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A Clinical Review on Chronic Obstructive Pulmonary Disease after COVID19 Situations

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Abstract **Review Article**

Chronic obstructive pulmonary disease (COPD) is a prevalent long-lasting airway inflammatory condition that features enduring respiratory symptoms and irreversible airflow restriction. Globally, COPD ranks third in terms of cause of death behind ischemic heart disease and cerebrovascular disease, owing to high morbidity and mortality rates. Respiratory failure (RF), especially hypercapnic RF (HRF), frequently occurs in most COPD patients at severe or end-stage. COPD complicated by RF (COPD+RF) increases the difficulty of treatment, resulting in poor prognosis. Despite the elusive pathogenesis of RF in COPD, aberrant immune activation and systemic inflammation constitute key contributors. The systemic immune-inflammation index (SII) is a comprehensive inflammatory indicator that is computed by peripheral lymphocyte, neutrophil and platelet counts. Neutrophils, platelets and produced cytokines are mostly tied to nonspecific immune responses. In contrast, lymphocytes are primarily linked to immune-related pathways for COPD patients. Overall COPD is one of the major airway diseases which need to be addressed and invent formulation for the treatment of this particular disease.

Keywords: Etiology of COPD, Pathophysiology, Risk factors, Symptoms.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common and treatable disease characterized by progressive airflow limitation and tissue destruction. It is associated with structural lung changes due to chronic inflammation from prolonged exposure to noxious particles or gases most commonly cigarette smoke. Chronic inflammation causes airway narrowing and decreased lung recoil. The disease often presents with symptoms of cough, dyspnoea and sputum production (Christenson *et al.,* 2024). Symptoms can range from being asymptomatic to respiratory failure. COPD is caused by prolonged exposure to harmful particles or gases. Cigarette smoking is the most common cause of COPD worldwide. Other causes may include second-hand smoke, environmental and occupational exposures and alpha-1 antitrypsin deficiency (AATD) (Wang *et al.,* 2021). COPD is primarily present in smokers and those greater than age 40. Prevalence increases with age and it is currently the third most common cause of morbidity and mortality worldwide. In 2015, the prevalence of COPD was 174 million and there were approximately 3.2 million deaths due to COPD worldwide. However, the prevalence is likely to be underestimated due to the under diagnosis.

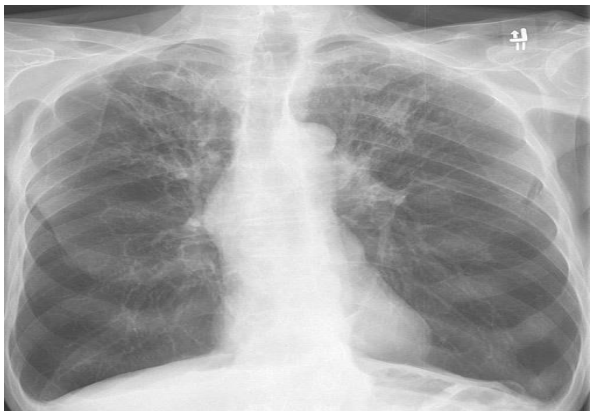

Figure 1: X-ray of Chest, Chronic Obstructive Pulmonary Disease

What Causes COPD?

Over time, exposure to lung irritants like tobacco smoke or chemicals can damage your lungs and airways (Rennard *et al.*, 2015). This long-term exposure can cause chronic obstructive pulmonary disease (COPD), which includes chronic bronchitis and emphysema (Singh *et al.,* 2019). The leading cause of COPD is smoking; however 1 in 4 people living with COPD never smoked.

