of potential side effects, is associated with poor adherence to an asthma treatment plan (Wöller, Kruse, Winter, Mans, & Alberti, 1993). Emotional support from the patient's closest person appears to reduce negative perceptions of steroid medication.

Assessment of adherence presents methodological problems. The majority of asthma medications are inhaled; only oral medications (e.g., theophylline) may be readily measured in sera and compared with established therapeutic ranges to infer the patient's level of adherence. There is evidence that both adults and children with asthma tend to overreport adherence (Bender, Milgrom, Rand, & Ackerson, 1998; Berg, Dunbar-Jacob, & Rohay, 1998; Bosley, Fosbury, & Cochrane, 1995; Braunstein, Trinquet, Harper, & Compliance Working Group, 1996; Gibson, Ferguson, Aitchison, & Paton, 1995; Milgrom et al., 1996). These results suggest that physicians may not receive an accurate estimate of patients' medication consumption from self-report. Adherence with inhaled medications may be assessed through patient self-report diaries, by weighing inhaler canisters and comparing the expected weight given the number of prescribed inhalations to the actual weight, and by attaching microprocessors to inhalers that count the number of inhaler actuations, to compare to expected actuations. There are limitations to all of these methods that generally result in the overestimation of actual patient medication use (see Rand & Wise, 1994). Newer devices that assess force of inhalation as a criterion for counting an actuation may provide more accurate assessment of medication consumption (Apter, Tor, & Feldman, 2001).

### *Family Dysfunction and Treatment Adherence*

Poor self-management behavior is common among dysfunctional families. Lower adherence with controller medications (Bender et al., 1998; Weinstein & Faust, 1997) has been associated with family dysfunction, specifically, absence of expressed affection. Parental criticism, a component of the expressed emotion construct, has also been associated with poor medication adherence for theophylline as well as oral steroids (F. S. Wamboldt, Wamboldt, Gavin, Roesler, & Brugman, 1995). This study also showed that adolescents from families with high levels of parental criticism showed greater improvements in asthma during an inpatient hospital stay, a finding that may be simply explained by the child's separation from the family. In one study (Weil et al., 1999) the existence of psychopathology among caregivers of children with asthma almost doubled the likelihood of hospitalization for asthma (Weil et al., 1999). The strong results persisted after controlling for morbidity at baseline. However, psychopathology among the *children* had an even stronger relationship with various indexes of asthma morbidity.

Most of the research on family relationships and asthma outcomes has focused on children with asthma. More research on the relationships of adults with asthma is needed because the scant extant research is suggestive of the importance of relationship

variables. For example, among 35–60-year-old adults with mild-to-moderate chronic obstructive pulmonary disease necessitating the use of inhaled bronchodilators, the best predictor of medication adherence was the presence of a significant other (Rand, Nides, Cowles, Wise, & Connett, 1995). Similarly, among couples with asthma discussing stressful topics, more anxiety seems to be associated with bronchoconstriction, but active problem solving appears to mitigate pulmonary variability (Schmaling, Afari, Hops, Barnhart, & Buchwald, 2002). Although adherence was not assessed in this latter study, medication adherence involves problem solving. A reasonable hypothesis for future research is that better medication adherence is associated with better problem-solving abilities.

Gavin, Wamboldt, Sorokin, Levy, and Wamboldt (1999) hypothesized that good treatment alliance between physician and patient may be a potential mediator between family functioning and clinical outcome, but they did not find a consistent relationship among these measures. Nevertheless, treatment alliance has been associated with better medication adherence and less frequent urgent physician office visits (Gavin et al., 1999). This finding is consistent with the research of Apter et al. (1998) showing that patients who reported barriers to communication with their physician displayed poor adherence with inhaled steroid regimens. Finally, Meijer, Griffioen, van Nierop, and Oppenheimer (1995) found that the impact of familial cohesion and rigidity on children with asthma may be adaptive, in that children from families with these characteristics tended to show better asthma control (as measured by frequency of hospitalization office visits and school absence). Baron, Veilleux, and Lamarre (1992) found that these family characteristics also are associated with higher panic-fear and higher doses of steroid medication, both of which could either be beneficial for control of asthma or indicate overreaction to asthma symptoms. Both studies were controlled for asthma severity, but neither study used a prospective design. It is possible that these family characteristics may be adaptive for asthma, even though rigidity is usually considered to be a maladaptive trait. Perhaps it may be beneficial in the presence of a disease that requires a complex medical regimen. In either case, family functioning may be understood as a contributor to asthma morbidity or, conversely, as a reaction to it (i.e., a coping mechanism) (Meijer et al., 1995).

### *Other Psychological Predictors of Adherence*

Case studies have provided examples of asthma patients with panic disorder who overmedicate with asthma drugs because of confusion between asthma and panic symptoms (Bernstein, Sheridan, & Patterson, 1991; Shavitt, Gentil, & Croce, 1993) given their similarity (Schmaling & Bell, 1997). Report of depression, on the other hand, has been associated with undermedication (Bosley et al., 1995), but more work is needed with clinical populations. Cluley and Cochrane (2001) showed that asthma patients classified as nonadherent (i.e., taking less than 70% of prescribed doses) scored higher on the Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983) than those classified as adherent. However, this relationship may have been moderated by age and gender, as older men were found to be more adherent (Cluley & Cochrane, 2001). The researchers did not try to control statistically for these demographic factors.

