# **CORRESPONDENCE**

# Symptoms of asthma, bronchial responsiveness and atopy in immigrants and emigrants in Europe

To the Editor:

I read with interest the article by Tobias *et al.* [1]. Firstly, I would like to state my doubts on the validity and relevance of using the umbrella term "immigrants" in reference to people from various countries of the world who settle in developed countries.

Most of the time, leaving one's home country is not a deliberate action. The main initiatives are related to economical and political reasons. Those who immigrate for economical reasons usually use legal routes. The legal process makes it obligatory to have a health check and does not permit diseased individuals to migrate. This obligation does not hold true for the immigrants who leave their countries for political reasons. Political immigrants are however, still expected to have a relatively better health status than the general population to enable them to escape the legal barriers and leave their country. The observation in Turkish workers and their families in Europe may give the main supporting evidence for this view. The prevalence of asthma, atopy and bronchial reactivity was even lower in second-generation Turkish children when compared with children of native German and Swedish populations [2, 3]. Immunoprotection is probably a multifactorial phenomenon that presumably includes bacille Calmette-Guérin (BCG) vaccination status, diet, breastfeeding, tobacco consumption, type of fuel used at home, infections in the early life (due to bacteria, viruses, and parasites), and genetic factors. In addition, I believe that a health-selection factor (similar to the "healthy worker effect") is involved in immigrants.

Different fates may be anticipated for the immigrants in their new countries. Frequently, they work in jobs that native people do not want to do. While a minority adapt to the new society (language, diet, hobbies, etc.) without any problems, most of the others suffer great difficulties in adapting. The latter "dive in their own ghettos", carry on their own traditions, consume their special food, try to establish their own business if they can, and visit their home town whenever possible. These two groups of immigrants, who share the same environment but have different levels of adaptation, could be compared to Germany before the unification.

Language has been cited as an outstanding problem in the epidemiological studies of asthma. "Wheezing" has been regarded as an awkward word, even in English speaking countries. As far as I know, "dyspnoea" is used as a diagnostic equivalent in many countries and asthma is a fearful "foreign phrase" for society. Thus, clinicians in various countries of

the world, have used alternative terms, such as allergic bronchitis, bronchitis and spastic bronchitis. In some African languages, there is no synonym for asthma.

It has been reported that prevalence of asthma is increasing in developed countries, whereas the status in less developed countries is not well known. For example, there was no significant difference in the prevalence of asthma in a series of cross-sectional studies conducted 5 yrs apart in Ankara, Turkey [4]. This is an important factor for the prevalence of asthma and allergic diseases in immigrants from less developed countries.

Another important factor is the duration immigrants stay in a new country. In Uppsala, Sweden, total immunoglobulin (Ig)-E levels of nonatopic immigrants from various parts of the world were higher than that of the nonatopic-, age- and sex-matched native Swedish people [5]. Total IgE levels of the immigrants from various parts of the world showed notable differences. This difference in the level of total IgE, which is used as a rudimentary marker of allergy and asthma in epidemiological studies, is likely to disappear after living in the new country for  $\geq 10.5$  yrs [5]. During the same period, allergen spectra in skin testing of atopic immigrants resembles that of the native population [5]. Another study conducted in Turkish immigrants in Sweden supported this finding [6]. In the atopic Turkish immigrants, sensitivity to birch, cat, and dog increased in parallel with the changes in their living conditions. We can therefore say that immigrants gain immunological adaptation to the new environment within a mean period of 10 yrs (time effect). Thus, investigations of asthma in immigrants should also assess the duration of stay in the new country.

In my opinion, studies conducted on immigrants should take into account the differences that exist within the immigrant populations. The problems of adaptation to the new society and duration of stay in the new country should also be carefully addressed. This is a promising field, as prospective studies conducted on immigrants could provide valuable information about protection from asthma and allergic diseases.

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## From the authors:

We appreciate the interest of A.F. Kalyoncu in our paper, and his comments on the possible healthy migrant effect and on the social and economic implications of moving abroad. The comprehensive research from Kalyoncu and co-workers [1, 2] on Turkish migrants is of great interest. As we mentioned in the Discussion section of our paper [3], the sampling frame of the European Community Respiratory Health Survey was from official sources in each of the participating countries and our conclusions apply only to "legal" migrants. Nevertheless, within this restricted group, the data showed an increased reporting of asthma, but neither bronchial responsiveness nor atopy, among migrants compared with

others living in both their host and native countries for a wide range of environments.

As medicine and public health is provided in a world with more extensive migration, we need to bear in mind the health needs of all kinds of migrants, and devise effective means of preventing and managing their respiratory or other health problems. The good news is that within the centres studied in the European Community Respiratory Health Survey the healthcare received by legal migrants, at least, is not greatly different from the population of the host countries.