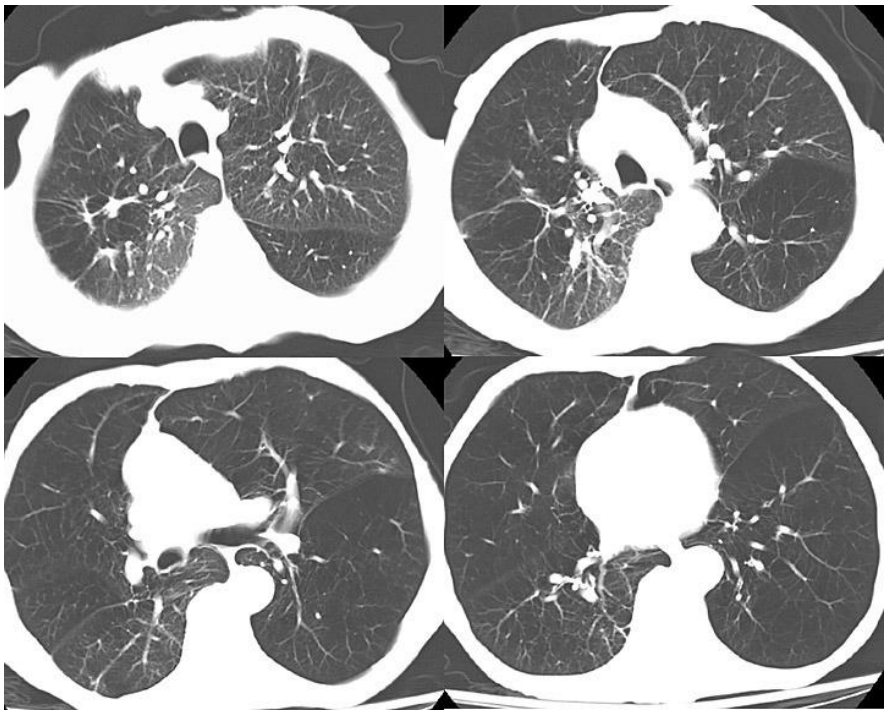

Figure 2: CT scan COPD Left Lung

COPD Risk Factors after COVID 19 Situations

COPD is often referred to as a "smoker's disease" however although smoking is one of the main risk factors for developing COPD, people who never smoke may also develop COPD. Other risk factors may include (GBD *et al.,* 2015):

- A history of childhood respiratory infections.
- Smoke exposure from coal or wood burning stove.
- Exposure to secondhand smoke.
- People with a history of asthma.
- People who have underdeveloped lungs.
- Those who are of age 40 and older as lung function declines as you age.

Reduce Your Risk for COPD

If you are concerned about developing COPD, you can take steps to reduce your risk.

- Quit smoking for good by finding support, programs and resources through the American Lung Association.
- Avoid secondhand smoke exposure.
- Stay up to date with your COVID-19, flu and pneumonia vaccinations. Vaccinations help protect you against respiratory viruses.
- If you are exposed to chemicals, dust and fumes through your workplace, use appropriate protective equipment and attend safety training (Stockley *et al.,* 1999).

COPD is a progressive disease and it can get worse overtime. If you have risk factors for COPD or are experiencing symptoms, do not wait to talk to your healthcare provider. The earlier you are diagnosed with COPD; the sooner treatment can begin.

Pathological Changes Found in COPD

Proximal Cartilaginous Airways (>2 mm in Diameter)

- Increased numbers of macrophages and CD8 T lymphocytes (Parker *et al.,* 2005).
- Few neutrophils and eosinophils (neutrophils increase with progressive disease).
- Submucosal bronchial gland enlargement and goblet cell metaplasia (results in excessive mucous production or chronic bronchitis) (Vestbo *et al.,* 2013).
- Cellular infiltrates (neutrophils and lymphocytes) of bronchial glands.
- Airway epithelial squamous metaplasia, ciliary dysfunction, hypertrophy of smooth muscle and connective tissue (Regan *et al.,* 2011).
- *Peripheral Airways (Non-Cartilaginous Airways <2 mm Diameter)*
- Increased numbers of macrophages and T lymphocytes (CD8 > CD4).