Particularly low levels of panic and anxiety about asthma symptoms also have been linked to increased asthma morbidity, because anxiety may be necessary to motivate the individual to seek appropriate treatment when symptoms appear (Kinsman, Dirks, Dahlem, & Heller, 1980), although even illness-specific anxiety, when very high, can convince physicians to prescribe higher levels of corticosteroids than pulmonary function would warrant (Hyland, Kenyon, Taylor, & Morice, 1993).

Theories of health behavior posit associations between specific psychological factors and adherence. Although a comprehensive review of relevant theories is beyond the scope of this article, we briefly describe the models most frequently applied to asthma. The health belief model (Becker et al., 1978) emphasizes the role of certain beliefs and cognitions, such as belief about disease severity and vulnerability to disability, in determining health behavior. For example, patients with asthma who believed it is a serious illness are more likely to use controller medications than patients who did not believe asthma is serious (Chambers, Markson, Diamond, Lasch, & Berger, 1999). Negative cognitions, such as concerns about medication side effects, have been linked to less medication use (Hand & Bradley, 1996). The transtheoretical stages-of-change theory (Prochaska, DiClementi, & Norcross, 1992) emphasizes cognitions regarding self-efficacy and perceived importance, thereby incorporating elements of the health belief model and other models, such as the theory of planned behavior (Ajzen, 1985), self-regulation theory (Bandura, 1986), and self-determination theory (Deci & Ryan, 1985). The transtheoretical model posits stages of behavior change that are differentiated by the ratio of perceived costs to benefits of engaging in the behavior at each stage, among other factors. Greater perceived necessity of medication use relative to negative concerns about medications has been associated with more adherence among patients with asthma (Horne & Weinman, 1999). Greater perceived advantages versus disadvantages regarding asthma medications have been linked to a greater intention to use medications as prescribed, which has been associated with more adherent medication use (Schmaling, Afari, & Blume, 2000). Schmaling et al. (2000) found an inverted-U relationship between pulmonary function (percentage of expected peak flow) and readiness for change in medication adherence; that is, better breathing was associated with precontemplation, action, and maintenance stages, and poorer airflow was associated with contemplation and preparation. Perhaps patients in the precontemplation stage had lacked motivation to take their asthma medication because they did not experience asthma symptoms. Patients in the contemplation and preparation stages may have been more willing to change their nonadherent behavior owing to greater respiratory difficulties (i.e., their pulmonary function was worse). The high adherence among individuals in the action and maintenance stages may be attributed to negative reinforcement (i.e., taking asthma medication is reinforced by reduction of unpleasant respiratory symptoms).

Other researchers have developed empirically derived typologies of psychological factors implicated in adherence. Adams, Pill, and Jones (1997) performed in-depth interviews with adult asthma patients and categorized them either as deniers, who rejected having asthma and relied on reliever medication ( $\beta$ -2-agonists) to treat what was perceived as an acute condition, or as acceptors, who admitted to having asthma and reported proper adherence with preventive medication. Future research is necessary to devise

assessment tools to measure and validate these constructs, particularly in the area of predicting treatment adherence.

Although self-efficacy has been shown to be a predictor of treatment adherence in other chronic diseases (Bock et al., 1997; Kavanagh, Gooley, & Wilson, 1993), this topic has not received as much attention in the field of asthma. Scherer and Bruce (2001) recently showed a modest relationship between self-efficacy and self-report of treatment adherence. However, a poor response rate to the survey and assessment of adherence through self-report substantially limits interpretation of these findings. Using an electronic counting device to measure adherence, Apter et al. (1998) found no relationship between health locus of control and adherence with an inhaled steroid regimen.

### Psychological Interventions for Asthma

In this section we review interventions for asthma management that target patients' knowledge, beliefs, and behavior. The enhancement of knowledge through educational interventions is crucial for disease management and has been well integrated into many disease management programs. By contrast, there is a striking dearth of interventions that address beliefs, behavior, and perceptions or that routinely screen for and treat conditions associated with poor asthma management or outcomes such as comorbid psychiatric conditions, family dysfunction, or poor communication between patient and provider. For example, asthma management programs have used physician education (e.g., Hendricson et al., 1994), peer education (e.g., Persky et al., 1999), and innovative educational methods, such as multimedia and computer-based programs (e.g., Bartholomew et al., 2000; Homer et al., 2000) to convey information, but an individualized focus on dysfunctional cognitive-behavioral variables is lacking.

### Asthma Education

The NHLBI's (1997) guidelines emphasize the importance of education in the treatment of asthma. They recommend that a written action plan instruct patients to take medication and to contact health care providers according to various zones of asthma severity that correspond to the colors of a traffic light. The zones are based on a combination of symptoms and peak flow values. The guidelines also recommend including the following components in asthma education: instructing the patient about basic facts of asthma and the various asthma medications; teaching methods for self-monitoring of asthma symptoms, including competent use of a peak flow meter; teaching techniques for using inhalers and avoiding allergens; devising a daily self-management plan; and completing an asthma diary for self-monitoring.

Asthma education has been shown to be cost effective for both children (Greineder, Loane, & Parks, 1999) and adults (Taitel, Kotses, Bernstein, Bernstein, & Creer, 1995). Numerous empirically validated educational programs are available for asthma patients of all ages, some of which have demonstrated improvements on measures such as frequency of asthma attacks and symptoms, medication adherence, and self-management skills (Kotses et al., 1995; Wilson et al., 1996).

