Evidence for the role of behavior in the etiology of asthma is reviewed here. Behavioral factors have not convincingly been shown to play a part in the inception of asthma. It has been suggested that asthma attacks can be precipitated by conditioning. Previous experiences leading to the expectation that an attack may occur can be regarded as a behavioral factor which may trigger an attack, possibly by inducing hyperventilation. Further research should focus on the possible role of behavioral factors in maintaining a state of hyperreactivity of the bronchi and psycho-immunologic aspects such as brain-controlled neuroendocrine immune mechanisms.

In considering asthma, the term “etiology” has been often misunderstood. A multiplicity of factors are capable of provoking attacks of asthma, but it is by no means clear what role they play in its pathogenesis. Confusion in the literature stems from the all-too-frequent assumption that factors which provoke asthma attacks also cause the condition itself. It is extremely important to clarify what is understood by the term as it applies to asthma. There are three separate, though interrelated, aspects of the etiology of asthma. Inception is the period when the bronchi first develop the capacity to undergo narrowing response to various provocative factors. Next occurs maintenance and reinforcement of this state of hyperreactivity. Third, hyperreactive bronchi are provoked, giving rise to clinically recognizable attacks of asthma.

The role that behavioral factors may play in the etiology of asthma has to be analyzed in the light of these distinctions. Behavioral factors cannot be considered to be the same as psychologic factors, although often it is impossible to make a clear distinction between them. Behavior can be recognized as a response to a determined stimulus. But in this context, patterns of asthma behavior could be due either to inherent psychologic factors associated with a distinctive type of personality, or they might be secondary to the limitations imposed by asthma on a normal life style.

Behavioral Factors in the Etiology of Asthma

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Supporters of the psychosomatic theory of asthma have made great efforts to prove that the personality and resulting behavioral patterns of individuals with certain bodily diseases are causally related and very specific to them. Several points of criticism can be raised against these conclusions. Much of the information is subjective or has come from studies in which a considerable bias arose due to the selection of patients who sought medical or psychiatric care. Hence, such patients may have been far from representative of the general asthma population. As has been pointed out by Menges, this "distortion due to selection" could have a profound influence upon the results of psychometric testing. Furthermore, since pre-asthmatic personality features and behavior patterns could not be ascertained, the findings made after the development of asthma may very well have been secondary to the disease.

Some studies have avoided these methodologic shortcomings by, for example, comparing psychologic characteristics of asthmatic patients with those of normal healthy subjects. They revealed no significant personality traits in either adults or children with asthma. In 1979, Creer concluded from a comprehensive review of the literature that it was justifiable to denounce even the slightest suggestion that personality profiles or psychodynamic conflicts may induce asthma.

A growing number of psychologists and psychiatrists disagree that there is a causal relationship between an "asthmatic personality," or a specific emotional conflict, and the inception of the disease (a comprehensive review is given by Kaptein). On the basis of controlled observations it would suggest that psychologic abnormalities and resulting patterns of behavior are rather a byproduct and unrelated to the inception of asthma.

Maintenance and Reinforcement of Asthma

The factors implementing the maintenance or periodic reinforcement of airway hyperreactivity are obscure. It is conceivable that repetitive exposure to some factors provoking asthma attacks might enhance increased bronchial hyperreactivity. In this context the maintenance and reinforcement of hyperreactivity appears to be closely related to provocative agents.

Cohen and Lask argued that, on the basis of a pathophysiological substrate, any form of emotional arousal may trigger asthma attacks. Obviously, there are emotional reactions to an asthma attack such as anxiety, fear of death, or increased dependence on others. For instance, initially, asthmatic attacks may be provoked by allergy, or recurrent infections, but later, emotional factors may become predominant in provoking attacks of asthma.

Based on the principle that behavior is learned, it has been suggested that this occurrence of asthma attacks can be precipitated by conditioning. Clinicians recognize a situation where a subject who knows that a particular flower has caused attacks previously will develop such an attack merely by seeing a paper imitation of the flower. A pattern of behav-
Behavioral factors have not been convincingly shown to play a relevant part in the inception of asthma. Most of the supporting findings resulted from subjective and uncontrolled observations containing numerous ambiguities and contradictions. However, further detailed control studies are necessary to evaluate whether behavioral factors per se can maintain a state of hyperreactivity of the bronchi once it has been acquired and thus act indirectly to enhance respon-

Recently, Neild and Cameron demonstrated that, in a highly selective group of asthmatic subjects, bronchoconstriction was induced by suggestion without evidence that it was due to airway cooling.