- Increased numbers of B lymphocytes, lymphoid follicles and fibroblasts (Vestbo *et al.,* 2008).
- Few neutrophils or eosinophils.
- Bronchiolitis at an early stage.
- Luminal and inflammatory exudates.
- Pathological extension of goblet cells and squamous metaplasia into peripheral airways.
- Peribronchial fibrosis and airway narrowing with progressive disease.

Lung Parenchyma (Respiratory Bronchioles and Alveoli)

Athogenesis

Inflammation is present in the lungs, particularly the small airways, of all people who smoke. This normal protective response to the inhaled toxins is amplified in COPD, leading to tissue destruction, impairment of the defence mechanisms that limit such destruction and disruption of the repair mechanisms (Couper *et al.,* 2014). In general, the inflammatory and structural changes in the airways increase with disease severity and persist even after smoking cessation (Lamprechet *et al.,* 2015). Besides inflammation, two other processes are involved in the pathogenesis of COPD - an imbalance between proteases and antiproteases and an imbalance between oxidants andantioxidants (oxidative stress) in the lungs.

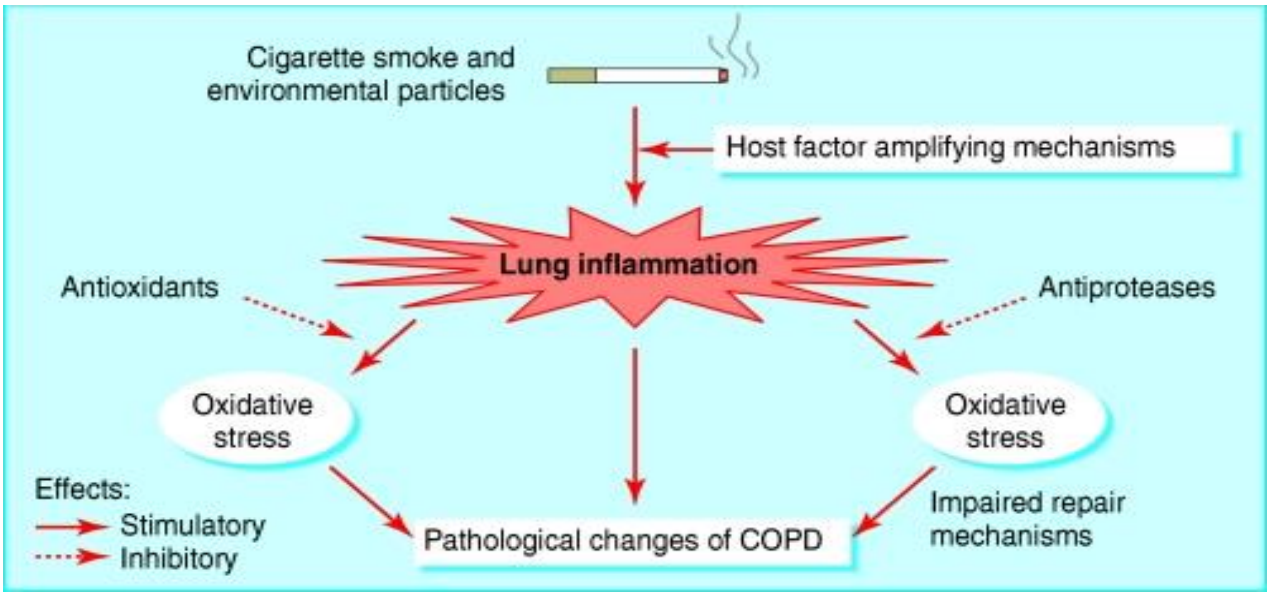

Figure 3: Pathogenesis of COPD

Inflammatory Cells

COPD is characterized by increased numbers of neutrophils, macrophages and T lymphocytes (CD8 more than CD4) in the lungs (Suzuki *et al.,* 2017). In general, the extent of the inflammation is related to the degree of the airflow obstruction (Lovasi *et al*., 2011). These inflammatory cells release a variety of cytokines and mediators that participate in the disease process (Mitchell *et al.,* 1970). This inflammatory pattern is markedly different from that seen in patients with asthma.

