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Editorial

Take your drug and climb Machu Picchu!

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Some years ago, chronic obstructive pulmonary disease (COPD) was considered as a primary bronchial disease, secondarily affecting the lung parenchyma and some other organs. The respiratory physician took care of smoking cessation and prescribed antiobstructive drugs and oxygen. This view has changed. According to an ATS/ERS task force, COPD is now considered as the pulmonary component of a chronic multimorbidity [1] and increasing attention has been paid to common underlying factors like western diet and inactivity. A growing number of COPD patients are motivated to change their life style and to live an active life including traveling and hiking at high altitudes. However, when COPD patients are exposed to high altitude they are at increased risk of complications as compared to healthy subjects - mainly due to pulmonary hypertension (PH) and exacerbation of the underlying disease.

Pulmonary vascular disease in COPD represents one of the important emerging challenges in medicine, due to the following reasons: First, the number of affected patients is high. The estimated prevalence of COPD is around 12% of the world population [3] and depending on the degree of airway obstruction, PH is present in 5–50% of patients [8]. Second, pathophysiology and clinical representation of pulmonary vascular disease in COPD is complex. The main mechanisms include tobacco smoking, endothelial dysfunction, structural and lung abnormalities, inflammation and genetic background [4]. Notably, it has been shown that cigarette smoke affects the pulmonary vasculature even earlier than the interstitial space, meaning that pulmonary vascular disease might precede the development of emphysema [9]. In addition to direct pulmonary vascular involvement, up to 30% of patients with COPD suffer from systolic or diastolic left heart failure [2] leading to a consecutive increase in pulmonary arterial pressure (PAP) and highlighting a close interplay between the airways, the heart and the pulmonary circulation [4]. This also explains why in the CHAMPION trial including patients with both left heart failure and COPD, optimized diuretic management, based on the measured mean PAP by an implanted transponder in the pulmonary artery, significantly reduced the number of exacerbations [5]. Third, and most importantly, an increased PAP in patients with COPD is associated with worse survival [10].

In the current issue of the *International Journal of Cardiology*, Lichtblau et al. report on changes in pulmonary hemodynamics in lowlander patients with COPD after ascending to 3100 m above sea level and the effects of a preventive corticosteroid treatment. Based on the results of this randomized, placebo-controlled, double-blind trial, the authors conclude that preventive dexamethasone treatment (beginning one day before ascent from 760 to 3100 m) ameliorates the altitude-induced increase in PAP [7]. The observations of this study are relevant and provide important clinical messages. Although the included COPD patients had a very mild bronchial obstruction, the observed increase in PAP in the placebo group was larger than in healthy lowlanders (11 vs. 8 mm Hg) [6]. This suggests that patients with COPD might develop a stronger hypoxic pulmonary vasoconstriction response and higher pulmonary vascular resistance values after ascending to high altitudes. This may pose them at risk for cardiopulmonary decompensation. Some of the most beautiful areas of the world including popular tourist destinations in Asia or South America lie above 3000 m, and several large cities including Mexico City and La Paz are between 2300 and 3600 m above sea level. Visiting them may be associated with significant circulatory stress in COPD patients. An even more frequent challenge is air travel, the cabin oxygen pressure being comparable to a height of around 2600 m above sea level.

The study by Lichtblau et al. provides the first evidence that dexamethasone may ameliorate the altitude-induced PAP increase in COPD patients. This may have an important impact on therapy recommendations for COPD. However, we need to define which patients have a favorable risk-benefit ratio before making general recommendations for prophylactic treatment. In addition, as COPD exacerbations are also associated with increased PAP values, further research is warranted to decipher how cardiac and pulmonary vascular factors contribute to the pressure increase and how this is influenced by prophylactic steroid treatment. Lichtblau et al. made an important contribution to the field but this was just the starting point for a number of important questions that should be answered to make world-wide traveling for COPD patients safer.

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Conflict of interest

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