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Alterations in Nutritional Status and Body Composition in COPD Patients

Joaquim Gea, MD, PhD, Cristina Estirado, MD and Esther Barreiro, MD, PhD

Respiratory Medicine Department, Hospital del Mar IMIM, DCEXS, Universitat Pompeu Fabra, CIBERES, ISCIII, Barcelona, Spain

ABSTRACT

This review focuses on nutritional abnormalities, one of the most prominent extrapulmonary manifestations occurring in chronic obstructive pulmonary disease (COPD). Diagnosis is usually made by either anthropometry or determination of body composition. Deficiencies in nutritional status, such as body weight and muscle mass loss, are the result of an interaction of several factors, including the imbalance between energy supply and requirements, tobacco, low physical activity, and systemic inflammation. These factors essentially determine the predominance of protein breakdown over synthesis. The loss of body weight and lean mass leads to muscle dysfunction and exercise limitation, also having a negative impact on exacerbations and mortality. Therapies include changes in lifestyle and nutritional supplements. Anabolic drugs may be administered in some cases. Obesity is also very prevalent in COPD patients, being associated with cardiovascular and metabolic comorbidities. Although, paradoxically, moderate obesity appears to reduce mortality, healthy lifestyle habits are recommended to avoid morbid obesity. **(BRN Rev. 2017;3:56-71)**

Corresponding author: Joaquim Gea, JGea@parcdesalutmar.cat

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Correspondence to:
Joaquim Gea
Servei de Pneumologia
Hospital del Mar
Pg. Marítim 27
08003 Barcelona, Spain
E-mail: JGea@parcdesalutmar.cat

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a highly prevalent disorder, characterized by its complexity and heterogeneous presentation[1]. COPD not only involves airways and lung parenchyma, but is also associated with systemic manifestations and different comorbidities. Moreover, additional deleterious circumstances, such as aging and an unhealthy lifestyle, are often present. Nutritional abnormalities stand out among the systemic manifestations of COPD for their frequency and impact on patients' lives. In this sense, it is very well known that deficits in nutritional status impair prognosis, bringing a poorer quality of life, higher exacerbation rate, and increased mortality[2,3]. Moreover, one of the most important consequences of lean mass loss is muscle dysfunction, which in turn results in reduced exercise capacity and physical activity[3-5]. To date much less attention has been paid to the association of COPD with obesity. The latter is also highly prevalent, being associated with cardiovascular and metabolic comorbidities[6]. This review will focus primarily on nutritional deficiencies and their consequences, and only a short section will be devoted to obesity and its implications for patients.

EPIDEMIOLOGY

The actual prevalence of deficits in nutritional status is relatively high, but depends both on the criteria used for diagnosis and the population that has been studied. In general, diagnosis is based on body mass index (BMI, ratio between weight in kg and height in square meters), derived from anthropometric measurements, and/or fat-free mass or lean index (FFMI, a variable that derives from a compartmental model of body composition, where fat free mass is specifically considered), being generally obtained with bioelectrical impedance or dual-energy X-ray absorptiometry (DEXA). According to BMI, the prevalence of nutritional and body composition deficiencies in COPD patients from Northern, Eastern, and Central Europe as well as North America ranges between 15-50%[7,8]. By contrast, the prevalence in Mediterranean countries appears to be lower, with figures ranging between 3-20%[9,10]. These discrepancies have been partially attributed to different lifestyles linked to cultural specificities (diet, physical activity), discrepancies in the variable and threshold values used for diagnosis by different authors (see next section), and healthcare level where patients have been recruited. In fact, the highest prevalences have generally been reported in individuals recruited in rehabilitation centres or hospital clinics, while they are much lower in other environments with less selection bias. An important issue is the potential difficulties of nutritional deficiency diagnosis in female patients, since here a significant discrepancy may occur between BMI and FFMI. The latter detects the loss of lean body mass, even though body weight remains stable or even increases[8]. Nutritional abnormalities affect not only muscle mass, but can also have an impact on bone and fat tissues, reaching a state of cachexia in the more advanced situations. Finally, it should be noted that nutritional deficiencies have been associated with different COPD profiles or phenotypes, especially the emphysematous type (pink puffer), and those patients with muscle manifestations[11].

