Cachectic COPD patients exhibit apoptotic nuclei and signs of injury in their peripheral muscles. These findings are associated with maintenance or even a rise in the number of satellite cells but a blunted myogenesis. This abnormality, however, is absent when muscle precursor cells of the same patients are cultured \textit{ex vivo}. We hypothesized that systemic signals present in COPD provide an inappropriate environment to satellite cells, affecting myogenesis efficiency.

**OBJECTIVE**

To analyze the function of muscle precursor cells when reproducing \textit{in vitro} some of the factors present in COPD.

**METHODS**

Muscle progenitor cells obtained from the vastus lateralis of control subjects and COPD patients were cultured in proliferation medium supplemented with human serum collected from healthy subjects, stable COPD patients (either cachectic or noncachectic) and exacerbated COPD patients. Cell proliferation was assessed through BrdU incorporation assays while cell differentiation was evaluated through myotube formation and expression profile of differentiation-specific genes.

**RESULTS**

Serum from healthy subjects and stable COPD patients had similar effects on myoblast proliferation and fusion to build myotubes, as well as on the expression of both myogenic regulatory factors and myosin heavy chains. By contrast, serum from exacerbated COPD patients increased proliferation of muscle progenitor cells but significantly blunted their fusion and differentiation.

**CONCLUSIONS**

Humoral factors present in serum from exacerbated COPD patients may contribute to deteriorate the satellite cell microenvironment blocking myogenesis. Identification of factors that negatively influence muscle regeneration may improve therapeutic strategies on cachectic COPD patients.