

## Review

# Lung function decline, obesity and lifestyle: Review

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### Abstract

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**Chronic obstructive pulmonary disease (COPD) and asthma are important healthcare issues. Lung function could be a mortality predictor. Obesity impact on lung function has not been so much emphasized in literature. The large European Community Respiratory Health Survey (ECRHS) population study is revealing that overweight and obesity is impacting lung function by reducing both forced expiratory volume in one second (FEV1) and forced vital capacity (FVC). On the contrary, weight loss is decreasing both. Mechanisms involved in lung function decline due to obesity are mechanical and inflammatory. Lifestyle elements are detailed and their impact on pulmonary function. Tobacco smoking had the worst effect. Physical activity and weight loss could attenuate lung function decline. Healthy lifestyle, with a Mediterranean diet (MD) is leading to a good lung function on long term. Regular physical activity, a good sleep and mindfulness accompany MD in a healthy lifestyle. In conclusion, comprehensive lifestyle interventions should become population based interventions for a better prevention for pulmonary diseases and NCD's and finally for a better health status.**

**Keywords:** Asthma, COPD, Lung function decline, Obesity, Nutritional intervention, Nutritional status

## INTRODUCTION

### Healthcare impact of pulmonary diseases and obesity

Almost 64million disability-adjusted life year (DALY) lost by COPD (Soriano et al., 2017) and a mortality of 3.2 mil/year, which increased in latest years by 11.6% (2015 vs 1990); 0.4 million people who died from asthma, but mortality decreased by 26.7% in the same period are important healthcare issues. And worldwide, prevalence is still increasing for both, by 44.2% for COPD and 12.6% for asthma. Globally, the Global Burden of Disease (GBD) study conducted by Soriano et al is showing these impressive numbers: 63850<sup>th</sup> DALY lost from COPD and 26169<sup>th</sup> DALY lost from asthma.

In this context, lung function may be considered a predictor of morbidity and mortality from noncommunicable diseases, even not so much emphasized in literature until now. In order to better quantify the size of this population issue, more updated measurements, at

population level should be performed and benchmark with other noncommunicable diseases and their association with other exposures, like air pollution, environmental exposure, smoking, ageing. First step in chronic respiratory diseases prevention should be the maintenance of a good lung function. Main risk factors in developing pulmonary diseases are genetics, poor growth in utero and or childhood, anatomical, physiological immunological changes in times, smoking, viral and bacterial infections, environmental pollution, nutritional status, meaning both, obesity and undernutrition, diet, sedentarism.

Lifestyle changes are important measures that should be applied, both in prevention and pulmonary diseases management, as part of multidisciplinary interventions. Increased numbers of life-years in good health will be the result of population prevention measures combined with multidisciplinary disease management.

Another public health issue is obesity. Worldwide,

**Table 1.** New 2020 abdominal obesity classification

<b>BMI = Body mass index WC = Waist circumference</b>	<b>Normal weight (18.5-24.9)</b>	<b>Overweight (25-29.9)</b>	<b>Obese grd I (30-34.9)</b>	<b>Obese grd II and Obese grd III (≥35)</b>
Women	≥80	≥90	≥105	≥115
Men	≥90	≥100	≥110	≥125

almost 2 billion adults were overweight and 671 million obese in 2016 (Heng et al., 2020). As a risk factor for many chronic diseases, like cardiovascular diseases, diabetes, liver diseases and respiratory diseases, obesity has not been so much correlated with pulmonary diseases, despite the fact that many previous studies proved this correlation. Early detection of impaired lung function, as indicator of respiratory injury is leading to a better diagnosis of airways dysfunction.

Not surprisingly, abdominal obesity is associated in many studies with lung function impairment. It is important to mention the newest classification of abdominal obesity from 2020 (Robert et al., 2020) a result of the consensus statement of two prestigious societies working group on visceral obesity (Table 1).

It proposes waist measurements as important opportunity for practitioners for a better obesity management. Waist circumference WC is a positive predictor of mortality, but Body mass index (BMI) is not related to this death risk. The better ability of WC in predicting health outcomes compared to BMI might be related to identify increased Visceral adipose tissue (VAT) mass, which is actually the source of multiple pathogenetic mechanisms.