Asthma education also has been shown to increase self-efficacy and internality on health locus of control (Bruzzeze, Markman, Appel, & Webber, 2001; Tieffenberg, Wood, Alonso, Tossutti, &

Vicente, 2000; Wigal et al., 1993). However, although these programs are effective when examining various parameters of morbidity, it has not yet been shown that changes in self-efficacy or locus of control mediate these improvements in asthma self-management. Understanding the mechanism of behavior change may allow for further integration of cognitive-behavioral techniques, motivational enhancement, and basic asthma education principles.

Evaluation of asthma education has been hampered by enormous variability between studies and inadequate reporting of the type of education provided (Sudre, Jacquemet, Uldry, & Perneger, 1999). A meta-analysis of asthma education programs for children revealed small effect sizes for various parameters of asthma morbidity (Bernard-Bonnin, Stachenko, Bonin, Charette, & Rousseau, 1995). However, only a small number of studies met criteria (e.g., that the study be a randomized clinical trial) for this meta-analysis. Devine (1996) conducted a meta-analysis on adult asthma patients using less stringent criteria, including studies using nonrandom assignment, and found that educational programs offered multiple benefits. For example, large effects were reported for treatment adherence and accuracy of inhaler technique, and medium effects were found for frequency of asthma attacks, quality of life, and psychological symptoms. Clearly, however, future research must correct methodological shortcomings of previous studies and provide better documentation to allow for replication.

Furthermore, studies of asthma education have typically compared experimental with control groups and examined *statistically* significant differences. Research is lacking on demonstration of *clinically* significant effects using formal criteria, such as in NHLBI's (1997) guidelines, as described above. Asthma education outcome studies developed after publication of the new guidelines have not yet been published.

### Peak Flow Monitoring as a Component in Asthma Education

An important component in asthma education is training patients to record their home peak flow values as their major guide for carrying out their asthma action plan. However, studies using electronic chips have consistently revealed very low adherence rates with long-term home peak flow monitoring (Côté, Cartier, Malo, Rouleau, & Boulet, 1998; Redline, Wright, Kattan, Kerckmar, & Weiss, 1996; Verschelden, Cartier, L'Archevêque, Trudeau, & Malo, 1996). A study of asthma patients who had received asthma education revealed that most would not even monitor their peak flow during severe asthma exacerbations (Kolbe, Vámos, James, Elkind, & Garrett, 1996). Also, it has been reported that peak flow meters have lower sensitivity than FEV<sub>1</sub> and measures of midexpiratory flow for detecting changes in pulmonary function (Eid, Yandell, Howell, Eddy, & Sheikh, 2000; Giannini et al., 1997; Miles, Tunnicliffe, Cayton, Ayres, & Miller, 1996; Sly, Cahill, Willet, & Burton, 1994). Overreliance on peak flow measures could thus provide false reassurance during the early stage of an attack and may lead to delays in carrying out action plans.

Most studies comparing peak flow monitoring with symptom monitoring among asthma sufferers have failed to show differences in clinical outcome (Charlton, Charlton, Broomfield, & Mullee, 1990; Grampian Asthma Study of Integrated Care, 1994;



K. P. Jones et al., 1995; Turner, Taylor, Bennett, & Fitzgerald, 1998). These findings provide support for symptom monitoring because it is easier to carry out than peak flow monitoring. Ignacio-Garcia and Gonzalez-Santos (1995) did find that peak flow monitoring, in combination with other self-management and educational components, led to reductions in asthma morbidity, although the specific beneficial effects of peak flow monitoring could not be isolated. Reeder, Dolce, Duke, Raczynski, and Bailey (1990) found that peak flow monitoring did not lead to significant improvements in symptom perception, although daily, long-term peak flow monitoring is still recommended for moderate-to-severe persistent asthma patients and patients with poor symptom perception (NHLBI, 1997). Consistent with this recommendation, Cowie, Revitt, Underwood, and Field (1997) recruited asthma patients who had experienced a severe exacerbation within the past year and found that home peak-flow monitoring with an action plan reduced the frequency of subsequent emergency room visits.

### *Individualized Self-Management*

In addition to adapting educational programs for high-risk populations, self-management plans can be tailored to the specific needs of individual patients. Typically, personalized plans are developed either from interviews with the patient or from self-monitoring during a baseline period (Kotses, 1998). Individualized programs have been shown to reduce the number of asthma attacks (Kotses, Stout, McConaughy, Winder, & Creer, 1996) and asthma symptoms (Evans et al., 1999). Wilson et al. (1993) found that personalized self-management was associated with better environmental control and inhaler technique. However, comparable benefits were found in a small-group educational program, a more cost-effective and supportive forum. Individualized training might most easily be accomplished in the physician's office during periodic reviews of asthma status, as recommended by NHLBI (1997).

### *Smoking Cessation Interventions as Components of Asthma Education*

There has been little attention to smoking cessation as a component in asthma education programs, either for adult patients or parents of children with asthma. Several studies have found that a brief educational session was ineffective in changing smoking behavior of parents whose children have asthma (Irvine et al., 1999; McIntosh, Clark, & Howatt, 1994; Silagy, 1999). A similar intervention for adult smokers with asthma also failed to affect smoking behavior (Wilson et al., 1993). Smoking also has been identified as a predictor of low attendance at asthma education programs among parents of young children with asthma (Fish, Wilson, Latini, & Starr, 1996). These studies demonstrate the need to devise and implement formal smoking cessation programs among people with asthma and their families.

### *Limitations of Asthma Education*

A major obstacle to successful implementation of asthma education is poor patient attendance (Abdulwadud et al., 1997; Yoon, McKenzie, Miles, & Bauman, 1991). Another problem is that patients who score high on measures of self-management knowl-

edge may not necessarily display appropriate behavior during actual attacks (Kolbe, Vámos, Fergusson, Elkind, & Garrett, 1996). Bridging the gap between knowledge and behavior remains an important area of development for asthma education.

