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*From the authors:*

According to our study, although more evidence is needed, the existence of asthma is associated with an independent increase in risk of bronchiectasis exacerbation [1]. Bronchiectasis and asthma share some similarities in clinical characteristics and bronchial hyperresponsiveness, which would bring some diagnostic challenges of asthma in bronchiectasis patients. As shown by Guan *et al.* [2], reversible airway obstruction or hyperresponsiveness can occur in patients with bronchiectasis. If the extent of reversibility in those patients meets the diagnostic criteria of asthma, they should also be diagnosed with asthma. In our study, the diagnosis and recruitment of subjects was in strict accordance with the relevant guidelines [1, 3–6]. Diagnosis of bronchiectasis was performed using chest high-resolution computed tomography scans in suspected patients with coughing and expectoration, or long durations of haemoptysis. An exacerbation is defined as the patient reporting four or more of the following symptoms: change in sputum production, increased dyspnoea, increased cough, fever >38°C, increased wheezing, decreased exercise tolerance, fatigue, malaise, lethargy, reduced pulmonary function, changes in chest sounds, or radiographic changes consistent with a new infectious process [1, 3, 4]. Asthma was diagnosed for patients with symptoms such as episodic breathlessness, wheezing, cough and chest tightness, and whose spirometry showed bronchial reversibility of 12% and 200 mL from the pre-bronchodilator value or airway hyperresponsiveness as a 20% decrease in forced expiratory volume in 1 s ($FEV_1$) caused by a provocative histamine with a cumulative dose <2.4 mg [5, 6].

After the follow-up, 97 patients had at least one bronchiectasis exacerbation within 1 year after their discharge from the hospital (49 patients with bronchiectasis alone and 48 patients with concomitant asthma). In our study, we used the end-point (exacerbation or not) as a binary variable to make a logistical regression, as other researchers have [1, 7]. Because of the different durations of follow-up, bronchiectasis exacerbation within 1 year after discharge from the hospital was chosen as the end-point in order to keep consistency. The rate of exacerbation, as usually used in a prospective study, would only conclude the difference of the mean exacerbations of the two groups, and not the hazard ratio of each variable, which was the main purpose of our research.

It is true that the patients we recruited from inpatients admitted with bronchiectasis were more likely to have severe clinical characteristics and poorer $FEV_1$ [8]. However, since the comparisons between both groups in this study were at the same stage, the results still have significant clinical value. In addition, the data from pulmonary function tests were collected when the patients were relatively stable.

Finally, all of the patients with bronchiectasis were routinely asked to sign a consent form when they were admitted to the hospital in our department. Patients signed the consent form to authorise follow-up every 3 months through face-to-face interviews, or telephone interviews if that was not convenient. The reason and date of the exacerbation were recorded according to their medical records. Our study sought to observe the impact of asthma on bronchiectasis exacerbation in a real-world setting. The diagnosis and treatment of bronchiectasis concomitant with asthma remains challenging, and more accurate spirometric and scoring diagnostic methods are needed.

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**Asthma increases the risk of bronchiectasis exacerbation; however, more evidence is needed**
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# Obstructive sleep apnoea and bone health

*To the Editor:*

We thank Liguori *et al.* [1] for sharing their interesting study about patients suffering from obstructive sleep apnoea (OSA) and their tendency towards lower bone mineral density (BMD) in the lumbar spine and femur compared with control subjects matched for age, body mass index and physical activity. The researchers defined "osteopenia" as a T-score value $<-1$ SD and "osteoporosis" as a T-score value $<-2.5$ SD. They also suggested that OSA could be detrimental to BMD, resulting in osteopenia and osteoporosis. However, there are some concerns with this study.

First, the mean±SD age of the subjects included in this study was 51.17±11.82 years in the OSA population and 51.10±11.68 years in the control group, which means that many of the subjects were <50 years old. The author classified these subjects according to T-score, which is the BMD value compared with a healthy subject of the same sex in who is at peak BMD. However, according to the recent consensus of the International Society for Clinical Densitometry [2], when researching males <50 years old, a Z-score should be used, representing a value that can be compared with those of subjects matched for age and sex. A Z-score of −2.0 or lower is defined as "below the expected range for age". We suggest that the authors