The group is recommending as an important target for treatment the WC decrease for reducing health risk for male and females. Treatment options comprise nutritional interventions endorsed by routine, moderate physical exercises. Previous gaps have been identified, actual classification is more sensitive, adjusting WC based on BMI and sex. Proper training is important in order to introduce this new classification as a routine practice.

In the future, waist circumference measurements should become a standard for general practitioner, as the Consensus group accepted it as "vital sign".

In this review we would like to address the detrimental effect of overweight and obesity, mainly abdominal obesity to lung function and possible lifestyle interventions.

## MATERIAL AND METHODS

We have searched database and we found 3522 articles related to obesity and lung function. From these we selected newest, with appearance later than 2018. Meanwhile, articles with data about lifestyle, and physical activity correlated with lung function, asthma and COPD have been evaluated in order to obtain a whole picture

about pulmonary diseases and lifestyle, mainly their correlation with obesity.

### Significant studies conclusions: ECRHS (Peralta G et al., 2020)

Many cohort and population studies are proofs of the detrimental effects of obesity, leading to lung function decline. The most detailed data have been obtained during the longest multicentric population based study, ECRHS. This is the most comprehensive population study, multicentric, following for 20 years a large population of 18000 adults, during 3 phases: ECRHS I (1991-1993), ECRHS II (1999-2003), ECRHS III (2010-2014) with very detailed information on BMI, lifestyle factors and spirometry measurements. Spirometry evaluated forced vital capacity (FVC) and forced expiratory volume in first second (FEV1), as markers of lung function. For weight changes evaluations has been considered: weight loss -0.25 kg/year, stable weight +/- 0.25 kg/ year, moderate weight gain 0.25-1 kg/ year. Asthma diagnosis was noted. Lifestyle elements have been recorded: smoking status, leisure time, physical activity. Generalized estimation equation was used to estimate lung function trajectories, appreciating FEV1, FVC, FEV1/ FVC evolution. Main results of this study are summarized in below Table 2.

What conclusions can be drawn? Weight gain, for all people, overweight, normal or obese at baseline, is leading to an accelerated decline of FVC and FEV1, meaning practically an accelerated decline of pulmonary function. Sex stratification during analysis evidenced a marked decline of FVC and FEV1 in male gaining weight compared to females, mainly in obese category but without FEV1 / FVC ratio changes. The mechanical effects mechanism will explain below these differences in the context of more frequent male abdominal obesity compared to females. The proportional decrease of FEV1 and FVC for overweight and obese could be interpreted in the context of the possible development of a restrictive syndrome (Zammit et al., 2011), typical for obese adults.

This conclusion is also in line also with all research about "obesity paradox" revealing that in COPD people with overweight have a lower mortality vs underweight patients, benefit recorded only below the level of BMI of 32kg/m<sup>2</sup>, but not above this value (Hâncu Anca, 2019). Rapid lung function decline in COPD is associated with: emphysema severity, smoking and frequency of acute

**Table 2.** ECRHS main study results

Baseline- young adulthood	During follow up for 20years	At the end of the study FVC, FEV1	FEV1/FVC at the end of the study
Normal BMI, overweight and obese	Weight gain	Accelerated decline of FVC and FEV1	No evidence of decline
Obesity	Weight loss	Attenuated FVC and FEV1 decline	No evidence of decline
Underweight in young adulthood	Stable weight	Attenuation of FVC and FEV1 decline	Accelerated FEV1/FVC ratio decline

FVC - forced vital capacity; FEV1- forced expiratory volume in first second; BMI- body mass index

exacerbations. COPD management has to identify these factors (Vogelmeier et al., 2017) but, still, what precisely influence lung function decline is not known (Yilan Sun et al., 2019). Observational studies mentioned low BMI as a mortality predictor in COPD (Landbo et al., 1999; Schols et al., 1998; Hasegawa et al., 2014; Gudmundsson et al., 2012). Obesity paradox, in contrast is showing the protective effect on mortality when BMI is high.