## *Other Psychological Interventions*

### *Symptom Perception Training*

Two studies have produced promising findings that suggest it may be possible to train asthma patients to improve their perception of airway obstruction. Harver (1994) used absolute threshold training with external resistive loads and found that patients who received feedback after each trial about the accuracy of their estimate performed better on the perception task than those who did not. This finding was upheld at follow-up when testing was repeated without feedback. Stout, Kotses, and Creer (1997) used difference threshold training and found that feedback combined with presentation of loads in order of increasing difficulty led to improvements in perceptual accuracy. Future research needs to investigate whether improvements with external inspiratory resistive loads are related to increased perceptual accuracy of pulmonary function and better clinical outcome, and whether including symptom perception training provides incremental benefits as a component in an asthma management program.

### *Cognitive Interventions, Psychotherapy, and Family Therapy*

The few studies that have examined the use of psychotherapy as an adjunctive treatment for asthma have been limited by the use of small sample sizes. Sommaruga et al. (1995) combined an asthma education program with three sessions of cognitive-behavioral therapy (CBT) focusing on areas that may interfere with proper medical management. Few significant between-group differences on measures of anxiety, depression, or asthma morbidity emerged between a control group receiving medical treatment alone and the CBT group. In an uncontrolled study, Park, Sawyer, and Glaun (1996) applied principles of CBT for panic disorder to children with asthma reporting greater subjective complaints and consuming excessive medication. In the 12 months following treatment, rate of hospitalization for asthma decreased, but other measures of clinical outcome were not analyzed. We have recently combined components of asthma education and CBT for panic disorder to develop a treatment protocol appropriate for adults with both asthma and panic disorder (Feldman, Giardino, & Lehrer, 2000). This treatment is currently being empirically evaluated.

There also is evidence from two controlled studies that family therapy can lead to improved asthma symptom control in some cases of severe childhood asthma (Gustafsson, Kjellman, & Cederbald, 1986; Lask & Matthew, 1979). However, the samples and effect sizes in these studies were small, and the results were inconsistent. It is possible that family therapy may be most helpful for families in which interpersonal difficulties interfere with carrying out the complex medical regimen required by children with severe asthma.

To the best of our knowledge, there have been no controlled treatment outcome studies applying psychotherapy to patients with comorbid asthma and psychiatric disease. It is important to deter-

mine whether decreases in psychopathology lead to concomitant improvements in quality of life, symptom perception, treatment adherence, and clinical outcome. Nevertheless, the NHLBI guidelines for asthma treatment recommend referral to mental health professionals when stress appears to interfere with medical management of asthma (NHLBI, 1997). In a study by Godding, Kruth, and Jamart (1997), pediatric patients with poorly controlled asthma demonstrated improved treatment adherence and clinical outcome when treatment was coordinated between a pediatrician and psychiatrist. We have found no similar studies on adult asthma patients. With recent improvements in screening devices for psychological disorders in primary care (Spitzer, Kroenke, & Williams, 1999), the time is ripe for behavioral scientists to explore the efficacy of psychotherapy among patients with asthma.

Several studies have evaluated cognitive interventions specifically derived from the health beliefs and transtheoretical models. P. K. Jones, Jones, and Katz (1987) found that patients with asthma who had received an intervention based on the health belief model were more likely to make and keep follow-up appointments than patients who had received treatment as usual. Schmalings, Blume, and Afari (2001) found that modifying patients' specific beliefs about medications using education plus motivational interviewing (W. R. Miller & Rollnick, 1991), an intervention based on the transtheoretical model, enhanced patients' reasons for using medications. The decisional balance (pros vs. cons) changed in an advantageous direction (with more "pros" and fewer "cons" for using medication as prescribed) among participants who had received the motivational interviewing than among people who received only education.

#### *Written Emotional Expression Exercises*

In recent years, several reports have been published promoting the health benefits of emotional disclosure of psychologically traumatic experiences through writing (e.g., Esterling, Antoni, Fletcher, Margulies, & Schneiderman, 1994; Pennebaker, Kiecolt-Glaser, & Glaser, 1988). Smyth et al. (Smyth, 1998; Smyth, Stone, et al., 1999) asked study participants to write an essay expressing their thoughts and feelings about a traumatic experience and found a clinically significant improvement in FEV<sub>1</sub> among asthma patients after a 4-month follow-up, with no improvement noted in a control group who wrote on innocuous topics. In a later analysis, they reported that these effects were not mediated by perceived stress, quality of sleep, affect, substance use, or medication use (Stone, Smyth, Kaell, & Hurewitz, 2000).

#### *Other Psychosocial Interventions*

One study evaluated the effects of a psychological intervention on the immune-inflammatory system in asthma. Castés et al. (1999) provided children with asthma a 6-month program that included relaxation/guided imagery, cognitive stress-management therapy, and a self-esteem workshop. Improvement occurred both in clinical measures of asthma and in asthma-related immune-system measures. The treatment group, but not a control group that received only medical treatment, significantly decreased their use of  $\beta$ -2 stimulant medication, showed improvements in FEV<sub>1</sub>, and, at the end of treatment, no longer showed a response to bronchodilators (consistent with improvement in asthma). Basal FEV<sub>1</sub>

improved to normal levels in the treatment group after 6 months of treatment. Children in the treatment group showed a significant reduction in specific IgE responses against the most common allergen in the study population. Stimulation of IgE responses by environmental allergens is an important contributor to asthma exacerbations. Treatment group children also exhibited increased natural killer cell activity and a significantly augmented expression of the T-cell receptor for IL-2. Natural killer cells produce agents that inhibit IgE synthesis, and IL-2, an immune system messenger molecule, acts to suppress lymphocyte activity associated with atopy.<sup>3</sup>

#### *Direct Effects of Psychological Treatments on the Pathophysiology of Asthma*

In addition to psychological interventions targeted at health care behaviors or stress management, research is continuing on the methods by which people can learn to exercise direct control over physiological processes involved in asthma. These methods have included various relaxation methods, biofeedback, hypnosis, and yoga.