Inflammatory Mediators

Many inflammatory mediators are increased in COPD, including

- Leucotriene B4, a neutrophil and T cell chemoattractant which is produced by macrophages, neutrophils and epithelial cells (Sanchez *et al.,* 2014).
- Chemotactic factors such as the CXC chemokines interleukin 8 and growth related oncogene α, which are produced by macrophages and epithelial cells (Takahashi *et al.,* 2008)
- These attract cells from the circulation and amplify pro-inflammatory responses.
- Pro-inflammatory cytokines such as tumour necrosis factor α and interleukins 1β and 6.
- Growth factors such as transforming growth factor β, which may cause fibrosis in the airways either directly or through release of another cytokine, connective tissue growth factor (Verschakelen *et al.,* 2007).

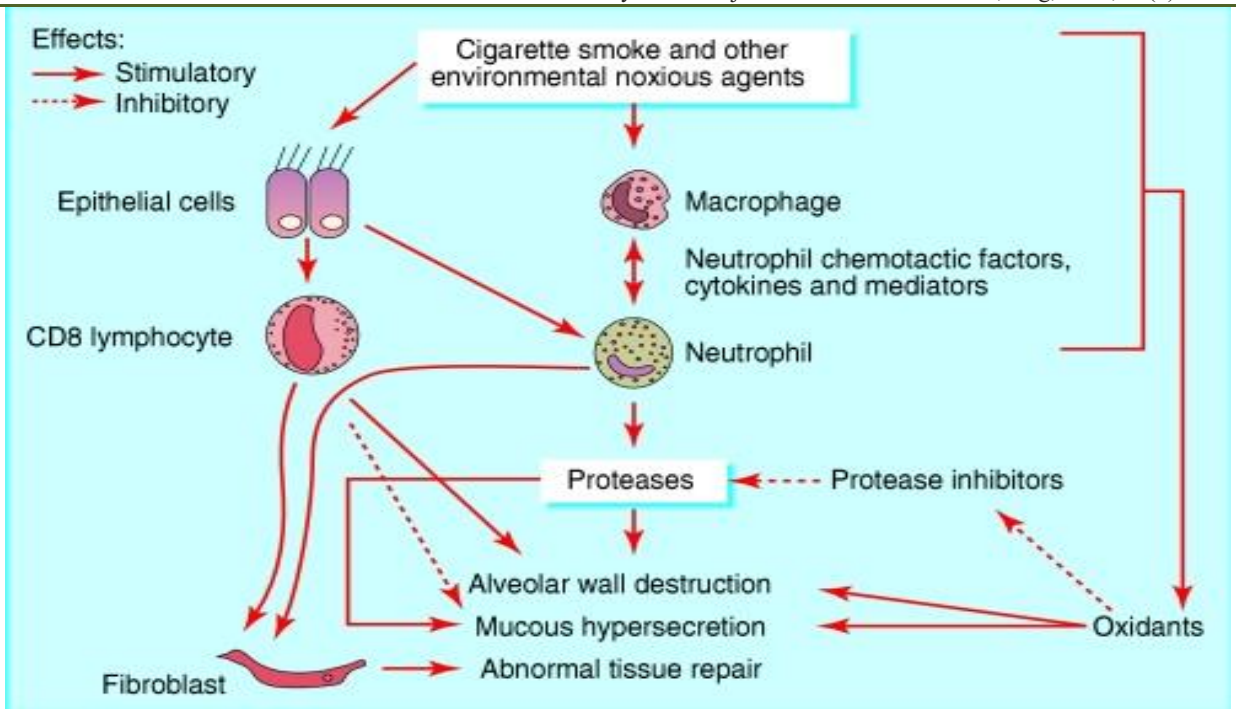

Figure 4: Inflammatory mechanisms in COPD

Inflammatory Cells and Mediators in COPD

Neutrophils, which release proteases, are increased in the sputum and distal airspaces of smokers; a further increase occurs in COPD and is related to disease severity (McDonough *et al.,* 2011).