It could be surprisingly, but for underweight group, FVC and FEV1 decline is attenuated, being a positive effect, but the decline of the ratio FEV1/FVC is accelerated, meaning that an airflow limitation, typical for obstructive pulmonary syndrome may be favored.

Very important is that for obese people at baseline, weight loss is bringing benefits, showing practically the importance of comprehensive lifestyle interventions, mainly nutritional interventions in obese patients for maintaining a good pulmonary function. The complex analysis from ECRHS allowed to see weight change effects on lung function on different weight categories followed on long term throughout adult life. We will analyze below what possible mechanisms could be involved.

## Mechanisms Involved in Lung Function Decline

### a) Mechanical effects on lungs (Gabriela et al., 2020)

Visceral fat mass and thoracic fat reduce FVC by limiting lung expansion which turns to expiratory flow limitation. Observed different results by sex may be explained according to more frequent male abdominal obesity with a direct mechanic impact on lung function. But, in ECRHS there are not compliance measurements or systemic inflammation markers related to obesity. In conclusion, results evidenced in this study could not been distinguished, to be attributable to mechanical or inflammatory effects. In abdominal obesity important fat depots are accumulated in abdominal cavities and mediastinum, limiting mechanical properties of the lung, with the result of a reduced compliance in the lungs (Pelosi et al., 1998; Hedenstierna and Santesson, 1976; Sharp et al., 1964). This is likely to lead to respiratory symptoms typical in obesity: orthopnea (Ferretti et al., 2001), dyspnea (Sin et al., 2002) and wheezing

(Schachter et al., 2001). In obesity there is an increase in pleural and intra-abdominal pressure, diaphragm movements are reduced and chest wall is restricted due to fat accumulation inside abdominal and thoracic cavities (Sugerman et al., 1997; Behazin et al., 2010). Functional residual capacity (FRC) and expiratory reserve volume (ERV) will be reduced consecutively in obesity. Several studies have reported that in obesity both, lung compliance and chest wall are reduced (Pelosi et al., 1998; Hedenstierna and Santesson, 1976; Sharp et al., 1964; Pelosi et al., 1996). The decrease in lung volumes that is induced by obesity may be a major contributor to the lower lung compliance, even tissue mechanics will not be affected (Ubong and Anne, 2018).

### b) Inflammatory processes

Adipose tissue is not an inert organ, but rather a systemic modulator of the response to environmental exposures and a potential target for novel therapeutic interventions. Meanwhile, adipose tissue is a source of inflammatory mediators that can damage tissue and reduce airway diameter (Heng et al., 2020). Meanwhile, is an organ that secretes adipokines, adipocyte- derived factors that could affect airway function. Their expression is different for obese subjects compared to lean subjects. This aspect was the subject of research mainly for obese asthmatic patients. A pro-inflammatory adipokine, leptin is increased in asthma, mainly at obese patients (Sideleva et al., 2012). Beyond the anorexigenic role, leading to an accelerated metabolism and modulatory immune function, leptin has a role also in the ventilatory drive regulation (Polotsky et al., 1985; Bassi et al., et al., 2014). Leptin is involved in neonatal lung development and surfactant production (Torday et al., 2010; De Blasio et al., 2016). Considering these, the pathogenic involvement of leptin in airway disease becomes more and more relevant. A strong association of high serum leptin levels and high BMI is seen in adults asthma patients. For both type of patients, with and without asthma, many investigators reported peak expiratory flow (PEF) and spirometric indices improvement after weight loss. Is important to understand that the obesity induced pathological effect is not permanent and weight loss may restore respiratory physiology.

**Table 3.** Plasmatic proportion of CRP correlated with anthropometric measurements and spirometry parameters

Mediated % of CRP	Correlated WC	WHR	WHtR
As with FEV1	7.96	9.59	5.76
As with FVC	8.33	11.4	-

CRP - C reactive protein; FEV1- forced respiratory volume in first second; FVC forced vital capacity  
WC waist circumference; WHR waist hip ratio; WHtR waist to height ratio

Very well described in a cross sectional study on 3442 Chinese participants (Heng et al., 2020) the possible mechanism of systemic inflammation explaining the pathogenesis relating obesity with lung function decline is an important perspective. Each unit increase in waist circumference WC has been associated with FEV1 and FVC decrease, 3,39 ml respective 3,96 ml ( $p < 0.05$ ). Waist- hip-ratio WHR and Waist to Height Ratio WHtR increase by 1% was correlated with decrease in FEV1 by 5,70 ml and FVC by 16,92 ml ( $p < 0.05$ ). The correlation between lung function and abdominal obesity was mediated by plasmatic C reactive protein (CRP), suggested the study.

