



SMOKING AND OCCUPATIONAL OBSTRUCTIVE LUNG DISEASE(OCC-ACO)

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- ❑ There are approximately 600 ingredients in cigarettes.
- ❑ When burned, cigarettes create more than 7,000 chemicals.
- ❑ At least 69 of these chemicals are known to cause cancer, and many are toxic.

- ❑ Cigarette **smoking** is the leading preventable cause of COPD.
- ❑ Efforts to prevent COPD focus on limiting exposure to smoking.
- ❑ Occupational exposures are important causes of COPD
 - ❑ 14% of all cases
 - ❑ 31% of cases among never smokers.

Other risk factors for COPD include:

- Exposure to air pollution
- Breathing secondhand smoke
- Working with chemicals, dust and fumes
- A history of childhood respiratory infection

- Tobacco **smoking** is the main leading cause of COPD in more than 90% of the cases
- (COPD) is the third leading cause of death worldwide
- It has always been considered a disease of the elderly, since it is generally diagnosed over the age of 45, after several years of **smoking**

Workplace exposures

□ Associated with COPD :

Dusts, gases, minerals, metals, grain, wood, cotton, vapors, welding fumes, and isocyanates

□ Associated with new-onset and work-exacerbated asthma :
workplace exposures, such as irritant chemicals, dusts, **secondhand smoke**, allergens, cleaners, disinfectants, welding fumes, isocyanates

Exposure to occupational risk factors in COPD patients FR

Types	Subtypes	Classification
Dust	Organic dust	Plant dust (grain dust, cotton dust, wood dust, paper dust, etc.). Animal dust (mixed animal dust, fur, etc.)
	Inorganic dust	Contains carbon dust (coal dust, carbon black, etc.)
		Artificial inorganic dust (cement, artificial mineral wool, artificial inorganic mixed dust, etc.)
		Mineral dust (silica dust, asbestos, talc, etc.)
		Metallic dust (metal mixed dust, iron, aluminum, copper, lead, etc.)
	Mixed dust	N/A
Irritating chemicals	Acids and acid-forming compounds	Hydrochloric acid, sulfur dioxide, sulfuric acid, nitric acid, hydrogen fluoride, etc
	Aldehydes	Formaldehyde, acetaldehyde, etc
	Nitrogen oxides	Nitric oxide, nitrogen dioxide, etc
	Metal compound smog	Copper oxide, iron oxide, manganese dioxide, copper hydroxide, iron hydroxide, etc
	Organic solvents	Benzene, toluene, xylene, vinyl chloride pyrethroid, etc.
	Chlorine and its compounds	Chlorine, hydrogen chloride, etc
	Ammonia	Ammonia, etc.

□ Asthma–COPD overlap (ACO):

- A separate phenotype of obstructive airway diseases
- included in several guidelines of asthma and COPD