- Macrophages, which produce inflammatory mediators and proteases, are increased in number in airways, lung parenchyma and in bronchoalveolar lavage fluid (Martinez *et al.,* 2018).
- T lymphocytes (CD4 and CD8 cells) are increased in the airways and lung parenchyma, with an increase in CD8:CD4 ratio (Polverino *et al.,* 2015). Numbers of Th1 and Tc1 cells, which produce interferon γ, also increase. CD8 cells may be cytotoxic and cause alveolar wall destruction (Qian *et al.,* 2023).
- B lymphocytes are increased in the peripheral airways and within lymphoid follicles, possibly as a response to chronic infection of the airways (Pons *et al.,* 2005).

Table 1: Proteases and antiproteases in COPD

Proteases	**Antiproteases**
Serine proteases	α_1 antitrypsin
Neutrophil elastase	
Cathepsin G	Secretory leucoprotease inhibitor
Protease 3	Elafin
Cysteine proteases	Cystatins
Cathepsins B, K, L, S	
Matrix metalloproteases (MMP-8, MMP-9, MMP-12)	Tissue inhibitors of MMP (TIMP1-4)

Systemic Features of COPD

- Cachexia.
- Skeletal muscle wasting and disuse atrophy.
- Increased risk of cardiovascular disease (associated with increased concentrations of C reactive protein) (Allinson *et al.,* 2017*)*.
- Normochromic normocytic anaemia (Marin *et al.,* 2013).
- Secondary polycythaemia (Burney *et al.,* 2014).
- Osteoporosis.
- Depression and anxiety.