Plasma CRP is playing a mediating role in the association between abdominal obesity and lung function, shows the mediation analysis, using MEDIATION described by Valeri (Frohlich et al., 2000). Mediated proportions of CRP are shown in the Table 3 above:

Higher plasma CRP levels is more frequent at older smokers with higher BMI, WHtR, WC, WHR but lower FVC and FEV1, being less active and eating mainly home prepared meals. The Chinese study (Heng et al., 2020) shows that each increase by 1% in WHtR and WHR has been associated with 0.010 and 0.012 elevated level of plasma CRP, statistical significant ( $p < 0.05$ ). Significant FVC and FEV1 decline is shown in category analysis as long as CRP is increasing. Interestingly, plasma CRP was not found to be associated with FEV1/FVC ratio. Chinese results found negative association between abdominal indices representing abdominal obesity and FVC and FEV1, but negative association between plasma CRP and FEV1 and FVC. In this study, BMI could not be correlated with lung function, other studies being inconclusive, too (Frohlich et al., 2000; Vatrella et al., 2015; Sin et al., 2002). Another British study performed on 2633 people didn't find an association between FEV1, FVC and BMI (Fogarty et al., 2011), consistent with Chinese study. But for a group of smokers, BMI was inversely associated with lung function (Pistelli et al., 2008). The distribution of body fat is important in the correlation with lung function, abdominal obesity being mainly correlated with FVC and FEV1 decrease, for that reason WC, WHR could be more accurate measurements in predicting lung function compared to BMI. In conclusion, the correlation between WC, WHtR, WHR mediated by plasma CRP is supporting the fact that systemic inflammation may be the mechanism beyond the association between abdominal obesity and decreased lung function.

### c) Metabolic dysregulation

Lung function is affected by metabolic imbalance observed in metabolic syndrome. Airways smooth muscle ASM proliferation and epithelial damage could be induced by dyslipidemia and insulin resistance (Agrawal et al., 2011; Dekkers et al., 2009). Restrictive pulmonary syndrome is linked to metabolic syndrome, obesity but not diabetes in elderly patients (Scarlata et al., 2013; Fimognari et al., 2007). Higher level of insulin resistance are seen in patients with a restrictive pattern compared to those with obstructive lung disease or healthy subjects.

### Lifestyle impact on pulmonary function – Smoking

Smoking is the leading recognised risk factor for chronic respiratory diseases. This link between lung function and smoking is already well studied. Looking to the lifestyle interaction with obesity and smoking the algorithm became more complex. It seems that smoking risk is substantially greater than obesity in low income countries (Jha, 2020).

Rate of death for smokers is three fold higher at middle age vs nonsmokers if we are looking to other common risk factors as alcohol use, obesity patterns, social status (Jha, 2020).

Smoking is accelerating the loss of lung function. This is very evident in COPD where different mechanical pathways are altered. In a sample of COPD patients it had been shown the presence of shorter telomeres compared with healthy smokers, and correlations between telomere shortening, premature senescence and lung inflammation was already reported in some studies (Mercado et al., 2015). Smoking affects differently and it is associated with an accelerated loss in lung function in individuals with short telomeres and with little/no effect in those with longer telomeres. This short telomere length in peripheral leucocytes might be a marker for increased susceptibility to the effect of smoking (Pascal et al., 2017).

The variability in telomere length is influenced not only by genetic factors but by modifiable lifestyle factors such as smoking, obesity, physical activity or diet.

Phenotyping remains important if we have to judge smokers lung function. The small airways COPD phenotype, present in younger people, with less emphysema, a preserved lung diffusion and a slower rate of lung decline is more frequent in smokers vs non-smokers (Salvi et al., 2020).

