shorter-course regimens that include newer oral agents, exclude injectables, and include drugs for which susceptibility is documented or highly likely.

The ATS/CDC/ERS/IDSA guidelines provide guidance for settings in which there is capacity to perform both rapid molecular testing and phenotypic DST, to tailor the regimen based on the drug susceptibility pattern identified, and to manage adverse events caused by drugs, including linezolid and bedaquiline (1, 7, 8). Whereas the newer and more potent drugs provide the opportunity to safely use all-oral regimens, we concur with Drs. Yew and Chang that there is still much more work to do on improving safety and tolerability of MDR-TB treatments as well as the development and scale-up of companion genotypic tools to test and monitor for resistance to our newest agents. ■

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Cardiovascular and Chronic Obstructive Pulmonary Disease Therapeutics: Two Paths, One Destination?

To the Editor:

Crim and colleagues tested whether vascular stiffness was affected by inhaled long-acting β_2-agonist, corticosteroid, or combination therapy in patients with moderate chronic obstructive pulmonary disease (COPD) (1). Baseline arterial pulse wave velocity predicted mortality but was unaffected by therapy (1). The authors conclude that inhaled therapy for COPD appeared unlikely to reduce cardiovascular (CV) risk (1). Although aggressive risk factor modification and smoking cessation are pivotal in addressing CV risk in COPD, optimal use of existing drugs and potential avenues for developing novel therapies deserve further discussion.

For the management of hypertension in COPD, limited contemporary data support the use of angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and thiazides (2). In an analysis of clinical trials involving more than 12,000 patients with COPD, treatment with roflumilast, a phosphodiesterase-4 inhibitor with a wide range of antiinflammatory actions, was associated with a 35% relative risk reduction in major adverse CV events (nonfatal myocardial infarction, nonfatal stroke, and CV death) independent of age, sex, smoking status, and concomitant COPD treatments (3). These findings warrant further investigation of the potential CV benefits of roflumilast (3). Endothelial activation earlier in life has been associated with the development

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of subclinical heart failure with preserved ejection fraction (4). In the Multi-Ethnic Study of Atherosclerosis Lung Study, an increase in endothelial intercellular adhesion molecule-1 was associated with an accelerated increase in percent emphysema (5). Novel antagonists of cell adhesion molecules have been tested in clinical trials in COPD (6). Could inhibition of endothelial activation yield benefits in COPD and mitigate progression to heart failure with preserved ejection fraction?

Data regarding CV benefits of COPD therapies are scarce, and Crim and colleagues are to be congratulated for adding to the body of evidence. However, the quest for mutually beneficial therapies for COPD and CV diseases must push on. Identifying suitable targets of inflammation and improved disease phenotyping will facilitate structured investigation. ■

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Erratum: Cardiopulmonary Exercise Testing Provides Additional Prognostic Information in Cystic Fibrosis

There is an error in the article by Hebestreit and colleagues, published in the April 15, 2019, issue of the *Journal*. The name of one of the authors, Dr. Liubou Varanitskaya, was inadvertently misspelled as Liobou Varanistkaya in the author line, owing to a typesetting problem. ■

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