In any case, regardless of its actual prevalence, the clinical negative impact of underweight and loss of muscle mass is very important for COPD patients (Fig. 1), requiring both an early diagnosis and appropriate management. In fact, it has been clearly demonstrated that even moderate increases in body weight can improve quality of life and prognosis in patients with nutritional abnormalities[2].

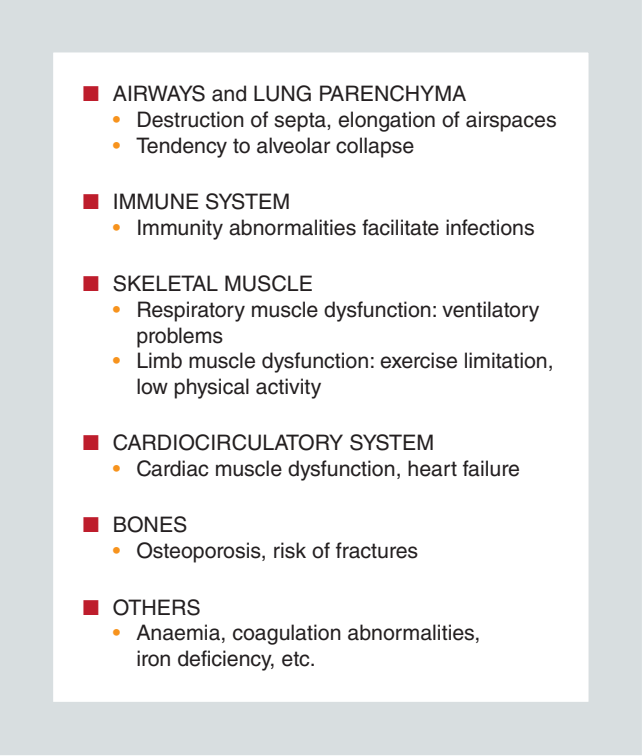

Figure 1. Consequences of nutritional abnormalities on different tissues and systems.

DIAGNOSIS

As already mentioned, nutritional abnormalities can be easily detected using different variables and measuring instruments (Fig. 2). The easiest to be obtained but less specific are anthropometric variables such as body weight, percentage of ideal body weight (%IBW) or BMI. Regarding IBW, values under 80-85% indicate that there is a deficit in nutritional status[12], while the BMI threshold is usually set at 18.0-18.5 kg/m^2 [13], although some authors prefer higher values (20-21 kg/m^2). The nutritional problem can be considered as already serious if the BMI is below 16 kg/m^2, and if it is less than 15 kg/m^2 it is considered as very severe[13]. A more specific approach is to determine the FFMI, using bioelectrical impedance or DEXA[3,14]. Both techniques provide an accurate approximation of muscle mass, which in the case of DEXA can also be compartmentalized in body portions (i.e. trunk, upper or lower extremities). The most accepted FFMI limit to define a loss of lean mass is 16 kg/m^2 in men, and 15 kg/m^2 in women[8]. However, higher values for men (18 kg/m^2) and lower values for women (14.5 kg/m^2) have been proposed for Mediterranean populations[15]. There are other methods that can also be used to assess nutritional status, although they are much less employed. This is the case for image techniques, such as computed tomography, magnetic resonance, and ultrasounds, which can quantify the mass of specific muscle groups or localized fat mass[16-18], providing even functional information[17,19]. Triceps skinfold or thigh circumference are some of the additional anthropometric measurements that can be used, but they have fallen into disuse in recent decades. Finally, there are more sophisticated techniques such as air displacement plethysmography, densitometry by immersion in water, deuterium dilution, and isotopic methods[3,11,14], although their use is either cumbersome or expensive. Blood analysis can also be useful to assess nutritional status. More specifically, determinations of serum total proteins, albumin fraction, cholesterol, and prothrombin time have been used by different authors[3].

