

Eosinophilic Chronic Obstructive Pulmonary Disease is Not Associated with Helminth Infection or Exposure

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Letter to the Editor

A sputum eosinophilia (>3%) is present in 10-40% of COPD subjects [1,2]. The underlying cause is poorly understood. We hypothesized that eosinophilic airway inflammation in COPD is associated with previous exposure to parasites. To test our hypothesis we analyzed serum samples from 150 COPD subjects that had participated in a longitudinal observational study [3,4]. Serum samples were analyzed in the Parasitology Reference Laboratory at the Hospital for Tropical Diseases, London, to test for antibodies to: *Strongyloides stercoralis*, cysticercosis (*Taenia solium*), *Toxocara canis* and *Echinococcus granulosus* [5,6].

All of the patients were white British. The sputum eosinophil count was assessed 3 monthly for 1 year at visits when subjects were exacerbation-free for at least 6 weeks. The sputum eosinophil count was expressed as the area under the curve/year. An extensive travel history was obtained retrospectively in those subjects with positive serology.

There were 16 positive results in 14 patients. None of the subjects had symptoms suggestive of active parasitic infection. There were 11 positive results for *Strongyloides stercoralis*, 2 for *Echinococcus granulosus*, 1 for *Taenia solium* and 2 for *Toxocara canis*. Among the 11 cases who were positive for antibodies to *Strongyloides stercoralis*; 6 had travelled abroad to South-East Asia (n=3), Tunisia (n=3), and among these 6, one travelled also to Israel and Syria and one to South Africa. Two had never travelled outside Europe and 2 were lost to follow-up and 1 had died.

Of the two subjects with positive *Toxocara canis* serology; 1 had traveled to South-East Asia, Australia, New Zealand, Israel and Syria and the other had died. Of the two subjects positive for antibodies to *Echinococcus granulosus*; 1 lived for 3 years in Egypt during childhood and the other travelled to South-East Asia and South Africa.

The patient with positive cysticercosis serology had travelled to Vietnam and South Africa. Among the eosinophilic COPD subjects 3/28 had positive serology for parasites, whilst 11/122 of non-eosinophilic patients were seropositive. There were no significant differences in clinical or sputum characteristics between those with positive and negative serum results (Table 1).

We report here for the first time helminth serology in a group of subjects with COPD. The proportion of COPD subjects with positive parasite serology was 9%, which was associated in most cases with a relevant travel history to endemic areas. The proportion of positive serum parasites is similar between eosinophilic and non-eosinophilic

COPD subjects (3/28 [10.7 %], versus 11/122 [9%], p=1.0 [Fisher's Exact test]).

	Parasite serology negative (n= 136)	Parasite serology positive (n= 14)	p value
Male, n (%)	94 (69)	11 (79)	0.46
Age (years) *	69 (0.8)	64.5 (3)	0.24
Body mass index (kg/m ²)	26 (0.4)	27 (1.7)	0.73
Pack years smoked	46 (30-60)	39 (28-79)	0.89
Exacerbations in last year	3 (1-5)	2 (0-7.5)	0.68
ICS (BDP equivalent) (mcg/day)	2000 (900-2000)	2000 (800-2000)	0.86
FEV1% predicted, (%)	49 (1.7)	45.7 (4.8)	0.69
FEV1/FVC (%) *	49 (1.1)	50 (3.2)	0.80
Reversibility, %	3.7 (-1.3- 10)	0.69 (-6.2- 6.7)	0.10
Sputum TCCx106/g	3.6 (1.5-7.9)	1.8 (0.75-4.5)	0.06
Sputum neutrophils (%)	73 (53-89)	60 (37-77)	0.11
Sputum eosinophils (%)	1.0 (0.25-3)	0.75 (0.44-2.1)	0.89
AUC sputum eosinophils %/year	0.79 (0.27-1.1)	0.41 (0.25-2.2)	0.30
PB. eosinophils (x109/L)	0.22 (0.13 - 0.34)	0.17 (0.09 - 0.29)	0.22
MRC Dyspnoea Scale	3 (2-4)	3 (2-3)	0.44
SGRQ Total Score*	51 (1.6)	56 (4.4)	0.34
CRQ total score*	16 (0.41)	17 (0.95)	0.47
VAS Cough (mm)	33 (13-54)	30 (13-72)	0.93
VAS Dyspnoea (mm)	46 (23-67)	50 (24-69)	0.79
VAS Sputum Production (mm)	31 (11-55)	37 (6-55)	0.96
VAS Sputum Purulence (mm)	21 (8-49)	26 (10-51)	0.82

Table 1: Baseline Characteristics of parasite serology positive versus negative COPD subjects

Data presented as median (IQR) unless stated, *mean (SEM), **Geometric mean of Log data (95% CI). SEM: standard error of mean, CI: confidence interval.

Abbreviations: AUC: area under the curve; TCC: total cell count; PB: peripheral blood; FEV1: Forced expiratory volume in 1 second; FVC: Forced vital capacity; ICS: inhaled corticosteroids; BDP: Beclomethasone Dipropionate; CFU: colony forming unit; MRC: Medical Research Council; SGRQ: St George's Respiratory Questionnaire; CRQ: Chronic respiratory health Questionnaire; VAS: Visual analogue score.

Critically, our findings do not support a role of helminth exposure or infection in eosinophilic COPD. Nevertheless, as for any patient due to receive corticosteroid or immunosuppressive therapy, *Strongyloides* infection must be sought in those with a relevant travel history and treated if found, to remove the risk of developing *Strongyloides* hyperinfection.

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References

1. Eltboli O, Brightling CE (2013) Eosinophils as diagnostic tools in chronic lung disease. *Expert Rev Respir Med* 7: 33-42.
2. Brightling CE, Monteiro W, Ward R, Parker D, Morgan MD, et al. (2000) Sputum eosinophilia and short-term response to prednisolone in chronic obstructive pulmonary disease: a randomised controlled trial. *Lancet* 356: 1480-1485.
3. Bafadhel M, McKenna S, Terry S, Mistry V, Reid C, et al. (2011) Acute exacerbations of chronic obstructive pulmonary disease: identification of biologic clusters and their biomarkers. *Am J Respir Crit Care Med* 184: 662-671.
4. Bafadhel M, McKenna S, Terry S, Mistry V, Pancholi M, et al. (2012) Blood eosinophils to direct corticosteroid treatment of exacerbations of chronic obstructive pulmonary disease: a randomized placebo-controlled trial. *Am J Respir Crit Care Med* 186: 48-55.
5. Kunst H, Mack D, Kon OM, Banerjee AK, Chiodini P, et al. (2011) Parasitic infections of the lung: a guide for the respiratory physician. *Thorax* 66: 528-536.
6. Collier S, Manser M, Chiodini PL (2010) External quality assessment scheme for parasite serology; a review of the scheme design and performance. *J Clin Pathol* 63: 441-444.