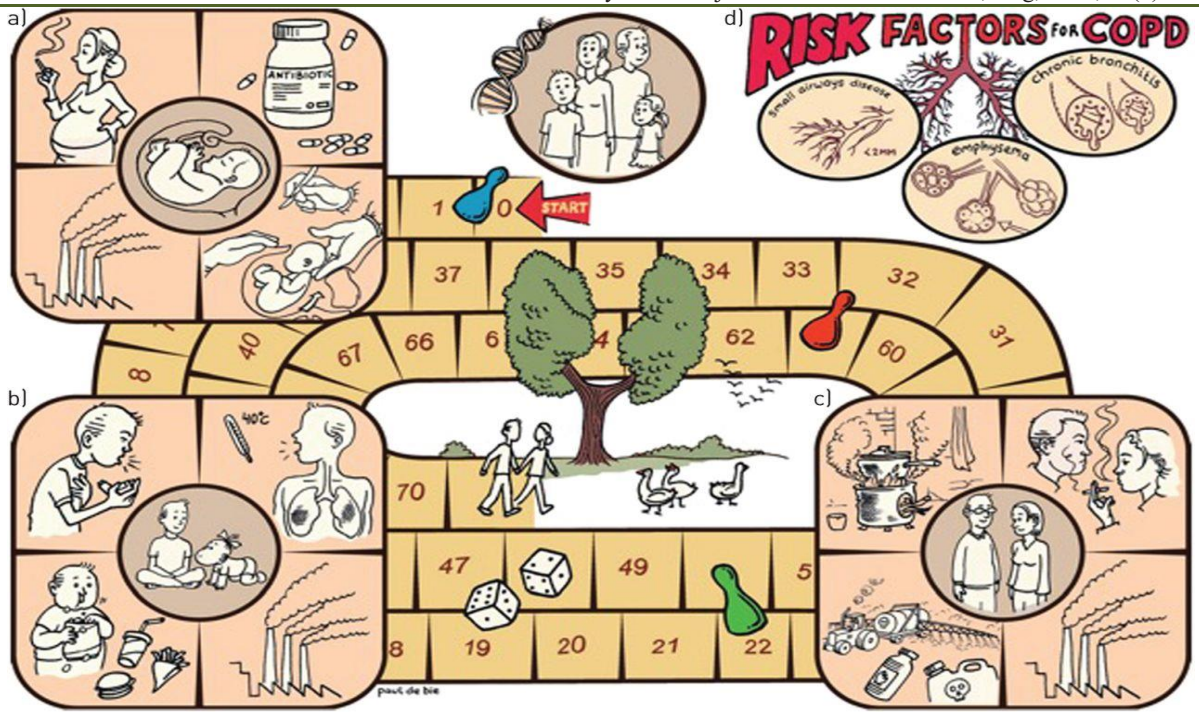

Figure 5: Graphic representation of the risk factors for chronic obstructive pulmonary disease (COPD) during the different stages of life

CONCLUSION

COPD has always been considered a disease of the elderly and little attention has been paid to the clinical and pathologic features of COPD in younger individuals. Current evidence suggests that early COPD is associated with poor clinical outcomes and it makes sense to think that early detection, diagnosis and maintenance treatment of COPD, alongside smoking cessation and exercise. Therefore it is very necessary to do regular health exercise, lung exercise, protect yourself from dust particles and allergens from regular inhalations, after COVID 19 pandemic situations.

REFERENCES

- Allinson, J.P., Hardy, R., Donaldson, G.C., Shaheen, S.O., Kuh, D. and Wedzicha, J.A., 2017. Combined impact of smoking and early-life exposures on adult lung function trajectories. *American journal of respiratory and critical care medicine*, *196*(8), 1021-1030.DOI: https://doi.org/10.1164/rccm.201703-0506OC.
- Burney, P., Jithoo, A., Kato, B., Janson, C., Mannino, D., Nižankowska-Mogilnicka, Studnicka, M., Tan, W., Bateman, E., Koçabas, A. and Vollmer, W.M., 2014. Chronic obstructive pulmonary disease mortality and prevalence: the associations with smoking and poverty— a BOLD analysis. *Thorax*, *69*(5), 465-473. DOI: https://doi.org/10.1136/thoraxjnl-2013-204460.
- Christensen, J., Prosper, A.E., Wu, C.C., Chung, J., Lee, E., Elicker, B., Hunsaker, A.R., Petranovic, M., Sandler, K.L., Stiles, B. and Mazzone, P., 2024. ACR Lung-RADS v2022: assessment categories and management recommendations. *Journal of the American College of Radiology*, *21*(3), 473-488. DOI: https://doi.org/10.1016/j.chest.2023.10.028.
- Couper, D., LaVange, L.M., Han, M., Barr, R.G., Bleecker, E., Hoffman, E.A., Kanner, R., Kleerup, E., Martinez, F.J., Woodruff, P.G. and Rennard, S., 2014. Design of the subpopulations and intermediate outcomes in COPD study (SPIROMICS). *Thorax*, *69*(5), 492-495. DOI: https://doi.org/10.1136/thoraxjnl-2013-203897.
- Anonymus 2015. GBD 2015 Chronic Respiratory Disease Collaborators, 2017. Global, regional, and national deaths, prevalence, disability-adjusted life years, and years lived with disability for chronic obstructive pulmonary disease and asthma, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. *The Lancet. Respiratory Medicine*, *5*(9),691 DOI: https://doi.org/10.1016/S2213-2600(17)30293-X
- Lamprecht, B., Soriano, J.B., Studnicka, M., Kaiser, B., Vanfleteren, L.E., Gnatiuc, L., Burney, P., Miravitlles, M., García-Rio, F., Akbari, K. and Ancochea, J., 2015. Determinants of underdiagnosis of COPD in national and international surveys. *Chest*, *148*(4), 971-985. DOI: https://doi.org/10.1378/chest.14-2535.
- Lovasi, G.S., Roux, A.V.D., Hoffman, E.A., Smith, L.J., Jiang, R., Carr, J.J. and Barr, R.G., 2011. Socioeconomic status is positively associated with percent emphysema on CT scan: the MESA lung study. *Academic radiology*, *18*(2),199-204. DOI: https://doi.org/10.1016/j.acra.2010.10.010.