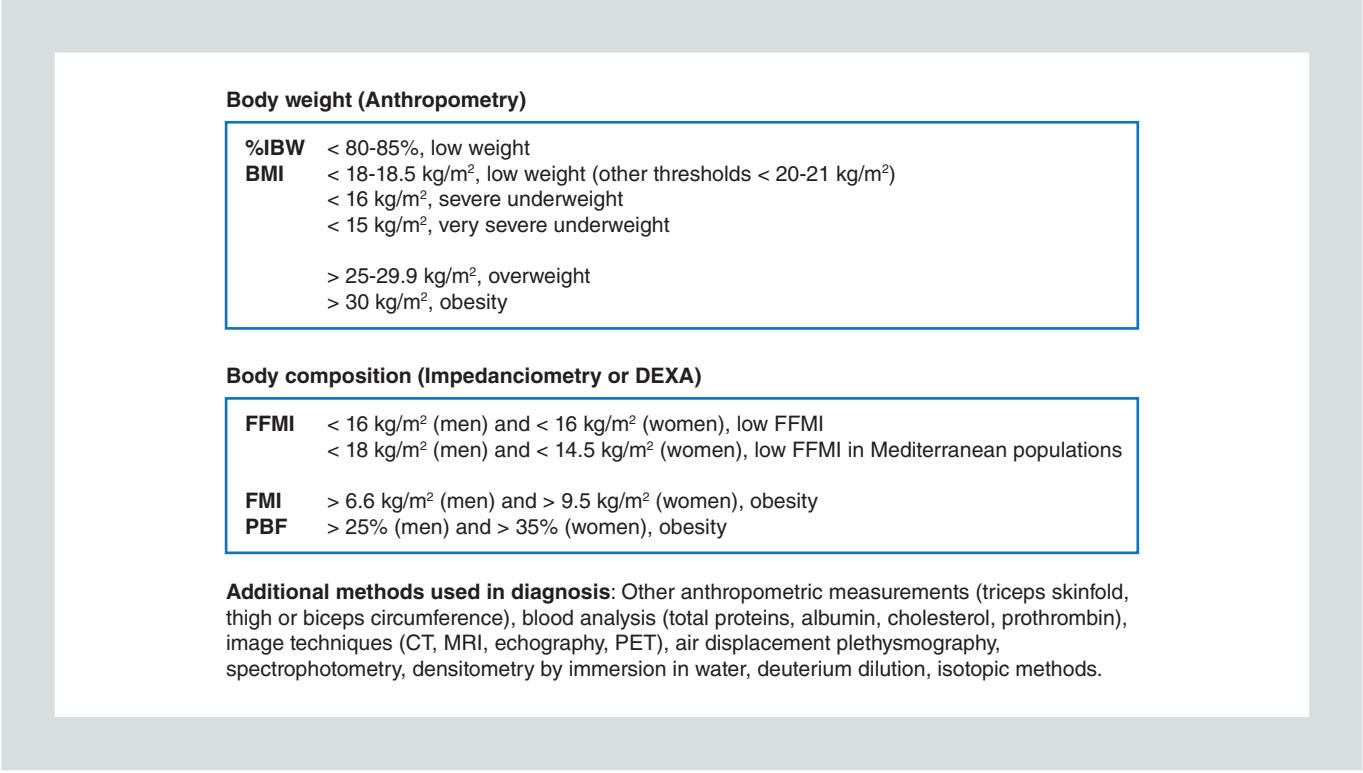

Figure 2. Techniques and thresholds used in the diagnosis of nutritional abnormalities.
%IBW: percentage of ideal body weight; BMI: body mass index; CT: computed tomography; DEXA: dual-energy x-ray absorptiometry; FFMI: fat free mass index; FMI: fat mass index; MRI: magnetic ressonance imaging; PBF: percentage of body fat; PET: positron emission tomogrpahy.

