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# [ATS] Inflammatory Markers In Different COPD Subgroups Compared To Smokers And Healthy Controls

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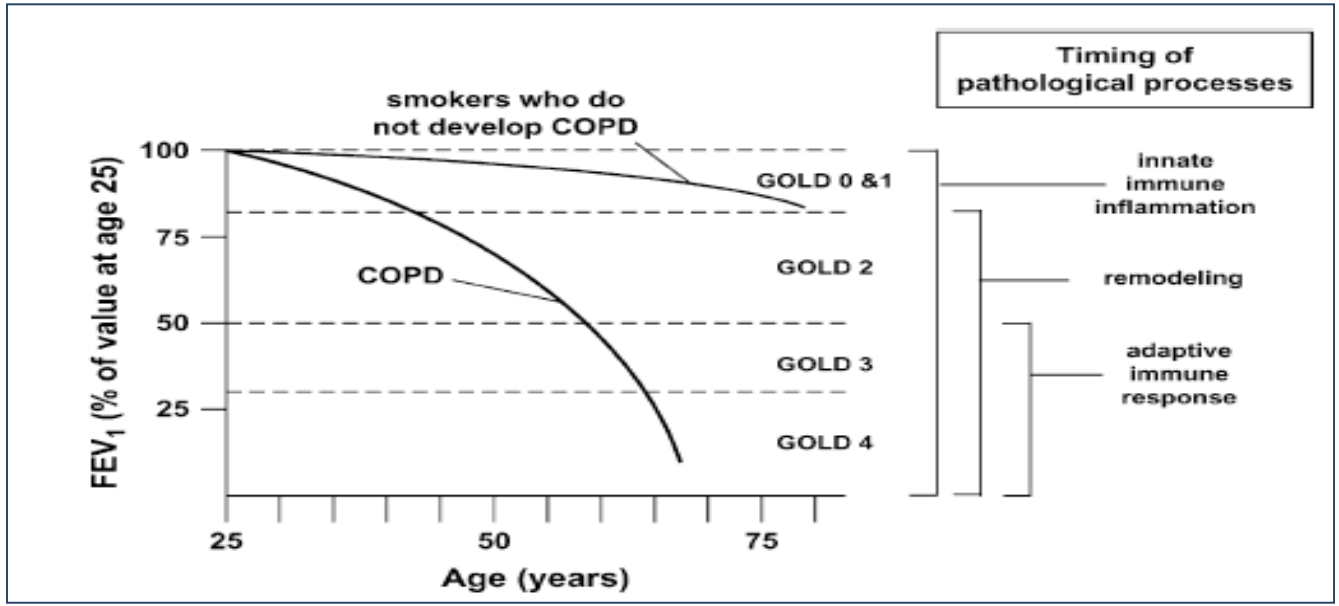
# The Immunopathogenesis of Chronic Obstructive Pulmonary Disease

## Insights from Recent Research




Curtis et al: Proc Am Thorac Soc 2007; 4:512-521

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# Inflammatory Markers in Different COPD Subgroups Compared to Smokers and Healthy Controls

The chronic airway inflammatory response in COPD is suggested to be a result of both innate and adaptive immune responses.
Natural killer cells (NK) and CD56-expressing T cells are cytotoxic lymphocytes that have been implicated in COPD pathogenesis. Natural Killer cell group 2 D (NKG2D) receptors have also been shown to be involved in emphysema development.

We **hypothesised** that cytotoxic T cells would be associated to the rate of lung function decline in subjects with COPD. Two groups of COPD subjects were recruited, rapid decliners (loss of $FEV_1$ of > 60 ml/year) and slow decliners (loss of $FEV_1$ of < 25 ml/year).

Am J RespirCrit Care Med 191;2015:A2884

## METHODS

As controls, we included two groups; smokers with normal lung function and healthy never smoking individuals. All subjects were recruited from the OLIN-cohort (Obstructive Lung disease In Northern Sweden) that has been followed with spirometry annually for a period of more than 10 years.

Bronchoscopy with bronchoalveolar lavage was performed. T cell subsets were determined in BAL-fluid from **17 individuals with COPD stage II-III (12 rapid decliner and 5 slow decliners)**, and compared to data from **15 smokers with normal lung function and 14 healthy never-smokers**. The cells were stained with monoclonal antibodies against CD3, CD8, CD16, CD56 and NKG2D, and analyzed using flow cytometry.

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## RESULTS

In BAL-fluid **NK-cells (CD3-/CD16+/CD56+) were higher in COPD subjects compared to healthy** (p=0.013). The expression of **CD 16+/CD56+ on CD3+lymphocytes was increased** both in subjects with COPD with rapid decline in lung function (p<0.001), in COPD with slow decline (p=0.0082) as well as in smokers with normal lung function (p=0.0065**), all compared to healthy non-smokers**. Furthermore, the **activating receptor NKG2D on CD8 positive lymphocytes was found to be up-regulated in rapid decliners (p=0.0078) compared to healthy, but not in slow decliners and in smokers.**

## CONCLUSIONS

**NK-cells and CD56-expressing T cells were increased in COPD subjects regardless of rapid or slow lung function decline**. These data do not support the hypothesis that cytotoxic T cells can be predictive for the rate of lung function decline in COPD. **The increase in NKG2D in rapid decliners may indicate that these cells can play a role in lung function decline**. Further research is warranted to confirm this finding.