OSA patients, where we will use Z-score to define if BMD is "below the expected range for age" [2]. In fact, the ISCD proposed not to use the terms osteopenia and osteoporosis in this young population, although BMD could be severely decreased in young OSA patients, as experienced in our previous study [1].

Moreover, Zhong and Yuan also suggest quantifying the fracture risk in OSA patients, since fractures are the most serious consequence of osteoporosis. Although they propose a clinic study evaluating this issue, we suppose that this study could be troublesome to complete since OSA is treatable using continuous positive airway pressure (CPAP) therapy, which could totally restore sleep bringing it back to physiological conditions.

Therefore, although the suggestions from Zhong and Yuan are interesting, they are not applicable to our study since there is no agreement about using Z-score or T-score in large group of patients ranging from young to elderly ages. Furthermore, considering that OSA is easily treated using CPAP, fracture risk could be challenging to assess in this population.

In conclusion, taking into account that bone health could be considerably affected by sleep apnoea, future studies are needed to better score the risk for osteopenia and osteoporosis, low BMD and fracture risk at all ages in patients affected by OSA.

@ERSpublications
Obstructive sleep apnoea represents a risk factor for osteopenia and osteoporosis in male patients at all ages http://ow.ly/c9Tn302PPKz

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Received: May 05 2016 | Accepted: June 04 2016

Conflict of interest: None declared.

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Eur Respir J 2016; 48: 1249–1250 | DOI: 10.1183/13993003.01219-2016 | Copyright ©ERS 2016

Pulmonary mucosa-associated lymphoid tissue lymphoma revisited

To the Editor:

We read with great interest the review of Borie *et al.* [1] on pulmonary lymphoma of the mucosa-associated lymphoid tissue (MALT). We agree with the hypothesis that chronic antigen stimulation of microbial origin may lead to the development of pulmonary MALT lymphoma. Indeed, MALT lymphoma has been associated with various chronic infections in extrapulmonary localisations. This link has previously been demonstrated between *Helicobacter pylori* infection and gastric MALT lymphoma, and suggested with *Campylobacter jejuni*, *Chlamydia psittaci* and *Borrelia burgdorferi* with small intestine lymphoma, ocular annexa lymphoma and cutaneous lymphoma, respectively. Similarly a link has recently been suggested between infection with *Achromobacter xylosoxidans* and pulmonary MALT lymphoma [2].

Only a minority of pathogens have the ability to persist in the lung for a long period of time. Our group has investigated a link between pulmonary MALT lymphoma and mycobacterial infection for three

reasons. Persistence of *M. avium* complex has been observed in diverse organs without clinical disease in HIV infection as in animal models [3, 4]. *M. tuberculosis* DNA has been found by *in situ* PCR within the normal lung of individuals that have died from causes other than tuberculosis [5, 6]. A previously reported case suggested a temporal link between the occurrence of MALT lymphoma and mycobacterial pulmonary infection, and an improvement of a pulmonary MALT lymphoma after an anti-tuberculous therapy regimen for concomitant pulmonary tuberculosis [7]. In addition, two patients with pulmonary MALT lymphoma have been successfully treated with prolonged clarithromycin therapy [8].

We performed conventional PCR on DNA extracted from frozen or paraffin-embedded lung tissue obtained by surgical biopsies in eight cases of primary MALT pulmonary lymphoma and two controls with lung cancer (involving the lesion in MALT cases and normal parenchyma in controls). We used two couples of primers: one targeting the IS6110 gene of *M. tuberculosis* and one targeting the hsp65 gene of mycobacteria genera. We also carried out a universal bacterial PCR amplifying the gene encoding for ribosomal 16S RNA.

No mycobacterial DNA was detected. 16S rDNA PCR was positive in one patient and one control. The sequence of the amplified products gave a mix of bacteria. These PCR products were then cloned and sequenced (six clones/PCR products). The results of the sequences showed the identification of environmental species (five clones in lymphoma patients and four clones in the control cases) and *Propionibacterium acnes* (one clone in the patients and two clones in the control cases).

Hence, our results do not suggest a link between chronic mycobacterial infection and the occurrence of pulmonary MALT lymphoma. However, bacteria may be difficult to identify in tissue by PCR because of the small amount of microbial DNA in the samples used. Sensitivity varies according to the target gene and the modality of PCR [9]. More recently, new generations of PCR have been developed. High-throughput sequencing allows the detection of any pathogen, bacterial, fungal or viral, in frozen tissue without the necessity for a defined target sequence [10]. We agree with Borie *et al.* [1] that a non-targeted approach now has to be performed in order to detect whether pulmonary MALT lymphoma is associated with specific pathogens.

@ERSpublications
Our results do not suggest a link between mycobacteria and pulmonary MALT lymphoma: high-speed sequencing is needed http://ow.ly/xXXm301AQ6r

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Received: May 05 2016 | Accepted: June 04 2016

Conflict of interest: None declared.

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