#### *Relaxation Training*

In an earlier review, Lehrer, Sargunraj, and Hochron (1992) concluded that relaxation training has often statistically significant but small and inconsistent effects on asthma, particularly after several weeks of training, although the immediate effect may be a worsening of pulmonary function due to parasympathetic rebound. More recent studies have yielded a similar pattern (Table 1), although in some studies clinically significant improvements in pulmonary function did occur. Outcome measures, populations, and relaxation procedures differ across studies and may explain some of the inconsistencies. Although possibly helpful for preventing stress-induced asthma exacerbations, relaxation training does not produce reliable effects of clinically significant magnitude for treating or preventing asthma. Further research is warranted on their use among asthma patients showing emotion- or stress-induced asthma symptoms and possible mediation of these effects by inflammatory processes.

#### *Biofeedback Techniques*

*Surface eletromyographic (EMG) biofeedback.* In contrast to the general relaxation approaches to treating asthma described above, Kotses and his colleagues hypothesized that only relaxation

<sup>3</sup> Immunoglobulins (Igs) are proteins that act as antibodies in the immune system. Antibodies react to antigens, substances that induce immune sensitivity or responsiveness. Igs are classified (A, D, E, G, M) on the basis of the structural and antigenic properties of their protein chains. An important component of asthma is the stimulation of IgE responses by environmental allergens. This stimulation leads to mast cell activation, which results in the release of vasoactive and bronchoconstrictive agents, which attract inflammatory cells to the area. Interleukin-2 is a cytokine derived from T helper lymphocytes that causes proliferation of T lymphocytes and activated B lymphocytes. This lymphocyte activity, called a Th-1 profile, acts to suppress lymphocyte activity associated with atopy (i.e., the Th-2 profile). T helper cells are a subset of lymphocytes that secrete various cytokines that regulate the immune response.

Table 1  
*Studies of Relaxation Therapy for Asthma*

Study	Participants	Treatment protocols	Training sessions	Control groups	Medication controlled	Results	Clinical significance
Smyth, Soefer, et al. (1999)	20 adults	20-min taped relaxation: awareness (muscle tension and breathing), and breathing into tension spots	10 sessions (at least once per day) within 21 days	None	No change in pre- to posttest of medication consumption	PEFR <sup>a</sup> (2/day) increased,* occurrence of reported stressors decreased*** after intervention. Asthma symptoms decreased.*	Yes. Mean of PEFR increased >24%.
Lehrer et al. (1994); Lehrer, Hochron, et al. (1997)	87 adults	Progressive relaxation (PR)	8 sessions, 1-hr daily home practice	Wait list (WL) and music placebo	Prestabilization; no differences in medication consumption between groups	PEFR <sup>a</sup> (2/day)* and FEF50% <sup>b**</sup> increased between sessions in PR group; PEFR*, FEF50%***, FEV <sub>1</sub> ***, and FEV <sub>1</sub> /FVC% <sup>c***</sup> decreased at pre- to postsession across groups (all groups relaxed).	No. Increase in mean FEF50% < 30%, mean of FEV <sub>1</sub> and in FEV <sub>1</sub> /FVC < 15%. Changes in FEV <sub>1</sub> were nonsignificant.
Loew et al. (1996)	17 adults	Functional relaxation (FR) <sup>d</sup> and bronchodilator (IT) <sup>e</sup>	1 session for each intervention	None	Withdrawal for 12 hr before each session	Raw <sup>f</sup> decreased after both interventions.**	Yes. 80% of participants after FR, 40% after IT decreased over 20% in raw.
Henry et al. (1993)	24 adults	Autogenic therapy	8 months (weekly 1-hr sessions)	Supportive group psychotherapy	Patients in both groups receiving equivalent maximum medical treatment; details not reported	FEV <sub>1</sub> <sup>g**</sup> and percentage predicted FEF25–75% <sup>g***</sup> and MEF50% <sup>b*</sup> increased in the autogenic training group pre- to postintervention.	Yes. Mean increase in FEV <sub>1</sub> > 15%.
Vazquez et al. (1993a, 1993b, 1993c)	27 children	Asthma self-management program (SM), and SM + PR	6 weekly 1-hr sessions	1. WL 2. SM	Prestabilized <sup>h</sup> ; no group differences in medication consumption	Pre- to posttreatment, 6- and 12-month follow-up (only among patients with emotionally triggered asthma): 1. Attack duration decreased in SM + PR group** 2. Nonsignificant decrease in use of $\beta$ -2 adrenergic medication. 3. Percentage predicted PEFR <sup>a</sup> (2/day) increased in all groups (SM > WL, SM + PR = WL, SM)	No

<sup>a</sup> PEFR = peak expiratory flow rate during a forced expiratory maneuver from maximum vital capacity. <sup>b</sup> FEF50% and mid-expiratory flow (MEF) = the flow at 50% of vital capacity in a forced expiratory maneuver. <sup>c</sup> FEV<sub>1</sub>/FVC = volume in the first second of a forced vital capacity maneuver from full vital capacity. <sup>d</sup> Functional relaxation is a body-oriented psychotherapeutic relaxation method. <sup>e</sup> Terbutaline, a dose of two puffs (0.25 mg each) using a multidose powder inhalation system. <sup>f</sup> Raw = respiratory resistance measured with a whole body plethysmograph. <sup>g</sup> FEF25–75% = forced expiratory flow between 25% and 75% of a forced expiratory maneuver from full vital capacity. <sup>h</sup> Medication stabilization refers to “controller” medications (inhaled steroids, theophylline, leukotriene inhibitors, long-acting  $\beta$ -2 medication, cromolyn, etc.).