- Marin, J.M., Alfageme, I., Almagro, P., Casanova, C., Esteban, C., Soler-Cataluña, J.J., De Torres, J.P., Martinez-Camblor, P., Miravitlles, M., Celli, B.R. and Soriano, J.B., 2013. Multicomponent indices to predict survival in COPD: the COCOMICS study. *European Respiratory Journal*, *42*(2), 323-332 DOI: http://dx.doi.org/10.1183/09031936.00121012.
- Martinez, F.J., Han, M.K., Allinson, J.P., Barr, R.G., Boucher, R.C., Calverley, P.M., Celli, B.R., Christenson, S.A., Crystal, R.G., Fagerås, M. and Freeman, C.M., 2018. At the root: defining and halting progression of early chronic obstructive pulmonary disease. *American journal of respiratory and critical care medicine*, *197*(12), 1540-1551. DOI: https://doi.org/10.1164/rccm.201710-2028PP.
- McDonough, J.E., Yuan, R., Suzuki, M., Seyednejad, N., Elliott, W.M., Sanchez, P.G., Wright, A.C., Gefter, W.B., Litzky, L., Coxson, H.O. and Paré, P.D., 2011. Small-airway obstruction and emphysema in chronic obstructive pulmonary disease. *New England Journal of Medicine*, *365*(17), 1567-1575. DOI: https://doi.org/10.1056/nejmoa1106955.
- Mitchell, R.S., Silvers, G.W., Goodman, N., Dart, G. and Maisel, J.C., 1970. Are centrilobular emphysema and panlobular emphysema two different diseases. *Human Pathology*, *1*(3), 433-441. DOI: https://doi.org/10.1155%2F2017%2F3502438.
- Parker, C.M., Voduc, N., Aaron, S.D., Webb, K.A. and O'Donnell, D.E., 2005. Physiological changes during symptom recovery from moderate exacerbations of COPD. *European Respiratory Journal*, *26*(3), 420-428. DOI: http://doi.org/10.1183/09031936.05.00136304.
- Polverino, F., Cosio, B.G., Pons, J., Laucho-Contreras, M., Tejera, P., Iglesias, A., Rios, A., Jahn, A., Sauleda, J., Divo, M. and Pinto-Plata, V., 2015. B cell–activating factor. An orchestrator of lymphoid follicles in severe chronic obstructive pulmonary disease. *American journal of respiratory and critical care medicine*, *192*(6), 695-705. DOI: https://doi.org/10.1164/rccm.201501-0107oc.
- Pons, J., Sauleda, J., Ferrer, J.M., Barcelo, B., Fuster, A., Regueiro, V., Julia, M.R. and Agusti, A.G.N., 2005. Blunted γδ T-lymphocyte response in chronic obstructive pulmonary disease. *European Respiratory Journal*, *25*(3), 441-446.DOI: https://doi.org/10.3390/cells11121864.
- Qian, Y., Cai, C., Sun, M., Lv, D. and Zhao, Y., 2023. Analyses of Factors Associated with Acute Exacerbations of Chronic Obstructive Pulmonary Disease: A Review. *International Journal of Chronic Obstructive Pulmonary Disease*, 2707-2723. DOI: https://doi.org/10.2196/22591.
- Regan, E.A., Hokanson, J.E., Murphy, J.R., Make, B., Lynch, D.A., Beaty, T.H., Curran-Everett, D., Silverman, E.K. and Crapo, J.D., 2011. Genetic epidemiology of COPD (COPDGene) study design. *COPD: Journal of Chronic Obstructive Pulmonary Disease*, *7*(1), 32-43.DOI: https://doi.org/10.3109%2F15412550903499522.
- Rennard, S.I. and Drummond, M.B., 2015. Early chronic obstructive pulmonary disease: definition, assessment, and prevention. *The Lancet*, *385*(9979), 1778-1788.DOI: https://doi.org/10.1016%2FS0140-6736(15)60647-X.