Surprisingly, although an extremely underweight patient was already included in the two classical COPD phenotypes, recent guidelines such as GOLD or GesEPOC have not highlighted the frequent presence of nutritional abnormalities[1,20]. This is probably due, at least in part, to the lack of a well defined therapy for these problems. In fact, the multidimensional BODE score and its derivates are practically the only ones that have included BMI in those variables used to establish the severity and prognosis of COPD[21].

ETIOLOGICAL FACTORS

It is commonly accepted that body weight and muscle mass loss occurring in COPD is due to the interaction of different factors. These include lifestyle circumstances as well as metabolic and hormonal imbalances, among others, all of them leading to a predominance of catabolism on anabolism (Fig. 3). Their interaction and the predominance of one or another factor is specific to each particular patient.

Factors related to lifestyle habits

Low level of physical activity and sedentary lifestyle

This factor is very common in COPD patients and results in cardiovascular and skeletal

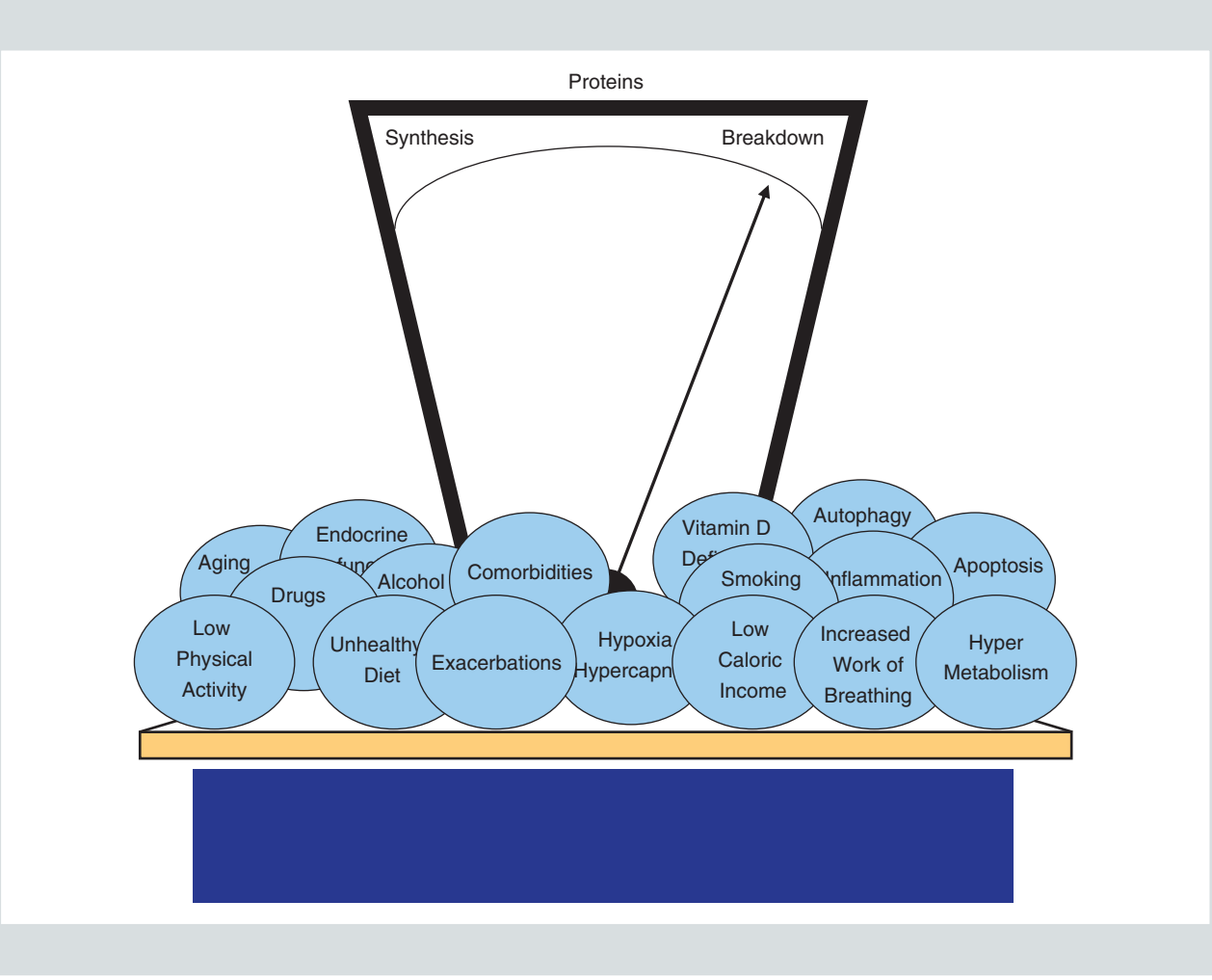

Figure 3. Factors and biological mechanisms involved in nutritional status deficiencies in COPD.

muscle deconditioning, also having a negative impact on the immune status, exacerbations, and mortality[22]. Moreover, reduction in physical activity can induce biological events such as increases in the level of systemic inflammation and decreases in muscle protein synthesis[23]. However, it should be noted that an excessive increase in the level of activity, as in intense exercise, may also induce systemic oxidative stress and adverse effects on muscles in untrained subjects[24]. Therefore, increases in physical activity must be progressive and well controlled in COPD patients.

Smoking status

Tobacco smoking has an anorectic effect and can reduce body weight and muscle mass through induction of systemic inflammation and oxidative stress, release of leptin, and an imbalance between protein synthesis and breakdown[25,26].

Alcohol abuse

Alcohol abuse is associated with smoking in many cases[27]. In fact, smokers are four times

more likely to have alcohol dependence than non-smokers[27]. As a result, 7-20% of COPD patients are heavy drinkers[28], a factor that has been associated with higher mortality and poorer quality of life[28]. Moreover, alcohol abuse is involved in weight, fat mass, and muscle mass loss in the general population[29]. Although no studies to date have evaluated the role of alcohol abuse in underweight COPD patients, its involvement is very likely.