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

in very specific muscles should produce an improvement in pulmonary function (Kotses & Glaus, 1981; Kotses et al., 1991; Glaus & Kotses, 1983). According to their model, changes in facial muscle tension directly produce respiratory impedance through a trigeminal-vagal reflex pathway, such that tensing these muscles produces bronchoconstriction, and relaxing them produces bronchodilation. They tested the model using frontal EMG biofeedback training to increase and decrease tension in the facial muscles. They found that training patients to decrease facial muscle tension produced improvements in pulmonary function, whereas training to increase it had the opposite effect. EMG biofeedback training to the forearm muscles had no effects. However, several recent studies from other laboratories have tried to replicate these findings, but the results have all been negative (Lehrer et al., 1994, 1996; Lehrer, Generelli, & Hochron, 1997; Mass, Wais, Ramm, & Richter, 1992; Ritz, Dahme, & Wagner, 1998).

Another EMG biofeedback strategy, suggested by Peper and Tibbetts (1992), provided EMG biofeedback to the muscles of the neck and thorax, incentive spirometry biofeedback, desensitization to asthma sensations, and training in relaxed abdominal breathing. At the follow-up, all participants significantly reduced their EMG tension levels while simultaneously increasing their inhalation volumes. Participants reported reductions in their asthma symptoms, medication use, emergency room visits, and breathless episodes. A small pilot study from our laboratory, however, did not show significant effects for this method (Lehrer, Carr, et al., 1997), although nonsignificant trends were observed. More research on this method is warranted.

Kern-Buell, McGrady, Conran, and Nelson (2000) found in a small sample that frontalis EMG biofeedback training along with relaxation therapy produced improvements in asthma symptomatology and a small but significant improvement in one measure of pulmonary function ( $FEV_1/FVC$ ). Compared with a waiting list control group, patients in the biofeedback group also showed marginally greater decreases in bronchodilator drug use and asthma symptoms. Participants in the biofeedback condition also showed greater decreases in neutrophils and increases in basophils, suggesting a decreased inflammatory reaction.

In general, EMG biofeedback training produces effects similar to those of relaxation: small but consistent asthma improvement over time, only rarely of clinical significance.

**Respiratory resistance biofeedback.** Mass and his colleagues (1991) attempted to directly train participants to decrease respiratory resistance by providing continuous biofeedback of this measure, using the forced oscillation method. In an uncontrolled trial, this feedback technique decreased average respiratory resistance within sessions but not between sessions (Mass, Dahme, & Richter, 1993), and it did not increase  $FEV_1$  (Mass, Richter, & Dahme, 1996). They concluded that this type of biofeedback might not be an effective treatment component against bronchial asthma in adults.

**Biofeedback training to increase heart rate variability.** A new biofeedback approach involves training people to increase the amplitude of heart rate oscillations that accompany breathing (respiratory sinus arrhythmia, or RSA; Lehrer, Carr, et al., 1997; Lehrer, Vaschillo, & Vaschillo, 2000). Multiple case studies from clinics in Russia support the hypothesis that RSA biofeedback training can help people with various neurotic and stress-related physical disorders (Chernigovskaya, Vaschillo, Petrash, & Ru-

sanovskii, 1990; Chernigovskaya, Vaschillo, Rusanovskii, & Kashkarova, 1990; Lehrer, Smetankin, & Potapova, 2000; Pichugin, Strelakov, & Zakharevich, 1993; Vaschillo, Zingerman, Konstantinov, & Menitsky, 1983), including asthma (Smetankine & Vartanova, 1995). The mechanism for the effectiveness of this procedure is unknown. It appears to be effective even though RSA amplitude tends to be either at normal levels or even somewhat elevated among asthma patients at rest (Kallenbach et al., 1985) and despite the fact that RSA is mediated by vagus nerve activity (Porges, 1992), which, when increased, can cause bronchoconstriction (Nadel & Barnes, 1984). However, RSA also reflects the activity of reflexes that modulate autonomic reactions (including parasympathetic reactions) and preserve homeostasis (Porges, 1992, 1995). Perhaps this is the function that RSA biofeedback enhances. Another possible mediator is slow breathing. In this method, patients' respiration rate is reduced to approximately 6 breaths/min (Lehrer, Carr, et al., 1997), and taking at least one slow deep breath is known to have a bronchoprotective effect when inhaling bronchoconstrictor drugs (Brown et al., 2000). This response is almost universal in healthy people but absent in people with asthma (Kapsali, Permutt, Laube, Scichilone, & Togias, 2000). Other contributions to the effects of the method may accrue from relaxed, abdominal, pursed-lips breathing, which is a component of the RSA biofeedback technique. Preliminary results suggest that the effect of this method may be of clinically significant magnitude, both immediately and over the long term, but measures consistent with NHLBI's (1997) guidelines have not been used in these studies. Additional evaluation of this method is warranted.