The likelihood that suggestion per se is capable of altering the bronchomotor tone poses the question of what the possible mediating mechanisms might be. McFadden et al and Neild and Cameron were able to show that an asthmatic response to suggestion was abolished by atropine therapy. These findings indicate that suggestion-induced airway constriction may be mediated through cholinergic pathways.

That brings up the question of how cholinergic pathways become involved. Converging evidence from immunology, neuroendocrinology, pharmacology, and physiology indicates that there exists an integrated network of neuroendocrine-immune-system interactions which is subject to regulation or modification by the brain.

It is obvious that the brain, previously considered to lack immunocompetence, can alter immune response. From animal experiments, direct evidence was obtained demonstrating that electrolytic lesions or electrical stimulations of selected area of the hypothalamus could change immune function. Destruction leads to immune suppression and stimulation to enhancement of immunity. Blalock pointed out that reciprocal interactions between the central nervous system and the immune system must be mediated by neuropeptides. Control of the immune system by the brain has been elegantly demonstrated using classic Pavlovian conditioning to suppress immunologic responses. Moreover, hypnosis has been shown to have an influence upon immunologic processes. Under deep hypnosis, immunologic phenomena (ie, tuberculin reactivity and allergic induced skin reactions) can be abolished. It has been suggested that hypnosis causes a physical stimulation of the brain, paving the way for mechanisms to alter immunologic reactions. It is conceivable that behavioral factors could implement physiologic events in the brain which lead to a disturbance in the balance of homeostasis and render the bronchi more susceptible to other factors which also are capable of disturbing it.

Most of the experimental data have been obtained from animals, and no study has yet been carried out on the psycho-immunologic aspects of asthma. Evidence from studies of hypnosis in asthma and other conditions suggests that the neuroendocrine system could be regulated or modified by events in the brain, including emotional experience. Thus the concept of psychoimmunologic reaction is still hypothetic. But further research in this field may well prove to be rewarding and lead to an extension of our understanding of asthma and its management.

CONCLUSION

Behavioral factors have not been convincingly shown to play a relevant part in the inception of asthma. Most of the supporting findings resulted from subjective and uncontrolled observations containing numerous ambiguities and contradictions. However, further detailed control studies are necessary to evaluate whether behavioral factors per se can maintain a state of hyperreactivity of the bronchi once it has been acquired and thus act indirectly to enhance respon-

**Figure 1.** Possible role of behavioral factors in provoking asthma attacks due to a conditioned stimulus.
siveness to any form of provocative factor, including those of an emotional nature. Future research should consider psychoneuroimmunologic aspects which might explain the role of behavioral factors in terms of disturbance of brain-controlled neuroendocrine-immune mechanisms. It will be essential to avoid methodologic shortcomings through the close cooperation of psychologists, scientists and clinicians.

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A Diet Rich in Sodium May Potentiate Asthma*

Epidemiologic Evidence for a New Hypothesis

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The geographic variation in asthma prevalence and mortality has not been explained adequately. The marked increase in prevalence in communities adopting a more Western lifestyle suggested that some of this variation could be due to changes in sodium intake. The theory was tested using regional data from England and Wales and a strong correlation was found between table salt purchases and asthma mortality in both men and children. Asthma mortality in women was more closely related to prevalence of cigarette smoking. The association was not explained by differences in age, sex or prosperity.

Prevalence and mortality rates for asthma show significant variation not only in developing countries where this is most marked, but also within Great Britain. Despite intensive research into the etiology of asthma, there is no coherent explanation for these geographic variations. Explanations which seem plausible in terms of individual subjects do not easily translate into convincing accounts of the epidemiologic data.

Differences in genetic susceptibility are unlikely to be the cause of this variability. The impressive increases in the prevalence of asthma in migrants from rural underdeveloped to urban Westernized areas is well documented. This supports the evidence of genetic studies which have shown differences in asthma status between monzygotic twins, and relatively poor heritability of the response to inhaled methacholine. It is not easy either to defend the assumption that differences in the prevalence of asthma reflect differences in the prevalence of atopy. The lack of standardized measure of atopy makes the interpretation of information on this subject particularly fallible. However, two studies in developing

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