Unhealthy diet

As mentioned, the caloric intake of COPD patients is often inadequate, not matching their energy requirements. Moreover, they consume less carbohydrates, proteins, vitamins A, B6, B9, B12, C, D, and E, and less carotenes and omega 3 fatty acids than healthy subjects of the same age[30,31]. These deficits are more pronounced in patients with weight loss[30,31].

Other deleterious factors

Exacerbations

These acute episodes increase the level of local and systemic inflammation markers as well as those of leptin. Moreover, anorexia, fever, reduced physical activity, and increases in work of breathing are often added to this adverse metabolic picture[11].

Imbalance between energy expenditure and caloric intake

A hypermetabolic status occurs in COPD in addition to the abovementioned increase in the work of breathing[32]. Both circumstances increase energy needs[32], which can not be supplied with normal caloric intake. This scenario worsens if the latter becomes reduced due to the presence of cofactors such as smoking, dyspnea, inflammation, or even an exacerbation.

Hypoxia and hypercapnia

Hypoxia induces inflammation and oxidative stress in different body tissues, deteriorating both metabolism and the actions of some peptides and hormones involved either in appetite or the maintenance of muscle, bone, and fat masses. This is the case for leptin, ghrelin, and AMP-activated protein kinase, which can induce autophagy and muscle apoptosis while reducing mitochondrial synthesis (biogenesis)[33]. In contrast, the main impact of hypercapnia in nutritional status is indirect, through the induction of respiratory acidosis. This in turn will result in less energy stores and an imbalance between protein synthesis and breakdown[34].

Endocrine dysfunction

The effect of anabolic hormones is frequently altered in COPD patients. This is the case of androgens, since a variable prevalence of hypogonadism has been described in COPD men with reduced muscle mass[35]. Furthermore, a decrease in growth hormone levels (GH) secreted by the adenohypophysis, and/or a dysfunction in the powerful anabolic axis that includes its hypothalamic-releasing factor, GH, and insulin-like growth factor 1 (IGF-1) have also been reported[36,37]. The IGF-1

participates in growth and muscle repair, while helping in the maintenance of bone mass and immunological status[37].