- Sanchez-Salcedo, P., Divo, M., Casanova, C., Pinto-Plata, V., de-Torres, J.P., Cote, C., Cabrera, C., Zagaceta, J., Rodriguez-Roisin, R., Zulueta, J.J. and Marin, J.M., 2014. Disease progression in young patients with COPD: rethinking the Fletcher and Peto model. *European Respiratory Journal*, *44*(2), 324-331.DOI: http://dx.doi.org/10.1183/13993003.00239-2023.
- Singh, D., Agusti, A., Anzueto, A., Barnes, P.J., Bourbeau, J., Celli, B.R., Criner, G.J., Frith, P., Halpin, D.M., Han, M. and Varela, M.V.L., 2019. Global strategy for the diagnosis, management, and prevention of chronic obstructive lung disease: the GOLD science committee report 2019. *European Respiratory Journal*, *53*(5).DOI: https://doi.org/10.1183/13993003.00164-2019.
- Stockley, R.A., 1999. Neutrophils and protease/antiprotease imbalance. *American journal of respiratory and critical care medicine*, *160*(supplement_1),S49-S52.DOI: https://doi.org/10.1164/ajrccm.160.supplement_1.13.
- Suzuki, M., Sze, M.A., Campbell, J.D., Brothers, J.F., Lenburg, M.E., McDonough, J.E., Elliott, W.M., Cooper, J.D., Spira, A. and Hogg, J.C., 2017. The cellular and molecular determinants of emphysematous destruction in COPD. *Scientific Reports*, *7*(1), 9562.DOI: https://doi.org/10.1038/s41598-017-10126-2.
- Takahashi, M., Fukuoka, J., Nitta, N., Takazakura, R., Nagatani, Y., Murakami, Y., Otani, H. and Murata, K., 2008. Imaging of pulmonary emphysema: a pictorial review. *International journal of chronic obstructive pulmonary disease*, *3*(2), 193-204.DOI: https://doi.org/10.2147%2Fcopd.s2639.
- Verschakelen, J.A. and De Wever, W., 2007. *Computed tomography of the lung*. A pattern approach. Journal of Nuclear Medicine 49(1):164-164. DOI: http://dx.doi.org/10.2967/jnumed.107.045757.
- Vestbo, J., Hurd, S.S., Agustí, A.G., Jones, P.W., Vogelmeier, C., Anzueto, A., Barnes, P.J., Fabbri, L.M., Martinez, F.J., Nishimura, M. and Stockley, R.A., 2013. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. *American journal of respiratory and*

critical care medicine, *187*(4), 347-365.DOI: https://doi.org/10.1164/rccm.201204-0596pp.

- Vestbo, J., Anderson, W., Coxson, H.O., Crim, C., Dawber, F., Edwards, L., Hagan, G., Knobil, K., Lomas, D.A., MacNee, W. and Silverman, E.K., 2008. Evaluation of COPD longitudinally to identify predictive surrogate end-points (ECLIPSE). *European Respiratory Journal*, *31*(4), 869-873.DOI: https://doi.org/10.1183/09031936.00111707.
- Wang, Z., Locantore, N., Haldar, K., Ramsheh, M.Y., Beech, A.S., Ma, W., Brown, J.R., Tal-Singer, R., Barer, M.R., Bafadhel, M. and Donaldson, G.C., 2021. Inflammatory endotype Associated airway microbiome in COPD clinical stability and exacerbations-a multi-cohort longitudinal analysis. *American Journal of Respiratory and Critical Care Medicine,* 203(12): 1488-1502.DOI: https://doi.org/10.1164/rccm.202009-3448oc.
- Anonymus2010.World Health Organization, 2010. *World health statistics 2010*, 147-169.