In addition, Dr Casoni and colleagues underline that metal particle concentrations are not indicated in the article. We agree that data on metal particle concentrations would be important when discussing potential adverse effects; however, all tested needles were washed with the same volume (2 mL) of distilled water prior to mineral analysis, and the density of particles observed on the grids was, therefore, correlated to the amount of particles released by the needles. However, the metal particle concentration is not the only parameter involved in the potential impact on health. The size of the particles, their chemical composition, shape, and surface; and the fact that they penetrate directly into the node without being phagocytosed by alveolar macrophages (as in the case of inhaled particles) must also be taken into account. The particles observed on light microscopy examination of the samples were 0.64 to 5.33 μm long, but could consist of aggregates of finer particles. On electron microscopy examination, the isolated particles measured 0.2 to 0.5 μm and were classified into fine to ultrafine particles. Fine and ultrafine particles are not only directly toxic but also are known to induce immunologic reactions not necessarily correlated to the quantity of particles. Publications on sarcoidosis have suggested the possible involvement of particles as a cause of the disease, and the potential risk of granulomatous reactions must be considered. In conclusion, although the health consequences of the release of metal particles by one type of dedicated EBUS-TBNA needle is a subject of debate, it seemed important to inform the medical community about this risk in order to discuss potential medical implications and for health administrators to assume their responsibilities.

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REFERENCES


COPD and Lung Cancer Linked at a Molecular Genetic Level

To the Editor:

We read with interest the recent editorial by Strange (December 2010), who suggests that with a greater understanding of COPD may come a greater understanding of lung cancer biology. Here, we review evidence from recently published genetic epidemiologic studies that supports this view by showing COPD and lung cancer are linked at a molecular genetic level.

In 2009, we showed that COPD affects between 50% and 50% of those diagnosed with lung cancer (depending on diagnostic criteria), more than sixfold that seen in smokers with comparable smoking histories randomly recruited from the community. Recent advances in genetic epidemiology have identified genetic loci implicating three genes in the development of COPD: the nicotinic acetylcholine receptor gene (α1α2 subunits) on chromosome 15q25 (CHRNA3/5) (susceptibility effect), the hedgehog interacting protein on chromosome 4q31 (HHIP) (protective effect), and the family with sequence similarity 13 member A gene on chromosome 4q22 (FAM13A) (protective effect). Although these genes were first identified through genome-wide association studies involving thousands of subjects, their dual role in COPD and lung cancer was established in a case-control study wherein these genetic associations were examined in smokers with normal lung function, those with COPD (GOLD [Global Initiative for Chronic Obstructive Lung Disease] 2+ criteria) and those with histology-confirmed primary lung cancer (subgrouped by COPD subphenotype). Using this approach, we found the CHRNA gene, originally associated with an increased risk of lung cancer, was also associated with an increased risk of COPD, and the HHIP and FAM13A genes originally associated with reduced risk of COPD were independently associated with a reduced risk of lung cancer. These findings provide compelling evidence that COPD and lung cancer are directly linked at a molecular genetic level.

Importantly, CHRNA3/5 subunits are expressed throughout the bronchial epithelium, are activated by nicotine, and appear to modulate pulmonary inflammation. Similarly, HHIP is expressed on bronchial epithelium and modulates epithelial repair, smoke-induced epithelial-mesenchymal transition (prenaligmint transformation), and cigarette smoke-induced oncogenic transformation of bronchial epithelial cells. Although little is known of FAM13A function, it is also expressed on the bronchial epithelium and sequence analysis indicates FAM13A has Rho GTPase activating protein activity, suggesting both antiinflammatory and tumor suppressor function. Although these genetic associations and their biologic effects require confirmation in further studies, these findings provide the first evidence that COPD and lung cancer share pathogenic mechanisms that are mediated by bronchial epithelial (airway) responses to cigarette smoke exposure.

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Serious Fun
Adding Summative Simulation-Based Testing to the CHEST Challenge

To the Editor:

In a recent issue of CHEST (January 2011), Khouli et al1 confirmed the power of simulation-based education for assessing trainee performance in a risk-free environment and improving that performance compared with traditional apprenticeship or video training. There was even an association with a clinical outcome, namely, reduced catheter-related blood stream infections, although the patient outcome control groups were historical or in different populations (surgical ICU vs medical ICU).

For educators starting or improving their own simulation-based medical training programs, the right conditions and best practices from the Agency for Healthcare Research and Quality and Best Medical training programs, the right conditions and best practices in different populations (surgical ICU vs medical ICU). Prior to beginning the simulation component of the CHEST Challenge, the facilitator and graders practiced the clinical scenarios and agreed on standardized scoring by consensus. Players were oriented to the capabilities and limitations of the mannequin. Graders utilized valid, behaviorally anchored checklists (core actions were either present or not), although holistic (global) scoring also has value. Two graders were used for interrater reliability, but adding simulation tasks (broader domain sampling) may best improve overall reliability. Generalizability studies, if done, can further assess the sources and magnitude of measurement errors and help with test design. A trained facilitator played the role of the ICU nurse to ensure consistency and respond to participants for all technical limitations (eg, simulator does not sweat or have changes in skin color). As much as possible, player participants had to perform rather than just verbalize interventions. Finally, encounters were recorded on video; although intended for prono-tional value and quality assurance and not necessarily considered better than oral debriefing,6 these recordings can provide learners with valuable feedback and insights.

In a post-simulation anonymous survey (942/7, 98%), 58% of players indicated that they had used simulation equipment at their home institutions (usually for bronchoscopy or advanced cardiac life support training): 42% had participated in a simulation activity of the American College of Chest Physicians, and 29% did so elsewhere. Using a 5-point Likert scale, fellows responded that they were very comfortable with the simulator (mean ± SD, 3.96 ± 0.85; 71% positive vs 4% negative responses). Most enjoyed the testing (3.90 ± 1.06, 67% vs 13%) and believed that it was fair (3.96 ± 1.20, 75% vs 17%), and the majority would like even more added to next year’s CHEST Challenge (3.63 ± 1.35, 54% vs 21%). Some recommended additional exposure to the simulator prior to testing. Identifiable player information would be required to assess criterion-related validity.

Since 2002, CHEST Challenge has offered a fun forum, rewarding fellows for their medical knowledge and professional attitude. With more experience and guided by best evidence, we believe that summative simulation-based testing also will allow us to measure skills both in our game and in our fellowship training programs.

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