Another evidence of this relation - obesity, smoking-lung function is coming from asthmatic patients. We already know that smoking is inducing a progressive airways hyper responsiveness (AHR) with a peak at elderly asthmatic people. Higher AHR was found in elderly obese; but obesity was considered a higher AHR risk factor only in subjects aged >65 years (Sposato et al., 2016).

For asthmatic obese waist to height ratio and insulin resistance can predict impaired lung function and also this insulin resistance can modify the association between excessive adiposity and respiratory function (Sadeghimakki and McCarthy, 2019).

Reduced lung function can be a predictor of future development of T2D independently of obesity, smoking and inflammation, as has been shown by the Korean Genome and Epidemiological Study which included 7583 non diabetics, aged 40-69y, followed for 12years. The association correlates incident T2D with lung function predicted forced vital capacity % PFVC and predicted forced expiratory volume in first second %PFEV1, but not with FEV1/FVC (Lee et al., 2019).

Prenatal and childhood exposures is with important impact on lung development, lung function trajectory, and incidence and prevalence of respirator disease. Avoiding known detrimental exposures including maternal smoking during pregnancy and initiation of active smoking, sustaining policies promoting reduction of population-level risk factors (restriction even on electronic cigarette sales and legislation to uphold air quality standard) bring some benefits like a maximal lung function and a reduce risk of chronic lung disease (Grant et al., 2020).

Studies of exposure to vaping, both in humans and animals have been associated with several pathophysiological alterations in the lung functions (evaluated by the forced expiratory volume in one second (FEV1) and the ratio of forced expiratory volume to forced vital capacity (FEV/FVC), combined with reported effects on the airway mucociliary clearance and dysregulated repair mechanisms. Many compromising factors are implicated like flavors, tetrahydrocannabinol (THC oil), vitamin E Acetate etc are involved in this new disease generated by e cigarette disease EVALI (Meo et al., 2019; Staudt et al., 2018). The best method to avoid death caused by smoking is early quitting, before the age of 40 (Jha et al., 2013).

### **Physical activity and lung function improvement (Elaine et al., 2018)**

- There is a growing evidence that physical activity has a beneficial effect in maintaining pulmonary health (Henriksen et al., 2017).
- Hunt Study (Henriksen et al., 2017) – Physical activity and lung function decline in adults with asthma was still unclear, recently was evaluated in Hunt study. A mean

follow-up of 11.6 years in 1329 people has been done in a population cohort from Norway. As a result of this study is noticed that for active participants the mean FEV1 decline was 32 ml/year, lower compared to 37 ml/ year characteristic for inactive participants. Difference is -5ml / year, 95% CI. Meanwhile, FVC decline was 31 ml/ year for physical active adults and 33 ml/ year for sedentary participants. FEV1/ FVC decline was statistically significant lower, 0,22% vs 0,36% for inactive asthmatic persons. Mean decline observed in peak expiratory flow PEF was slower for active asthmatics – 10 ml/year compared to 14 ml/ year observed for inactive people. In conclusion, physical activity is correlated with a lower decline in lung function for people with asthma. That means in asthma physical activity should be promoted as part of management tool, message supported by Global Initiative for asthma, too (Global Initiative for Asthma, 2015).

Similar recommendations have been suggested in COPD (Hayley et al., 2017). Hayley et al, in their review, have shown that 21 (60%) clinical practice guidelines included in this review provided specific recommendations for physical activity. Most recommended activities are: walking, cycling, strength training and other non-specific aerobic exercises. The context for performing physical activity is to include it in person's lifestyle and social life, in recreational clubs, independently or at home. Four from six guidelines included in the review recommended for COPD patients to be active as long as their capacity allows it or until breathless (French Language Pneumology Society, 2010; Gupta et al., 2013; Ministry of Health Malaysia, Academy of Medicine Malaysia, Malaysian Thoracic Society, 2016). Duration should be from 20 min to 45 min/day. In severe COPD short intervals rather than continuous activity is indicated [56]. Frequency should be regularly, under supervision (The Institute for Healthcare Improvement CBO, 2010; French Language Pneumology Society, 2010; Danish Health Authority, 2016). The most suggested strategy to improve physical activity is active encouragement from health care providers (57%) and education (11,31%) but also an exercise training program (17%).