## Yoga

Yogis practice a breathing technique known as pranayama, which includes (a) slowing and regularizing the breath by prolonging the expiratory phase, (b) enhancing abdominal-diaphragmatic breathing, and (c) imposing resistance to both inspiration and exhalation (Chandra, 1994). These components of yoga produce physical and mental relaxation and are thought to have a stabilizing effect on bronchial reactivity, through reduction of vagal efferent activity (Nagarathna & Nagendra, 1985).

Three uncontrolled studies of yoga among asthma patients found improvement in asthma symptoms, as well as a more positive attitude, feelings of well-being, and fewer symptoms of panic. Jain et al. (1991) found a statistically significant improvement in pulmonary function (percentage predicted  $FEV_1$ ) after 40 days of twice-daily practice among 46 adolescents with asthma. The initial values were low (67.9% expected  $FEV_1$ ), and mean improvement was clinically significant among women (23%) but not across sexes (11.6%). The improvement in midexpiratory flow was statistically significant across sexes (28%), but this measure is not generally used to assess clinical significance in asthma. Exercise tolerance also improved. Nagendra and Nagarathna (1986) treated 570 asthma patients with yoga and followed them for a period ranging between 3 and 54 months. They found clinically significant improvements in peak expiratory flow and large decreases in asthma medication consumption. Sathyaprabha, Murthy, and Murthy (2001) treated 37 adult patients with asthma using yoga therapy and found significant improvements in a variety of pulmonary



function measures, including FEV<sub>1</sub>, peak flow, and FEV<sub>1</sub>/FVC, of clinical significance in some patients.

We have found two controlled studies of yoga therapy for asthma. Nagarathna and Nagendra (1985) randomly assigned 106 asthma patients to either yoga therapy, emphasizing slow breathing practices, or a control group, where no yoga was given. All patients continued to receive usual medical care outside of the study. Patients in the yoga condition showed a clinically significant increase in mean peak flow of 25%, as well as a decrease in symptomatology and use of drugs. These measures all were significantly greater than in the control condition. However, Vedanthan et al. (1998) found no between-group differences in pulmonary function between patients exposed to yoga therapy or to a no-treatment control condition. Vijayalakshmi, Satyanarayana, and Rao (1988) found greater asthma improvement among a group of 34 patients for whom a combination of yoga and psychotherapy was added to standard medical treatment relative to a group of 14 asthma patients given only the medical care. However, participants were not randomly assigned to treatments in this study.

These studies present preliminary evidence that yoga may be helpful for asthma and suggest that larger controlled trials may be warranted. Inconsistencies among studies may reflect differences in the yoga techniques used and the expertise with which they were taught. A component analysis of yoga treatment might be useful at this stage, particularly of components involving slow breathing; muscular relaxation, stretching, and exercise; and meditation. Each of these components overlaps with specific other self-regulation methods mentioned in this section.

### *Hypnosis*

In a controlled study of hypnosis as a treatment of asthma among children, Kohen (1995) noted improvement in asthma symptoms but not in pulmonary function, compared with no-treatment and waking-suggestion groups. A greater decrease in emergency room visits and missed days in school also was found in the hypnosis group. These data suggest that hypnotic interventions may improve asthma quality of life but not pulmonary function. Further evaluation of these effects is warranted. Similar findings were obtained in a later uncontrolled study among preschool children (Kohen & Wynne, 1997) for parental reports of asthma symptoms but not pulmonary function. Hackman, Stern, and Gershwin (2000) published a thorough review of the literature on asthma and hypnosis and concluded that the technique may be helpful, particularly among individuals who are highly hypnotizable, but requires further evaluation to prove efficacy.

### *Discussion*

It is well established that behavioral factors play an important role in exacerbation and treatment of asthma. Behavioral factors such as exposure to asthma triggers, accurate perception and evaluation of asthma symptoms, seeking proper medical care, and adherence to medical regimen strongly predict the frequency and severity of asthma exacerbations. Mediators of these behaviors, including psychopathology and family disorganization, can exacerbate asthma, decrease asthma quality of life, and increase asthma-related medical care costs. Under some circumstances these factors may increase threat to life from asthma. Also, the care

required for severe asthma is complex and difficult. It involves avoidance of multiple common environmental stimuli, including cockroaches and various pollens, household pets, foods, and air pollutants. It also involves taking several medications, some expensive, each on different schedules and for different purposes (e.g., for the control of chronic inflammation vs. acute bronchodilation), with different side-effect profiles. The ability to follow such a regimen requires a high degree of behavioral organization. Any factors that impair behavioral functioning would thus naturally be expected to affect asthma health. Health beliefs may interact with such exacerbating factors in affecting asthma self-care, and measures of readiness to change may be useful for predicting patients' willingness to engage in the rigors of proper asthma self-care, although the clinical relevance of these factors remains to be studied using the criteria of NHLBI's (1997) guidelines. On the other hand, it also is clear that asthma is not a behavioral disease. There is no solid evidence indicating that behavioral factors *cause* asthma, although there is suggestive evidence that asthma may play a role in the development of some psychiatric disorders.

However the pervasiveness of psychological influences on asthma and mechanisms for these effects still require clarification. Understanding the relationship between psychiatric disturbance and asthma requires more extensive epidemiological research, including direct assessment of pulmonary function as well as more representative sampling of the entire spectrum of asthma severity. In addition, the separate and additive effects of psychopathology on asthma require evaluation, including the effects of psychopathology on asthma self-care and the perception and interpretation of symptoms, as well as direct psychophysiological effects of psychopathology on the respiratory system.

