Causes of asthma

Asthma is a chronic lung disease that inflames and narrows the airways. It can be both short term and long term. Asthma causes recurring periods of wheezing (a whistling sound when you breathe), chest tightness, shortness of breath, andcoughing. The coughing often occurs at night or early in the morning. Asthma affects people of all ages, but it most often starts during childhood. In the United States, more than 25 million people are known to have asthma. About 7million of these people are children.

The airways are tubes that carry air into and out of your lungs. People who have asthma have inflamed airways. The inflammation makes the airways swollen and very sensitive, the airways tend to react strongly to certain inhaled substance. When the airways react, the muscles around them tighten. This narrows the airways, causing less air to flow into the lungs. The swelling also can worsen, making the airways even narrower. Cells in the airways might make more mucus than usual. Mucus is a sticky, thick liquid that can further narrow the airways. This chain reaction can result in asthma symptoms. Symptoms can happen each time the airways are inflamed (national institute of health).

Understanding and Explaining Asthma

The aetiology of asthma has been explained in terms of heredity, incitement, and induction. ‘ Incitement’ is the immediate broncho-constriction noted among asthmatics and others to ‘ nonspecific’ stimuli. Induction is the enhancement of the response to ‘ inciters’ that can follow exposure to certain stimuli, mostly inflammatory agents such as allergens, infections and other irritants, including industrial contaminants such as`4he plicatic acid

from Western Red Cedar wood. There is a good reason to believe in a hereditary component to asthma, different components of the condition having possibly independent genetic origins. The best documented genetic component is the familial allergy to ragweed6, and Sibbald et al. 7 have argued for independent hereditary components that influence atopy and bronchial responsiveness. However, migrant studies strongly suggest the major determinant to be environmental and not genetic. Both Tokeluans4 and Xhosas3 clearly have the genetic potential for developing asthma, but this is expressed only on migrating to an economically more developed area (Journal of the Royal Society of Medicine Volume 80 June 1987 367).

Isolated examples, such as the increase in asthmatics attending emergency rooms following the eruption of the Mount Helen’s volcano8, may be explained in terms of incitement, but there are major objections to using this mechanism as a general explanation for differences in the prevalence of asthma. The wide variety of substances capable of inducing broncho constriction in asthmatic subjects can be divided into endogenous mediators such as histamine, prostaglandins or leukotrienes, and exogenous agents such as sulphur dioxide, volcanic ash or cold dry air. For the epidemiologist attempting to explain the prevalence of asthma, the endogenous mediators are unhelpful. The discovery of a mediator whose presence distinguishes asthmatic from non asthmatic populations would be useful, but in turn would raise the question of why one population had more of the mediator than the other population. For the reason discussed above, a genetically controlled explanation can be discounted, which leaves an environmental factor to increase the amount of mediator in the asthmatic population. This is

essentially the same as the initial question of what environmental factor might account for differences in the prevalence of asthma.

Exogenous inciters, being environmental in origin, theoretically offer a better hope of advancing the issue further, but, in the event, are no more helpful. Differences in the prevalence of asthma are not accounted for simply by differences in the incidence of clinical symptoms provoked by environmental triggers. They also reflect differences in the under-lying prevalence of bronchial hyper-responsiveness. In van Niekerk's study3, it was the response to the exercise challenge that differed between urban and rural areas. This cannot be explained simply in terms of the prevalence of inciters in the environment. ' Induction' provides a more promising general theory with which to explain differences in prevalence. There is plenty of evidence that induction does occur, particularly in an industrial context and this has been used extensively as a general model for the aetiology of asthma. It does not, however, explain the distribution of asthma in developing countries. Agents that have been shown to induce asthma are abundant in the Third World (Journal of the Royal Society of Medicine Volume 80 June 1987 367).

Fatality of Asthma

Acute respiratory infections are probably the single most important cause of death among children. It is thus appealing to consider the hypothesis that fatal and near fatal attacks of asthma may have common causes, and that studying non-fatal asthma attacks (as well as being of value in itself) may provide useful information on the factors associated with fatal asthma attacks. Campbell investigated the levels of agreement between information

obtained directly from cases of near fatal asthma attacks with those obtained independently from close acquaintances, almost all of whom were family members living in the same household. They found very high levels of agreement for recognized markers of chronic asthma severity (such as a hospital admission in the last 12 months or an intensive care unit admission), utilization of medical services, the use of prescribed agonists (although there was less agreement for other asthma medications such as oral xanthines), and psychiatric features. These findings are generally encouraging in that they indicate that information relating to these risk factors and clinical characteristics obtained from close acquaintances has reasonable reliability in studies of near fatal asthma attacks and therefore such information may also be reliable for persons dying from asthma. In contrast, information on features of the long term asthma history such as sleep disturbance and the frequency of, or trends in, symptoms in previous years was not reliable. The reliability of the reported circumstances associated with a near fatal episode was not examined. Thus, while it would seem appropriate to continue to examine deaths from asthma using clinical information that has been recorded before the fatal attack, the findings of Campbell suggests that interviews with acquaintances may provide additional useful data. Richards have compared the demographic characteristics of patients suffering fatal asthma attacks throughout New Zealand during 1980-6 with those experiencing severe life threatening asthma attacks in the Auckland region during 1981-7. The information avail-able for the study was limited, but the findings once again confirm the importance of a previous life threatening attack of asthma as a marker of subsequent risk of death from asthma. About one quarter of the subjects who died in Auckland during the period of

the study had a previous admission to an intensive care unit with asthma. This suggests that asthmatic subjects who experience a life threatening attack are a group in whom intensive medical treatment and follow up should be arranged. These considerations indicate that, although there are some practical advantages in studying near fatal asthma attacks, there may well be greater problems of interpretation of the findings than in studies of fatal asthma. Nevertheless, when interpreted cautiously, studies of near fatal attacks may provide a useful complement to studies of fatal attacks in monitoring the time trends in severe asthma, and in identifying the causes of those changes which occur (Thorax 1993; 48: 1093-1094).