Physical activity, lung function and pro-inflammatory lifestyle risk factors:

❖ Current smokers, characterized by a systemic inflammatory burden will benefit more from anti-inflammatory effects of physical activity (Fuentes et al., 2018; Garcia-Aymerich et al., 2007; Gan et al., 2005; Kasapis and Thompson, 2005).

❖ Another pro-inflammatory risk factor for lung function decline is the exposure to air pollution. Individuals exposure to air pollution during outdoor physical activity may affect pulmonary function? The interplay between physical activity, lung function and air pollution is important to be understood.

In order to answer this question many studies have been developed. The largest is ECRHS, which collected

extensive lifestyle information from 18000 adults, using questionnaires. Individuals were classified as physically active when the frequency of exercises was 2 times/week at least 1 h and otherwise were considered non-active. In ECRHS, the combination of duration and frequency characterizing physical activity has been positively associated with FVC, FEV1, but also with decreased bronchial hyper-responsiveness (Fuertes et al., 2018). Pollution in ECRHS II was estimated by the model LUR-was 31 ml / land use regression (Fuertes et al., 2018). It was designated the individual level of air pollution for home addresses. Then, as confirmatory analysis, it was created a set of markers of NO<sub>2</sub>, PM<sub>2.5</sub>mass and PM<sub>10</sub>mass exposure. Observed results, mean FEV1 was lower among those living in high pollution areas (NO<sub>2</sub> and PM<sub>10</sub> mass). For smokers, physical activity was improving FVC and FEV1 independent of air pollution level. But physical activity for non-smokers was correlated statistically significant with higher average FVC and FEV1 for those living in areas where air pollution was low.

### Effect of weight loss on pulmonary function

ECRHS shows the attenuated lung function decline for subjects with obesity or overweight that lost weight in time (Wang et al., 1997; Bottai et al., 2002; Carey et al., 1999; Chen et al., 1993; Chinn et al., 1996; Chinn et al., 2005; Thyagarajan et al., 2008; Pistelli et al., 2008). Is important to understand mechanisms that could explain this association:

- 1) Mechanical load on lungs will decrease during weight loss, improving lung function, this could be the first mechanism
- 2) second, systemic inflammation will be reduced in the meantime with weight loss, and that can attenuate lung function decline which is related to excessive weight. Previous research revealed that air pollution affects lung function via systemic inflammation and air quality improvement will attenuate lung function decline (Schikowski et al., 2013).
- 3) another proposed mechanism is related to the decrease of metabolic alterations observed after weight loss, after removal of excessive adipose tissue, glycemic levels, insulin resistance, hyperlipidemia will be improved (Baffi et al., 2016; Phelan et al., 2007).
- 4) other confounding factors should be taken into consideration, lifestyle improvement is more complex and changing diet, starting physical activity or quitting smoking could all bring beneficial effects on lung function (Chinn et al., 2005; Fuertes et al., 2018; Garcia-Aymerich et al., 2007; Willemse et al., 2004).

More research on lifestyle factors involved in lung function evolution will bring additional data on this subject.

### The role of nutritional intervention in lung function improvement/ prevention

Adherence to Mediterranean Diet, as a predictor of lung function was evaluated in a study using data from Health and Retirement Study HRS. There have been evaluated 2108 adults, aged 50 and more, MedDiet score was calculated and peak expiratory flow PEF was measured. Results observed in this cross sectional study: a medium Med Diet score of 28, meaning a moderate adherence to MD correlated with lung function (95% CI, 0,039-0,104). A positive association has been established between PEF rate ( $p < 0.05$ ) and an important food category: dairy, grains and fish. High adherence to Mediterranean Diet is associated with lower incidence of PEF rate <80% from predicted value. (OR: 0.65, 95% CI: 0.48-0.89).