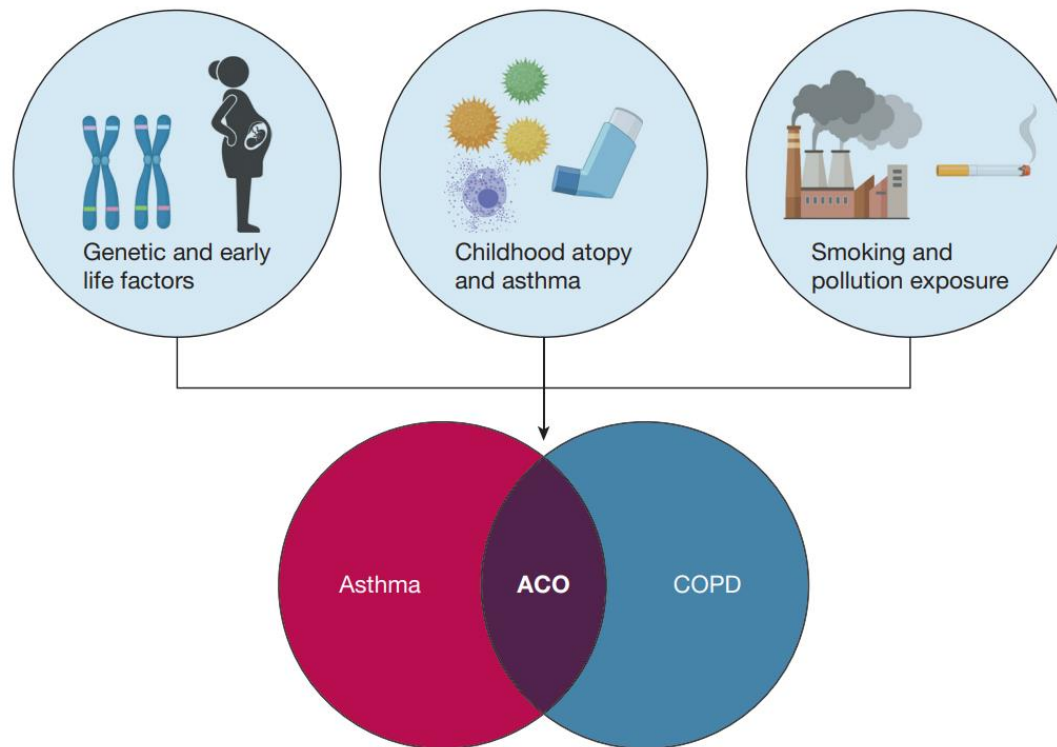


Figure 2 – Diagram showing major contributors in the development of ACO. A plausible explanation for ACO occurs in an individual with particular genetic and early life factors and a history of childhood atopy and asthma who subsequently is exposed to chronic smoking and indoor or outdoor pollution. Chronic airflow limitation can develop in this individual, leading to the clinical presentation of ACO. ACO = asthma-COPD overlap; COPD=chronic obstructive pulmonary disease.

- ❑ In 2015, the Global Initiative for Asthma (GINA) and the Global Initiative for COPD (GOLD) released a joint statement describing what was then called ACOS
- ❑ As "persistent airflow limitation with
- ❑ several features usually associated with asthma
- ❑ and several features usually associated with COPD

- ❑ In 2017, the American Thoracic Society and the National Heart, Lung, and Blood Institute published a joint workshop report on ACO
- ❑ ACO be used to describe :
 - ❑ long-standing history of asthma
 - ❑ Having a modest smoking history
 - ❑ And fixed airflow obstruction.
- ❑ At the same time, patients with COPD who have features of asthma, such as bronchodilator responsiveness and peripheral eosinophilia, could also be described as having ACO.
- ❑ While clinically, and likely pathologically, these patients remain distinct, both fall under the ACO umbrella.

Prevalence of COPD among workers with work-related asthma

- ❑ Never-smokers with WRA were twice as likely to have COPD as adults with non-WRA
- ❑ Suggesting that some occupational exposures may place individuals at greater risk for asthma-COPD overlap.
- ❑ Although cigarette smoking is the primary cause of COPD
- ❑ As much as 15% (range 0%–37%) of COPD can be attributable to workplace exposures (OCC-COPD)

□ workplace exposures are causative agents for both WRA and COPD.

□ In adult-onset asthma, occupational exposure alone is not driving the risk of ACO but rather the combination of occupational exposure and **smoking**.

