Introduction COPD is a risk factor for cardiovascular comorbidities. Elastin degradation represents a shared mechanism for the pulmonary and vascular features.

Methods and Results Plasma desmosine (pDES), a marker of elastin degradation, was measured in 953 COPD patients (609 male, age 63.1 ± 7.2 years, FEV1 50.6 ± 15.1%predicted) by an isotope dilution LC-MS/MS method. Coronary artery calcification (CACS), a surrogate of atherosclerosis, was assessed in 440 standard CT scan images (low 1000 AU).

Results pDES was elevated in patients with cardiovascular comorbidities (p < 0.01) and correlated with FEV1 (r = 0.39, p < 0.0001), MMRC (r = 0.16, p < 0.0001), 6MWD (r =-0.16, p < 0.0001), BODE index (r = 0.10, p < 0.005), fibrinogen, IL6, IL8, CCL18, and SPD but not with emphysema. These variables showed significant higher values in the patients in the highest pDES quartile. pDES was elevated in patients with very high CACS in comparison with patients with lower CACS (Figure 1) and in patients that died during a 3 year follow-up (p < 0.0001).

Conclusion pDES relates to lung function, systemic inflammation, cardiovascular comorbidities, and CACS in patients with COPD. pDES is a predictor of all cause overall mortality.

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How does clinical respiratory physiology help the clinician?