Based on these results it can be assumed that a high MD score is a good predictor of lung function in adults. Nutritional interventions may be possible preventive interventions and should be indicated especially for persons at high risk of pulmonary disease. Mediterranean Diet represent an evidence based intervention with protective role in lung function maintenance (Ionas and Sheikh; Gutierrez-Carrasquilla et al., 2019).

### Healthy lifestyle in pulmonary diseases

When a pulmonary disease is already developed, a multidisciplinary team, with pneumologist, nutritionist and even kineo-therapist should take care to recommend a healthy lifestyle with optimal diet, physical activity, good sleep and mindfulness based cognitive therapy. Rehabilitation programs does not include yet nutrition in their activity, but it could be a future plan.

### Medical nutrition therapy MNT for patients with obesity and COPD

As a priority in obesity, for patients with COPD, muscular mass should be considered, due to the fact that muscular mass decrease is an important mortality risk factor. Recommended protein intake is 1,2-1,5 g/kg/day (Rebecca et al., 2017). There is a need for specific guidelines in this area.

Macronutrients will represent: proteins 15-20%, fats 30-45%, carbohydrates 40-55% from total energy intake. Macronutrients proportion is important in order to achieve the optimal respiratory quotient (RQ), marker for respiratory tolerability for the recommended dietary pattern. RQ is the report between CO<sub>2</sub> expired and the volume of O<sub>2</sub> consumed. The value is 1 when the diet is based only on carbohydrates, 0.85 for a mixed diet, 0.82 for a protein diet, 0.7 for fats and 0.65 in ketosis. Vitamin C is necessary, 75 mg/day for females and 90 mg / day for males; for smokers an additional amount of 35 mg/

day should be added. Recommended daily intake for vitamin D should be 600 IU/day, at > 70y, 800 IU/ day, daily sun exposure. Calcium and Magnesium plasmatic values should be tracked (Kathleen and Janice, 2016). Energy requirements for COPD obese patients should be adapted to their activities and will be measured by bioimpedance and calorimetry. Obesity paradox, was already mentioned, the dilemma in COPD with obesity is to recommend weight loss and achieve a better cardiovascular prognosis but could lower lung performance (Vanessa et al., 2017; Ingeborg et al., 2018). And which nutritional intervention will be the best in order to have an optimal weight? Actual data associate BMI ~ 30 kg/m<sup>2</sup> with highest survival rate (Hâncu Anca, 2019), but this benefit disappear at BMI > 32 kg/m<sup>2</sup>. Then, the possible lifestyle intervention will be: caloric restriction, in order to maintain a BMI level ~ 30 kg/m<sup>2</sup> (meaning we will not encourage weight loss for overweight people), high protein intake and physical exercises to strengthen muscular mass. Pulmonary rehabilitation is the multidisciplinary intervention that improves effort capacity, dyspnea, healthcare access, including physical activity trainings. This intervention should include nutritional intervention and maybe in the future mindfulness based cognitive therapy in order to obtain complex results and a better quality of life.

### **MNT and lifestyle recommendations for patients with obesity and asthma**

In asthma, 2019 Guideline for obesity management for general practitioners, elaborated by European Association for the Study of Obesity (DurrerSchutz et al., 2019) is suggesting a 7-8 % weight reduction for obese asthma patients in order to obtain an improvement of FEV<sub>1</sub>.

New guidelines for asthma lifestyle recommendations have been published in Breathe, 2019 (Stoodley et al., 2019). As dietary pattern, Mediterranean diet MD, high in vegetables, fruits, omega 3 and fibers is related to anti-inflammatory pathways. Cross sectional studies are showing asthma control improvement by adherence to MD.

### **Specific nutrients analysis**

**Sodium:** Increased sodium intake is very common in western diets and the link with asthma could be established due to changes in sodium transport across smooth muscle cells, which could affect properties of airway smooth muscle (Global Initiative for Asthma, 2018). Not clearly documented by clinical research for asthma (Hirota and Janssen, 2007) current general recommendations that include avoiding added salt and highly processed food should be followed.