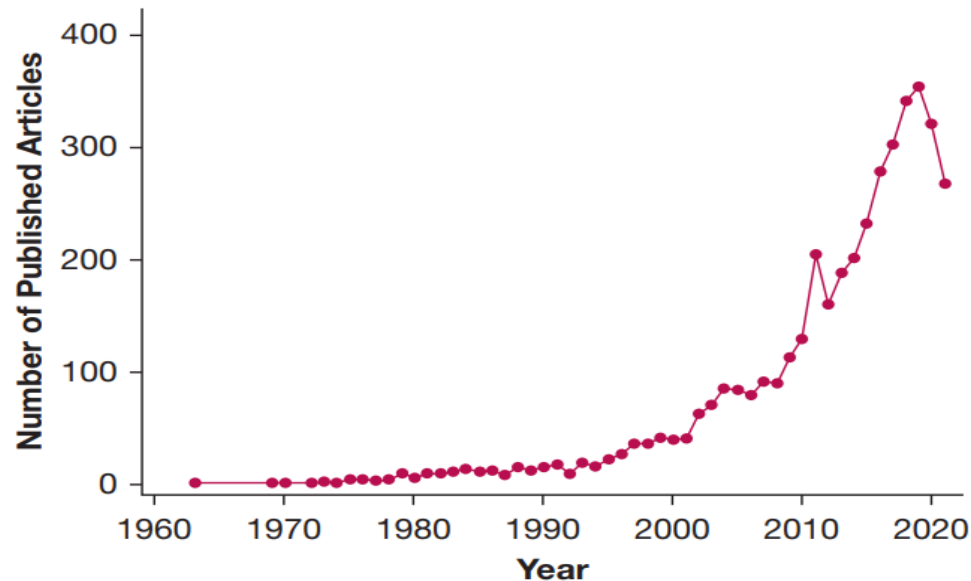


Figure 1 – Graph showing the number of published articles by year on asthma-COPD overlap (ACO) based on a PubMed search. The Dutch hypothesis was introduced in 1961.¹ The Global Initiative for Asthma and Global Initiative for Chronic Obstructive Lung Disease joint statement that recognized ACO as a condition was published in 2014.⁸ Publications on this topic accumulated slowly from 1960 through 2010, followed by a rapid increase from 2010 through 2020.

Definitions



Asthma

Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation. [GINA 2014]

COPD

COPD is a common preventable and treatable disease, characterized by persistent airflow limitation that is usually progressive and associated with enhanced chronic inflammatory responses in the airways and the lungs to noxious particles or gases. Exacerbations and comorbidities contribute to the overall severity in individual patients. [GOLD 2015]

Asthma-COPD overlap (ACO) [a description]

Asthma-COPD overlap (ACO) is characterized by persistent airflow limitation with several features usually associated with asthma and several features usually associated with COPD. ACO is therefore identified by the features that it shares with both asthma and COPD.

Usual features of asthma, COPD and ACOS



Feature	Asthma	COPD	ACOS
Age of onset	Usually childhood but can commence at any age	Usually >40 years	Usually ≥ 40 years, but may have had symptoms as child/early adult
Pattern of respiratory symptoms	Symptoms vary over time (day to day, or over longer period), often limiting activity. Often triggered by exercise, emotions including laughter, dust, or exposure to allergens	Chronic usually continuous symptoms, particularly during exercise, with 'better' and 'worse' days	Respiratory symptoms including exertional dyspnea are persistent, but variability may be prominent
Lung function	Current and/or historical variable airflow limitation, e.g. BD reversibility, AHR	FEV ₁ may be improved by therapy, but post-BD FEV ₁ /FVC <0.7 persists	Airflow limitation not fully reversible, but often with current or historical variability
Lung function between symptoms	May be normal	Persistent airflow limitation	Persistent airflow limitation

Box 1. Current definitions of asthma and COPD, and clinical description of asthma-COPD overlap

Asthma

Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation. [GINA 2017]²²

COPD

Chronic obstructive pulmonary disease (COPD) is a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases. [GOLD 2017]²³

Asthma-COPD overlap – not a definition, but a description for clinical use

Asthma-COPD overlap is characterized by persistent airflow limitation with several features usually associated with asthma and several features usually associated with COPD. Asthma-COPD overlap is therefore identified in clinical practice by the features that it shares with both asthma and COPD.

This is not a definition, but a description for clinical use, as asthma-COPD overlap includes several different clinical phenotypes and there are likely to be several different underlying mechanisms.

The BD response of both FEV_1/FVC and FEV_1 to salbutamol (400 mcg) inhalation was defined as:

- a “responder” was a post-BD $FEV_1/FVC \geq LLN$ or an increase of $FEV_1 \geq 200$ mL and $\geq 12\%$. Among them:
 - a “full responder” was a “responder” with a post BD $FEV_1 > 80\%$ PV.
 - an “incomplete responder” was a “responder” with a post BD $FEV_1 < 80\%$ PV.
- an “irreversible” was a patient with a post BD response inducing a $FEV_1/FVC \leq LLN$ and no significant increase of FEV_1 .

