high-density lipoprotein cholesterol levels. It remains unclear whether OSA or excessive daytime sleepiness affects objectively measured physical activity. In a recent study, men with untreated OSA had less metabolic improvement than obese patients without OSA after a 1-year healthy eating and physical activity/exercise intervention program. A previous randomized controlled trial found no increase in daytime physical activity under CPAP therapy, despite improvement in daytime sleepiness. There is no established method to best identify physical activity in patients with OSA, obesity, or both. A limited agreement has been found between self-reported questionnaires on physical activity and objective activity measurement with wrist actigraphy. It would have been very difficult to objectively measure physical activity in the large sample of our multisite study. Further studies are required to evaluate the contribution of physical activity on dyslipidaemia in patients with OSA.

As mentioned by Dr Balta and colleagues, there is growing evidence in support of an independent association between OSA and cardiovascular diseases. Therefore, we entered into our multivariate regression analyses most cardiovascular diseases associated with OSA, including hypertension, ischemic heart disease, cardiac arrhythmia, congestive heart failure, and stroke. Recently, a high prevalence of OSA was observed in patients needing surgery for peripheral arterial disease. We acknowledge that a medical history of peripheral arterial disease has not been recorded in our database. Screening for pulmonary hypertension requires additional investigation, such as echocardiography. In isolation, OSA typically causes only mild pulmonary hypertension that does not require specific treatment. We acknowledge that renal, hepatic, and thyroid dysfunction may be observed in patients with OSA; however, in our large multisite study, biologic investigations were limited to those recommended by French guidelines for the management of OSA in routine practice, including fasting blood glucose, glycated hemoglobin, and fasting serum lipid levels.

Wojciech Trzepizur, MD
Angers, France
Marc Le Vaillant, PhD
Villejuif, France
Nicole Meslier, MD
Angers, France
Thierry Pigeonne, MD
Olonnes-sur-Mer, France
Philippe Masson, MD
Cholet, France
Marie P. Humeau, MD
Nantes, France
Acya Bizieux-Thaminy, MD
La Roche-sur-Yon, France
François Goupil, MD
Le Mans, France
Sylvaine Chollet, MD
Nantes, France
Pierre H. Duchateau, MD, PhD
Frédéric Gagnadoux, MD, PhD
Angers, France
for the Institut de Recherche en Santé Respiratoire des Pays de la Loire (IRSR) Sleep Cohort Group

Affiliations: From L’Université Nantes Angers le Mans University (Drs Trzepizur, Meslier, Duchateau, and Gagnadoux); Department of Respiratory Diseases (Drs Trzepizur, Meslier, Duchateau, and Gagnadoux); and Department of Endocrinology-Diabetology-Nutrition (Dr Duchateau), Angers University Hospital; INSERM U1063 (Drs Trzepizur, Meslier, and Gagnadoux); Centre de Recherche Médicale et Sanitaire (Dr Le Vaillant), CNRS UMR8211-INSERM U985-EHESS; Department of Respiratory Diseases (Dr Pigeonne), Pôle Santé des Olonnes; Department of Respiratory Diseases (Dr Masson), Cholet Hospital; Department of Respiratory Diseases (Dr Humeau), Nouvelles Cliniques Nantaises; Department of Respiratory Diseases (Dr Bizieux-Thaminy), La Roche-sur-Yon Hospital; Department of Respiratory Diseases (Dr Goupil), Le Mans Hospital; and Department of Respiratory Diseases (Dr Chollet), Nantes University Hospital.

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Correspondence to: Frédéric Gagnadoux, MD, PhD, Université d’Angers, CHU Angers, Département de Pneumologie, 4 rue Larrey, 49033 Angers Cedex, France; e-mail: frgagnadoux@chu-angers.fr © 2013 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians. See online for more details.

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References


Statins Reduce Lung Inflammation by Promoting the Clearance of Particulate Matter From Lung Tissues

To the Editor:

We read with interest the article by Miyata and colleagues (February 2013) in which the authors reported that lovastatin attenuates lung inflammation in a rabbit model by attenuating macrophage and neutrophil recruitment and activation. We outline here why this is such an important finding.

There is growing interest in the potential role of statins as adjunct therapy in COPD. Human and animal studies suggest that statins reduce both pulmonary and systemic inflammation, which is characterized by reductions in IL-8, IL-6, and C-reactive protein. In a published review, we suggested that this immunomodulatory effect is directed by the bronchial epithelium, mediated primarily through the innate immune system (neutrophils and macrophages), and critical to the pathobiology underlying chronic aeropollutant-induced airway disease (eg, COPD) (Fig 1). This might partly explain why corticosteroid treatment, targeting primarily the adaptive immune system, does not significantly improve important clinical outcomes in COPD, such as slowing the progression of disease or reducing mortality. The study by Miyata et al elegantly demonstrates that inhibition of the innate immune system has an important effect on the response of the lungs to chronic aeropollutant exposure and how this may be inhibited through systemic-based therapy.

The importance of the finding that statin therapy effectively inhibits both pulmonary and systemic inflammation goes beyond...
that of COPD. Evidence from a large prospective study of patients with COPD showed that systemic inflammation conferred its greatest risk on susceptibility to lung cancer. A recent population-based study showed that statin therapy reduced mortality for a number of cancers by about 20% to 30%, particularly those linked with smoking and obesity. In absolute terms, we estimate that 42% of cancers by about 20% to 30%, particularly those linked with smoking and obesity.

Figure 1. Proposed relationship among pulmonary inflammation, systemic inflammation, and COPD-related comorbidities. *Statins have been shown to attenuate both pulmonary and systemic inflammation through their effects on the innate immune response and NFκB/STAT3-mediated inflammatory pathways. CRP = C-reactive protein; ETS = environmental tobacco smoke; MΦ = macrophage; NFκB = nuclear factor κB; PMN = polymorphonuclear neutrophil; SAD = small airways disease; STAT3 = signal transducer and activator of transcription 3; TNFα = tumor necrosis factor-α.

Robert P. Young, BMedSci, MBChB, DPhil
Raewyn J. Hopkins, RN, MPH
Auckland, New Zealand

Affiliations: From the School of Biological Science and Faculty of Medicine and Health Science, The University of Auckland.

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Correspondence to: Robert P. Young, BMedSci, MBChB, DPhil, School of Biological Science and Faculty of Medicine and Health Science, The University of Auckland, Private Bag 92019, Auckland, 1142, New Zealand; e-mail: roberty@adhb.govt.nz © 2013 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians. See online for more details. DOI: 10.1378/chest.13-0496

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