**Antioxidants:** A negative role in plasma development is played by oxidative stress (Guo et al., 2012). Nutritional interventions in asthma should address an antioxidant diet, with beneficial effects on lung airways inflammation, asthma control and lung function (Wood et al., 2008). Fruits and vegetable intake was proved to reduce asthma exacerbations risk (Wood et al., 2012). Vitamin C, despite its well known antioxidant effect is lacking in evidence and more research is needed to establish it as a tool in asthma management. Dietary fiber are important in order to maintain a healthy microbiome; Short Chain Fatty Acids SCFA's, produced by commensal bacteria after a proper intake of fibers exert an anti-inflammatory effect. Probiotics are effective in the reduction of systemic inflammation (McLoughlin et al., 2017). Probiotics, present in yoghurt, kefir are living microorganisms that may increase gut bacteria benefits. Microbiome modulation can be relevant in asthma (West et al., 2016). Synbiotics, with a potential immune function improvement should ameliorate asthma outcomes. A RCT showed that 4 weeks of symbiotics intake decreased cytokine production without any effect on bronchial inflammation (van de Pol et al., 2011). More research is needed in this area, but still a healthy microbiome is a good asset.

**Omega 3 fatty acids,** found in salmon, sardines and herring (Stoodley et al., 2019) exert anti-inflammatory properties by pathways involving airway hyper-responsiveness. Despite the fact that supplementation with N-3 poly-unsaturated fatty acids PUFA was proposed as a support in asthma treatment, evidence is contradictory. Guidelines are suggesting that people with asthma should follow general recommendations for fish intake. An important meta-analysis showed a decreased risk of hospitalized asthma exacerbations after vit D supplementation. Sunlight exposure 10-15 min / day on arms and face, 2-3 times per week is recommended to avoid vit D deficiency. Some practical advices for people with asthma: vit D supplementation will exert protective effects against acute respiratory infections and will reduce exacerbation rate. As main sources: salmon, sardines, orange juices, fortified milk, eggs. Due to tocopherol vit E may decrease the risk of some asthma symptoms like wheezing or cough. Good sources are broccoli, hazelnuts, almonds. Sulfits should be avoided, causing worsening asthma symptoms. They are found in many dried fruits, leading to adverse reactions.

A light bronchodilator, caffeine can be found in coffee and tea may support asthma treatment (Stoodley et al., 2019). A systematic review found that 5-10 mg caffeine / kg body weight ameliorates lung function after 4 hours of consumption (Stoodley et al., 2019). Maximum allowed caffeine intake per day is 400 mg. The bronchodilator effect will appear after 4-5 cups of coffee. But this recommendation is valid only if other comorbidities are not limiting this quantity.

Physical activity should support a healthy nutritional intervention in asthma. In asthma 3-5 days per week (Morton and Fitch, 2011) with 20-60 min should be recommended, to reduce wheezing and exacerbations, except exercise induced bronchoconstriction (EIB). However, EIB should be prevented by anticipated treatment with short acting beta-agonists SABA's and exercise should be regularly. Little evidence is exploring different styles of physical activity. In conclusion, in asthma, a lifestyle intervention with antioxidant diet, with caloric adjustment to obtain a healthy weight, plenty of fruits, vegetables, and dairies, characteristic for MD will be appropriate and regular physical activity, quitting smoking, mindfulness approach and a healthy sleep should be included.

Mindfulness based cognitive therapy could be a support in asthma treatment and should be considered as a part of pulmonary rehabilitation

## CONCLUSIONS

Obesity impact on lung decline is well explained by mechanical and inflammatory mechanisms. Significant studies, like the large ECRHS have shown on long term an accelerated decline in FVC and FEV1 consecutively to weight gain and an attenuated lung function decline after weight loss. Smoking, Western diet, sedentarism as unhealthy lifestyle factors may affect lung function and are risk factors for pulmonary function decline. Nutritional intervention with a healthy MD is the key element of a healthy lifestyle, accompanied by physical activity, non smoking status good sleep and mindfulness. Comprehensive lifestyle interventions should become population based interventions for a better prevention for pulmonary diseases and NCD's and finally for a better health status.

## Conflict of Interest

Authors declare that there are no conflicts of interests related to this subject.

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