to conservative treatment, or a history of cancer lead us to suspect this entity. The lack of early recognition leads to a delay in diagnosis and, consequently, inadequate treatment. As a result, a simple X-ray and a histopathological exam should be performed in all cases of doubtful interpretation.

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The Athlete With Asthma and the New 2010 Anti-Doping Regulations. Less Work to Change for a Limited Therapy

El deportista con asma y la nueva normativa antidopaje de 2010. Menos trabajo a cambio de una terapia limitada

To the Editor:

Clinical manifestations of greater bronchial reactivity in athletes are no longer news in our specialty or even in the world of sports journalism. Undoubtedly for many years the excess of information on the fact has been related to something more scandalous, the world of doping, an aspect that has had to be addressed.[1] This has been so since the swimmer Rick Demont at the Munich Olympic Games in 1972 had his medal withdrawn because he tested positive to a substance considered to be used for doping, which was a characteristic of the medication he was using to treat his asthma. To date, the road has been a long one. The most significant facts are: athletes with asthma are allowed to use medication to treat their condition. Initially it was only necessary for a physician to indicate that the athlete suffered from asthma, subsequently medication was restricted to certain drugs, and for more than a decade now, control has been more exhaustive. Regulations were adjusted to include the use of only two models of short and long lasting beta agonists and the application that must be sent to the evaluating agency, the therapeutic use exemptions (TUEs), must comply with certain requirements. A sine qua non condition was that bronchial liability had to be demonstrated by the bronchodilator test or a certain degree of bronchoconstriction be determined by the test performed, isocapnic hyperventilation, stress, mannitol, metacoline or hypertonic saline.[2] I must add that the metacoline test is considered positive internationally at $PD_{20}FEV_1$ < 4 mg/ml whereas nationally a dose of < 8 mg/ml is admitted in subjects that have already been treated with steroids. In both cases, these are acceptable criteria although somewhat demanding for a test that only measures bronchial hyperreactivity in an individual with a history compatible with sports asthma.[3] How difficult it has been to achieve these criteria in some athletes during good periods. We know that not a few of them have stopped using medication for fear of being found positive to doping at moments in which their TUE was not in order, or even if it was, with the consequent risk to their health and performance. The TUE must be requested annually with the consequent discomfort for the patient and investment of time for them and the physician/s responsible for the process and the follow-up of the athlete. However, it finally seems that the good judgments fairies have illuminated (without any darkness) the factotum of the organizations in charge of this process. The regulations of the World Anti-Doping Agency (WADA) that comes into force on the 1st of January 2010, says on this matter:[2] "All beta-2 agonists are banned with the exception of salbutamol (maximum 1,600mcg in 24 hours) and salmeterol, both by inhalation, and they require a declaration of use according to the International Standard for Therapeutic Use Exemptions. The presence of salbutamol in urine at values > 1,000 ng/ml is presumed not to be due to therapeutic use of the substance and will be considered an adverse analytical result while the athlete does not prove, by means of a controlled pharmacokinetic study, that the abnormal result was the consequence of the use of therapeutic doses (maximum 1,600 mcg in 24 hours) of inhaled salbutamol" (text similar to Spanish regulations BOE 25/12/2009 page. 109757). Therefore, we are all free of the hassle of carrying out tests and filling in reports and papers for merely bureaucratic reasons and we are only requested to indicate that the subject uses medication for asthma. We can administer what treatment we consider opportune steroids, antileukotriene agents, cromomes, antihistamines, immunomodulators…But, be careful! For beta-agonist bronchodilators only salbutamol and/or salmeterol can be used. For the time being we must forget terbutaline and formoterol, which were allowed before or request a TUE according to the rules for banned medication and see what happens. The decision to remove these beta-agonists is not justified in the text. Is there any scientific reason? In principle, in the case of salbutamol there is a way of quantifying exactly the concentration in urine as well as identifying possible oral administration with a certain degree of precision,[4] which is difficult with terbutaline.[5] Maybe more work should be done on this aspect? Certainly, if necessary. However, the possible effect of therapeutic doses as an anticatabolic agent or a CNS stimulator is inexistent or irrelevant for both substances,[6] and so far this has been considered so. What is the reason for the change? International organizations, the AMA and the national State Anti-doping Agency (AEA) should make statements in this sense, since we would like to present reasonable arguments to the reason for this modification. However, quod sripsi, scripsi. We must try to think how we can modify confidence in treatment using these products, if the regulations are not changed within a prudent period of time. And what will happen with those athletes who have TUEs for use during the (2009-10) season? I understand these should be admitted, but they might not. Once more, it is necessary that the AMA and AEA make statements. Finally, to complete the information

and answer queries on asthma and doping, Royal Decree 641/2009 of the 17th of April, BOE 8/5/2009, that regulated doping control processes says, "All athletes with a license to participate in official state competitions may be selected at any time to undergo tests during competitions or outside competitions". No comments.

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Mortality in Lung Cancer and COPD

Mortalidad en cáncer de pulmón y en enfermedad pulmonar obstructiva crónica

To the Editor:

In the interesting article by Abal et al[1] entitled «Lung Cancer and COPD: a frequent association» a broad clinical cohort of 996 patients over a period of 5 years is assessed. The first conclusion is relevant and current: The association of both pathological conditions is frequent, and the most frequent histological diagnosis is squamous carcinoma; however, it is more difficult to take in its second and significant conclusion. The survival of patients with lung cancer and COPD is greater than that of patients with lung cancer without COPD. This second finding seems contrary to all that has been published so far with reference to comorbidities of both conditions, and we make reference to the excellent review in these same pages by Díez Herranz[2] in 2001 and other more recent sources.[3-5] It has even been reported that greater mortality has been seen in non-smokers with both conditions.[6] The authors themselves are surprised and theorize in the «Discussion» about a possible diagnostic bias and about the fact that patients with COPD may be diagnosed with lung cancer before patients without COPD. Probably, a repeat analysis of the data would make it possible to reconsider said conclusion, based on a view of the Kaplan-Meier survival curves (Fig. 1) and a significant difference of p = 0.016 obtained using the Mantel-Haenszel test (logarithmic ranges). Maybe due to the baseline differences between both groups (Table 4), it would be more appropriate to use an adjusted Cox regression model for the significant variables in the bivariate analysis, including sex, age, smoking and stage of lung cancer or other variables. The time in months/years between COPD diagnosis and cancer diagnosis could also be modelled. Finally, although in «Methods» section it is indicated that diagnosis and COPD classification were carried out according to GOLD directives, it would be interesting to repeat the same model eliminating those COPD cases diagnosed without spirometry.

Nevertheless, as Brody and Spira[3] state, most smokers never will develop either COPD or lung cancer, it is important to investigate this relationship in detail.

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Response by the Authors

Respuesta de los autores

To the Editor:

We thank Joan B.Soriano for the interest shown in our recently published article.[1] We consider his comments are relevant and correct, and the suggestions undoubtedly improve on the original.

Following his advice, we have re-analysed the data eliminating the 16 COPD patients without spirometry. The Kaplan-Meier survival curves continue to be significantly different between patients with and without COPD, being greater in COPD patients (p = 0.006).

Indeed, as the author points out in his letter, and this is seen in the results, an adjusted Cox regression model was used for significant variables in the bivariate analysis, although only stage and treatment remained in the final model. COPD, on the contrary, was not statistically significant. In the discussion we commented on the