Vitamin D deficiency

A significant number of COPD patients show a decreased intake of vitamin D and calcium[31]. This circumstance can also be associated with a reduction in outdoor physical activities, with a lack of sunlight exposure, which is required for vitamin D endogenous synthesis. Therefore, it is not surprising that vitamin D deficiency ranges between 40% in patients with mild COPD and rises to 77% in those with more severe disease[38]. It is well known that vitamin D plays an essential role in the mineralization and maintenance of bone mass, but it also has important effects on muscle mass, the cardiovascular system, and immune status[38]. In fact, between 20-60% of COPD patients have osteoporosis[39], a factor that can contribute to both underweight and an increased risk of fractures (Fig. 1).

Aging

Aging by itself can also induce loss of body weight and even sarcopenia (loss of muscle mass associated with senescence) and osteoporosis. These effects on anthropometry and/or body composition can be further impaired in the presence of COPD[40].

Comorbidities

Other chronic diseases are commonly associated to COPD due to both sharing the same aetiological factors and the effects of aging in many patients. Chronic heart failure, diabetes, and cancer are among the most frequent comorbid conditions associated with nutritional status deficiencies[3].

Drugs

Although other drugs can impair muscle structure and/or function, systemic glucocorticoids, which can cause chronic or acute myopathy and muscle loss due to a reduced synthesis of structural proteins[41], should be specially highlighted.

Biological mechanisms

Loss of proteostasis

Probably the most important mechanism involved in the reduction of body weight and lean mass is the imbalance between reduced protein synthesis and an increased protein breakdown[3,11,37]. This imbalance has devastating effects on different tissues and physiological functions (Fig. 4). It occurs due to factors mentioned in the preceding section and additional biological mechanisms, such as inflammation and oxidative stress, taking place at both systemic and local levels (see below)[3,42,43]. On the one hand, a reduction in protein synthesis signalling has been observed in limb muscles of COPD patients[44,45]. This is probably due both to the low availability of amino acids to assemble new proteins and a failure in the synthesis pathways[45]. The former, in turn, appears to be secondary to a reduced protein intake[30] and the low uptake by skeletal muscles[46], among other factors.

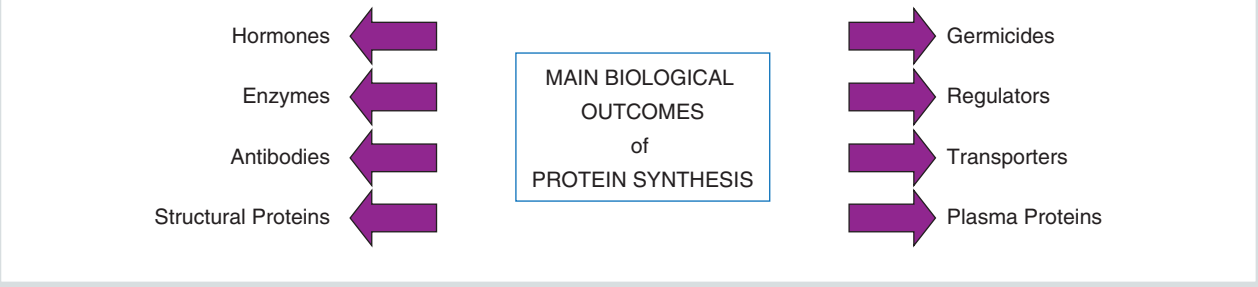

Figure 4. Main biological outcomes that become impaired by a reduction in protein content secondary to the loss of proteostasis.

