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Tobacco Is Not the Only Risk Factor for COPD: Let's Not Forget the Genetic Factor!!!

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Short communication

Chronic obstructive pulmonary disease (COPD) is a major health problem as it causes increasing mortality and morbidity in all countries of the world. Its pathogenetic aspect is characterized by progressive irreversible airflow limitation. However, the disease is also a chronic inflammatory disease of the bronchopulmonary tissue with systemic consequences [1].

Cigarette smoking is the major risk factor implicated in the genesis of COPD. It is the most studied risk factor in the literature, however only a minority of cigarette smokers develops symptomatic disease. Indeed, only 10%–20% of all heavy cigarette smokers develop COPD [1]. Studies of families and twins suggest that genetic factors also contribute to the development of COPD. These findings suggest the presence of other risk factors implicated in the development of COPD and which are poorly studied as the genetic factor and its role in the genesis of COPD [1,2].

We report here the case of homozygous twin brothers, aged 54, with no notable pathological history, both consulting for respiratory symptoms.

The attached table 1 reports their clinical and spirometric data.

It should be noted that the smoking twin is more symptomatic (cough and expectoration) but the dyspnea is more severe in twin who is a teacher than twin 1 who is a taxi driver. The twins have the same grade of spirometric ventilator defect (severe obstructive ventilator defect (OVD)). Thus, the genetic factor in the pathophysiology of COPD should not be overlooked since it can lead to bronchial obstruction as severe as that obtained by smoking.

Although one is a smoker and the other twin is non-smoker, they both developed COPD confirmed by spirometry and bronchodilation test with the same grade of severity GOLD III. wilk et al. [3] highlighted the important role for the *CHRNA5/3* region as a genetic risk factor for airflow obstruction that may be independent of smoking and implicate the *HTR4* gene in the etiology of airflow obstruction [3]. Sandford et al. [4] have shown that variants in genes involved in xenobiotic metabolism, antioxidation, and the inflammatory response have been associated with COPD.

Thus all the elements of the pathophysiology of COPD (clinical signs, bronchial and systemic inflammation, bronchial obstruction, severity of COPD and response to treatment) can be encoded by specific genes for each element.

Table 1: Clinical and spirometric data of the homozygous twin brothers

	Twin **1**	Twin **2**
Profession	Taxi driver	Teacher
BMI (Kg/m2)	25	26
Active smoking (Pack year)	51	0
Clinical signs	Cough+sputum+dyspnea	Dyspnea
Detection (year)	1	2
Spirometric ventilator defect	Severe OVD	Severe OVD
COPD	COPD GOLD III	COPD GOLD III

BMI: body mass index

OVD: obstructive ventilator defect

COPD: chronic obstructive pulmonary disease

GOLD: Global initiative for obstructive lung disease

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