There is evidence that certain negative emotions, particularly panic and depression, even when not severe enough to be classifiable as a psychiatric disorder, may produce respiratory effects that are consistent with exacerbation of asthma, both by their effects on asthma self-care and by their direct psychophysiological effects on the lung, and, conversely, that these emotional states can be triggered by asthma. However, the clinical significance of this association is still not clear. Further research is required using the criteria of NHLBI's (1997) guidelines for assessing the clinical significance of the effects on asthma. Other psychological factors showing a similar relationship to asthma exacerbation also require evaluation of their clinical relevance, particularly the influence of defensiveness and its relationship to perception of asthma sensations.

The mechanism of emotional asthma triggers also requires further research. Recent findings in respiratory psychophysiology have documented that airways are just as reactive to psychological state as other physiological systems (cf. the well-known electrodermal and cardiovascular effects of emotional states) and that psychologically induced airway responses include changes in airway caliber that are characteristic of fluctuations in asthma. Although there is suggestive evidence that the pathway for such effects may be via parasympathetic rebound, direct parasympathetic effects of specific psychological states (e.g., sadness or passive response to stress), or a specific inflammatory response, this evidence remains sketchy and requires replication. Psychological influence on a third common component in asthma exacerbation, mucous congestion, remains completely uninvestigated. Sim-

ilarly, the pathway by which psychological factors affect perception and interpretation of asthma symptoms requires exploratory research. Current findings hint that endorphin activity as well as interpretation of respiratory sensations may play a role in accuracy of asthma symptom perception. The clinical significance of these effects remains mostly uninvestigated.

Although there is considerable preliminary evidence that asthma education should play an important role in asthma care, the reliability and generalizability of the clinical effects of such interventions require more research. The time is ripe for a series of multicenter randomly controlled trials of asthma education programs. Within these programs, the importance of specific components requires investigation. It has not, for example, been firmly established that training people to take accurate home peak flow readings is necessarily better than learning accurate monitoring of asthma symptoms. Similarly the usefulness of relaxation and stress management components in asthma education is unclear. Also, the willingness of asthma patients to avail themselves of asthma education interventions requires investigation, as does the usefulness and adequacy of short interventions that can be performed during routine physician visits for asthma care.

We may find that more complex or comprehensive interventions might be more generally useful among particularly targeted populations (e.g., full-scale asthma education for school children at risk for major asthma flares or people with life-threatening asthma; the use of stress management, relaxation, or EMG biofeedback therapy specifically among individuals with clear evidence for stress-induced asthma exacerbations; a smoking-reduction program for asthma patients who smoke). Similarly, because of the apparently facilitative interactions between asthma and psychopathology, it may become worthwhile to devise and evaluate psychological therapies that are specifically targeted at particular comorbid conditions.

Additionally, although NHLBI's (1997) guidelines conclude that specific respiratory strategies such as pursed-lips breathing may be useful for managing the stressfulness of asthma but not its physiology, recent research on slow deep breathing methods and RSA biofeedback suggests that such a conclusion may be overly pessimistic. Further evaluation of these methods is warranted.

Finally, evaluation of the clinical significance of psychological effects requires that the criteria for asthma severity and exacerbation described in NHLBI's guidelines be accurately and reliably scorable. Because of the complexity of the decision processes needed to diagnose asthma severity, it is unclear whether the distinctions can be made reliably. The extent of measurement error in using these criteria is unknown. This is particularly the case for the symptom domain. How severe must symptoms of dyspnea or chest tightness be to be classifiable as having occurred? It is possible that patients who are more concerned about their symptoms or who are more attuned to or frightened by body sensations may report incidences of very mild discomfort, whereas others may report only more severe symptoms.

It may be useful to devise and use more accurate measures of asthma symptom intensity as well to distinguish symptom intensity from the discomfort and impairment of function that symptoms produce and to assess the relationship among these sometimes unrelated measures. Lehrer, Hochron, Isenberg, Rausch, and Carr (1993) have done preliminary research on devising a scale that makes this distinction for asthma symptoms.

The usefulness of palliative measures in treating asthma might be investigated for people who overperceive symptoms or who experience disproportionate discomfort from them. However, it is possible that under some circumstances, decreasing the discomfort of asthma alone may be found to *increase* the risk of severe illness, because discomfort may motivate the individual to obtain necessary treatment; such research must therefore be done with great care. The usefulness and safety of such procedures require evaluation.

Although seemingly more objective, the reliability of assessing the pulmonary function and medication domains of asthma severity also requires evaluation. Spirometry measures, particularly peak flow, can be affected by the patient's effort and technique, and patients' self-report of home pulmonary function assessments is known to be unreliable. The use of new computerized home-measurement devices with prompts and corrections for poor technique and built-in memory may eventually be useful for both research and clinical purposes, particularly when the price of these devices decreases and renders them more accessible. Similarly, the complexity of some medical regimens for asthma makes them difficult to scale, and the technique by which a patient uses a metered dose inhaler or discus may materially affect the dose of medication that is actually administered. Although the use of modern computer technology and other technological advances in asthma pharmacology may eventually improve these problems, the reliability of measuring both the pulmonary function and the medicine domains of asthma severity, as defined by NHLBI's guidelines, still is unknown. This system for diagnosing asthma severity criteria itself thus requires further development as a research tool. Creating such a tool could be a potent boon to clinical research on asthma.

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