The “incomplete responder” and “irreversible” groups were subdivided into cumulative smoking of \geq or <10 packs of per year.

The same phenotypes were also defined with the 2021 ATS/ERS definition of bronchial reversibility (i.e., a $>10\%$ change of FEV_1 predicted value) instead of an increase of $FEV_1 \geq 200$ mL and $\geq 12\%$.

Chronic respiratory symptoms* with FEV1/FVC < LLN				
Reversibility			Irreversibility	
Full	Incomplete			
ASTHMA (GINA treatment)	Non smokers	Smokers	Non smokers	Smokers
	ACO			
		DLC0 ≥ 80 %	DLC0 < 80 %	
			COPD (GOLD treatment)	

Figure 4. The strategy to select GINA or GOLD treatment among Vietnamese patients with chronic obstructive respiratory diseases based on symptoms, spirometry with response to BD, cumulative smoking and DLCO measurement among ACOs. GINA: Global Initiative for Asthma. GOLD: Global Initiative for Chronic Obstructive Lung Disease guidelines. COPD: chronic obstructive pulmonary disease. ACO: asthma—COPD overlap. * chronic respiratory symptoms: cough and/or sputum and/or dyspnea and/or wheezing and/or chest tightness lasting > 3 months.



- ❑ The pooled prevalence of ACO was
- ❑ 2.0% (95% CI: 1.4–2.6%) in the general population,
- ❑ 26.5% (95% CI: 19.5–33.6%) among patients with asthma,
- ❑ 29.6% (95% CI: 19.3–39.9%) among patients with COPD.
- ❑ The global prevalence of asthma-only was 6.2% (95% CI: 5.0–7.4%) and COPD-only was 4.9% (95% CI: 4.3–5.5%).

- Global prevalence of asthma-COPD overlap (ACO) in the general population: a systematic review and meta-analysis

- The prevalence of ACO was higher in the group with occupational VGDF exposure, compared to patients with no exposure.
- Patients with occupational VGDF exposure were older and more often males.

- ❑ The additive effect of smoking and other environmental exposures in the development of COPD
- ❑ Occupational VGDF exposure was independently associated with ACO among patients with ≥ 10 pack-years of smoking

- ❑ Obesity seemed to lower the risk of ACO.
- ❑ Reduction of FVC in obese patients, leading to decreased sensitivity of FEV1/FVC ratio in detecting obstruction

TABLE 1 Clinical characteristics and prevalence of asthma–COPD overlap in groups of different occupational exposure to vapours, gases, dusts or fumes (VGDF)

	No occupational exposure to VGDF	Occupational exposure to VGDF	p-value
Patients n	150	44	
Asthma–COPD overlap	21 (14)	13 (30)	0.024 [¶]
Age years	57.3±13.3	62.3±13.9	0.030 [¶]
BMI kg·m⁻²	27.7 (24.2–30.8)	29.6 (25.4–32.0)	0.057
Males	50 (33)	31 (71)	<0.001 [¶]
Allergic rhinitis or conjunctivitis	90 (62)	28 (65)	0.723
ICS in daily use	120 (80)	33 (75)	0.530
Smoking history ≥10 pack-years	47 (31.3)	19 (43.2)	0.152
Obstructive asthma[#]	15 (10)	5 (11)	0.782

Data are presented as n (%), mean±SD or median (interquartile range), unless otherwise stated. BMI: body mass index; ICS: inhaled corticosteroids. [#]: <10 pack-years and forced expiratory volume in 1 s/forced vital capacity ratio <0.7; [¶]: p<0.05.

□ poorer asthma control in ACO may reflect the influence of chronic airway changes related to the COPD component

- Longitudinal comparison of outcomes in patients with smoking-related asthma-COPD overlap and in non-smoking asthmatics with incomplete reversibility of airway obstruction

- ❑ This is in keeping with the previous suggestions of **the additive** effect of smoking and other environmental exposures in the development of COPD

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