In a great many jobs, workers inhale aerosol substances that can cause asthma, aggravate pre-existing asthma, or cause symptoms that mimic asthma. Both occupational asthma and work-aggravated asthma are common entities. Ten percent of adult asthma cases are thought to be job-related, and more than 25% of asthma patients of working age can suffer exacerbations related to their working conditions.1

In occupational asthma, the best therapy is to avoid the causal agent; this involves leaving the job and obtaining a certificate of incapacity due to an occupational disease, which would entitle the patient to receive disability benefit. Being classed as “unfit for work” is not always advantageous, and depending on the age, profession and social and economic circumstances of the worker it can even be detrimental to a greater or lesser extent. However, in the case of work-aggravated asthma due to non-specific stimuli, controlling factors such as cold, irritants, exercise, stress and treatment non-compliance, and facilitating adherence and optimizing therapy can often suffice to allow a return to work. Other processes that mimic asthma, such as reactive airway dysfunction syndrome, irritable larynx syndrome and COPD, however, must be ruled out. Therefore, when a worker reports asthma symptoms linked to occupational exposure, it is essential, from a therapeutic, occupational and economic perspective, to determine which process is involved.2 Occupational asthma must be diagnosed as soon as possible on the basis of objective tests that can ensure the highest degree of certainty; this, however, is far from simple. Scientific societies and groups of experts have devised algorithms that can be used with available tools to facilitate what is essentially a complex diagnosis.4,5 Today’s recommendations are reliable and can help clinicians take the right decisions.6

According to the widely accepted definition, occupational asthma is “a disease characterized by variable airflow limitation and/or airway hyperresponsiveness due to causes and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace”,7 and the first step is to confirm the presence of the disease. Diagnostic criteria for asthma, which mainly consist of confirming reversible obstruction, bronchial hyper-responsiveness and characteristic inflammation, are clearly defined in universally accepted guidelines.8 An important factor to bear in mind in occupational asthma is that prolonged separation from the causal agent can mitigate these manifestations; therefore, it is best for the patient to continue working while the physician immediately starts diagnostic studies. Only once asthma has been objectively confirmed should the next step be taken, namely, establishing the causal relationship with a specific agent present in the workplace.

In most cases of occupational asthma, the pathogenic mechanism is an allergic response to high or low molecular weight antigens, either through the interaction with specific IGE antibodies or by other immune mechanisms. Irritant-induced asthma is not caused by an allergic response, and as such is beyond the scope of this article. A great many asthma allergens have been described, with latency periods from first exposure ranging from weeks to years. In all cases of adult-onset and poorly controlled asthma it is essential to obtain a detailed occupational history of the patient. Symptom onset can vary, depending on the length of exposure and the trigger allergen. Rhinitis often precedes asthma symptoms, although this has been shown to have a low positive predictive value and should never suffice for a diagnosis of occupational asthma.9

Positive skin prick tests indicate sensitization and help identify the likely trigger. However, such tests have poor sensitivity and specificity, and it is important to bear in mind that they cannot identify the target organ of the allergic response, and therefore cannot diagnose asthma when used alone. Once the disease has been diagnosed, the causal relationship must be identified, starting with an observation of changes in clinical signs, lung function (serial peak flow measurements and hyperresponsiveness to methacholine) and markers of inflammation (eosinophil count and measurement of exhaled nitric oxide) in response to exposure and separation from the workplace trigger in alternating periods of at least 2 weeks. Evaluating the results of these studies as a whole will increase their diagnostic yield, and although they have their limitations, they are often sufficient to give a high degree of certainty.

The specific bronchial challenge test is the gold standard for identifying the allergy trigger; however, it is rarely used and poorly implemented.10 In some hospitals, it is a first-line procedure, although in most it is used when alternative methods have
failed to provide conclusive evidence. Other indications include identification of new triggers not previously described, cases where re-exposure to the workplace is impossible, and as a gold standard test in research.

Diagnosis of irritant-induced asthma depends on evidence of asthma together with a history of exposure and clinical signs, none of which are wholly conclusive. All the aforementioned studies will only yield reliable results when performed following strict quality and safety standards, but they are time-consuming, and not all hospitals have the required resources. Moreover, it is important to gain the trust of the worker and help them understand the need for patience and cooperation in reaching a firm diagnosis that will always be to their advantage. The cost of obtaining an accurate diagnosis is no higher than the cost of poorly controlled disease.11

A brief look at current recommendations reveals areas of improvement, and it is clear that diagnosis is a process that involves the general practitioner, occupational physician, pulmonologist and allergist, and often requires specialized units. Professionals in the field of occupational medicine and primary care should bear in mind that: (1) Occupational asthma should be suspected in all workers presenting with initial symptoms of asthma or with poorly controlled disease, (2) A comprehensive occupational history is crucial when dealing with workers with respiratory symptoms, (3) Not all asthma related with a patient’s job is necessarily occupational asthma, (4) Occupational asthma cannot be ruled out on the basis that the job is not considered high-risk, and (5) Both causal and aggravating factors can be present, and non-specific causal factors such as fungi and plants can be found in the workplace. In addition to these factors, pulmonologists must ensure they reach an objective diagnosis of asthma, and when they lack the resources needed to establish a causal relationship, they must refer the patient to specialized units to complete the diagnostic process and confirm their suspicions.

The epidemiological significance of occupational asthma is beyond dispute. In recent years, a number of tools have been developed to help physicians overcome diagnostic uncertainty. Despite this, the data show that occupational asthma is under-diagnosed, and it is up to healthcare professionals to make good use of existing recommendations to improve their diagnostic skills and improve their understanding of this disease.

**Conflicts of Interest**

The authors declare they have no conflicts of interest.

**References**