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# Evaluation of bronchodilator responses in patients with "irreversible" emphysema

To the Editor:

In a recent paper by O'Donnell et al. [1], the "hidden" reversibility of a component of overinflation in emphysema, which pertains to the changes in the inspiratory capacity (IC) rather than to forced expiratory volume in one second (FEV1), is discussed.

Since a large number of studies, in addition to that by O'Donnell *et al.* [1], have appeared on this subject in recent years (MILIC-EMILI [2] along with TAUBE *et al.* [3] and numerous others), I would like to direct the

reader's attention to the observation by Labadie [4]. In 1960, he demonstrated an isolated increase in the IC in chronic obstructive pulmonary disease (COPD) after isoprenaline (figs. 1 and 2). This phenomenon has been known to the older generation of central European pneumologists as being an important hint of the subjective improvement of patients with severe COPD after bronchodilation, in the absence of an increase in FEV1.

Since an increase in the inspiratory capacity is usually accompanied by a decrease in thoracic gas

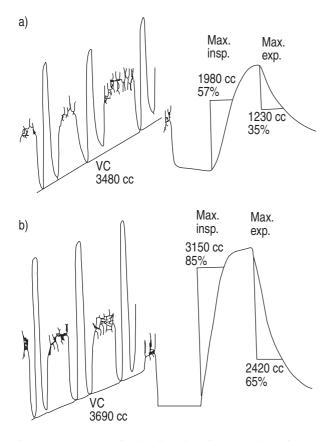


Fig. 1.—Improvement in the forced expiratory volume in one second and the forced inspiratory volume in asthma in response to a bronchodilator. Asthma a) before and b) after isoprenaline. VC: vital capacity; Max insp.: maximum inspiratory volume; Max exp.: maximum expiratory volume; cc: cubic centimetre.

volume, it can be postulated that the subjective improvement of dyspnoea is based on an improvement in the breathing mechanics, due to a decrease in overinflation and intrinsic positive end-expiratory pressure, particularly during exercise.

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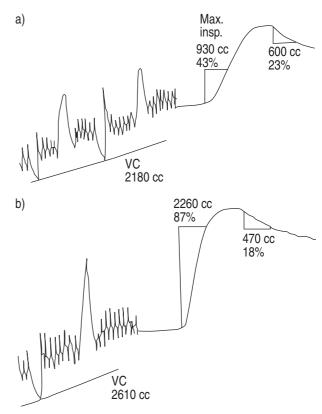


Fig. 2.—Improvement in the inspiratory capacity and forced inspiratory volume but not the forced expiratory volume in one second after bronchodilation in severe chronic obstructive pulmonary disease (COPD). COPD a) before and b) after isoprenaline. VC: vital capacity; max insp.: maximum inspiratory volume; cc: cubic centimetre. Quoted from [2] and reproduced from a historical copy owned by N.G.M. Orie, University of Gronigen, Gronigen, Winsum, the Netherlands, personal communication, 1994.

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## From the authors:

We thank F. Kummer for pointing out the observation by Labadie [1] in 1960, on the acute effects of isoprenaline on inspiratory capacity in chronic obstructive pulmonary disease (COPD) and asthma. The idea that lung hyperinflation contributes to respiratory discomfort in obstructive airways disease, and that its reduction by bronchodilator therapy may result in symptom alleviation, is certainly not new and has been held by many pneumologists worldwide, not only those of Central European origin! The main purpose of our study was therefore, to challenge current concepts of "irreversibility" in COPD. An exclusive focus on the forced expiratory volume in one second (FEV1) as the physiological outcome measure of interest in clinical trials of bronchodilators has led to a significant underestimation of the beneficial effects of these drugs on small airway function, lung

CORRESPONDENCE 983

deflation, and symptom reduction, particularly in those with severe disease. We believed it was important to point out that the greatest lung volume responses to bronchodilators occurred in those with the most severe baseline pulmonary function impairment.

The recent development of validated scales to measure dyspnoea has led to the systematic study of sensory-mechanical interrelationships during rest and exercise in chronic obstructive pulmonary disease. These studies have confirmed the important causative role of resting and dynamic hyperinflation in dyspnoea and exercise intolerance [2]. Indirect proof of the importance of dynamic hyperinflation has come from a number of studies that have shown that dyspnoea relief, and improved exercise endurance following bronchodilator therapy [3, 4], surgical lung-volume reduction [5], and ambulatory oxygen [6, 7], is closely linked to the reduced lung volumes achieved. Renewed interest in lung hyperinflation as an important and potentially reversible factor in contributing to the disability of chronic obstructive pulmonary disease, has prompted a fresh therapeutic optimism for the future management of advanced symptomatic disease. The ability of newer, long-acting bronchodilators to achieve sustained (24 h) pharmacological lung-volume reduction is a significant therapeutic advance.

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