Regarding protein synthesis itself, it has been described that anabolic hormone signals can be reduced[37] and some key factors, such as protein kinases B (Akt) involved in signalling pathways, seem to be decreased or partially inactivated in COPD patients[47,48]. Moreover, nuclear apoptosis has also been reported in muscle tissue of COPD patients[49]. The significance of this finding is unclear as muscle cell is a syncytium (several nuclei in the same cell)[49]. Since muscle apoptosis results in pauci-nuclear fibres, it has been suggested that it would determine a decrease in protein synthesis (nuclear muscle wasting)[3]. On the other hand, protein catabolism is increased due to multiple mechanisms[11]. In COPD, the most relevant catabolic system for muscle proteins is probably the one linked to proteasome[11]. This cell corpuscle degrades proteins, with or without prior ubiquitination, and its activity is greatly increased in underweight COPD patients[50]. However, ubiquitination needs the intervention of atrogenin-1 and MuRF1, both regulated by FoxO transcription factors, which are overexpressed in skeletal muscles of COPD patients[51-53]. Calpains, proteolytic enzymes that are very abundant in skeletal muscles and can increase their action by factors such as a reduction in physical activity, also seem to be involved in the loss of muscle mass of these patients[54]. Autophagy is another cellular event that is also upregulated in muscles of COPD patients with low body weight[54,55]. This process, closely related to lysosome enzymes (such as lipases, glycosidases, and cathepsins), occurs through the autophagosome-lysosome system and is a physiological mechanism of protein degradation[54]. Its abnormal increase would also lead to the loss of proteostasis observed in patients. Interestingly, there is some evidence supporting a probable role for epigenetic mechanisms such as lysine-hyperacetylation of histones that may play a relevant role in some of these pathways involved in protein hypercatabolism occurring in COPD[55].

Inflammation and oxidative stress

Both phenomena are closely interrelated and have been demonstrated in COPD patients, both at the systemic level and in

lungs, muscles, and adipose tissue[24,49,50,56]. Their deleterious action may be direct or through the activation of other biological processes and metabolic pathways that also favour disturbances in nutritional status. Inflammation is involved in the aforementioned apoptosis and autophagy, also activating other catabolic systems[3]. Reciprocally, different factors such as exacerbations, hypoxia, inactivity, or intense exercise are able to increase the baseline levels of inflammation and oxidative stress in COPD patients[23,24,57].

Other tissues

Most studies of weight loss in COPD patients have focused on skeletal muscle tissue and much less attention has been devoted to other tissues such as fat or bone. However, there is evidence that they are also affected by nutritional abnormalities associated with this lung disease. As already mentioned, bone involvement is very common in COPD patients (20-60%)[39,58]. Regarding fat mass, this can also be reduced in some patients, particularly in those with advanced stages of COPD and cachexia[59]. However, the mechanism of this loss is unclear since lipolysis seems to be preserved[3]. In addition, the adipose tissue is involved in the production of biological substances that can contribute to the low body weight of the patients. In this regard, some authors have reported an increase in adiponectin and other inflammatory mediators in the fat of COPD patients, along with a reduction in the levels of leptin[60,61]. Factors that have been involved in the loss of either adipose or bone tissues are similar to many of those that have been implicated in muscle mass reductions (smoking, inactivity, systemic inflammation, metabolic imbalance, etc.)[3].

THERAPEUTIC INTERVENTIONS

As diverse factors and mechanisms are involved in weight and muscle mass loss in COPD, the therapeutic approach should be multidimensional (Fig. 5). Inappropriate lifestyle habits must be corrected, avoiding smoking and alcohol, increasing the level of physical activity, and improving the diet quality. In addition, whenever possible, those drugs known to have a deleterious effect on nutritional status, such as systemic steroids, should be avoided. Furthermore, and as a second step in the treatment, appropriate dietary supplements can be used and, in the most extreme cases, even drugs with anabolic properties can be prescribed.

Diet

Some simple tips, such as increasing the intake of protein-rich foods (especially those containing polyunsaturated fatty acids), fibre, and the vitamins where they are most deficient (A, B6, B9, B12, C, D, and E), seem to be sufficient to maintain and even regain weight in many COPD patients with nutritional deficiency[62]. In fact, a healthy diet and diet interventions can even delay the onset and progression of COPD[63-65]. In this regard, a diet rich in fruits and vegetables seem to have a positive influence on lung function and mortality[63]. Moreover, it is important that diet should be rich in fibres as this has demonstrated a positive impact on delaying

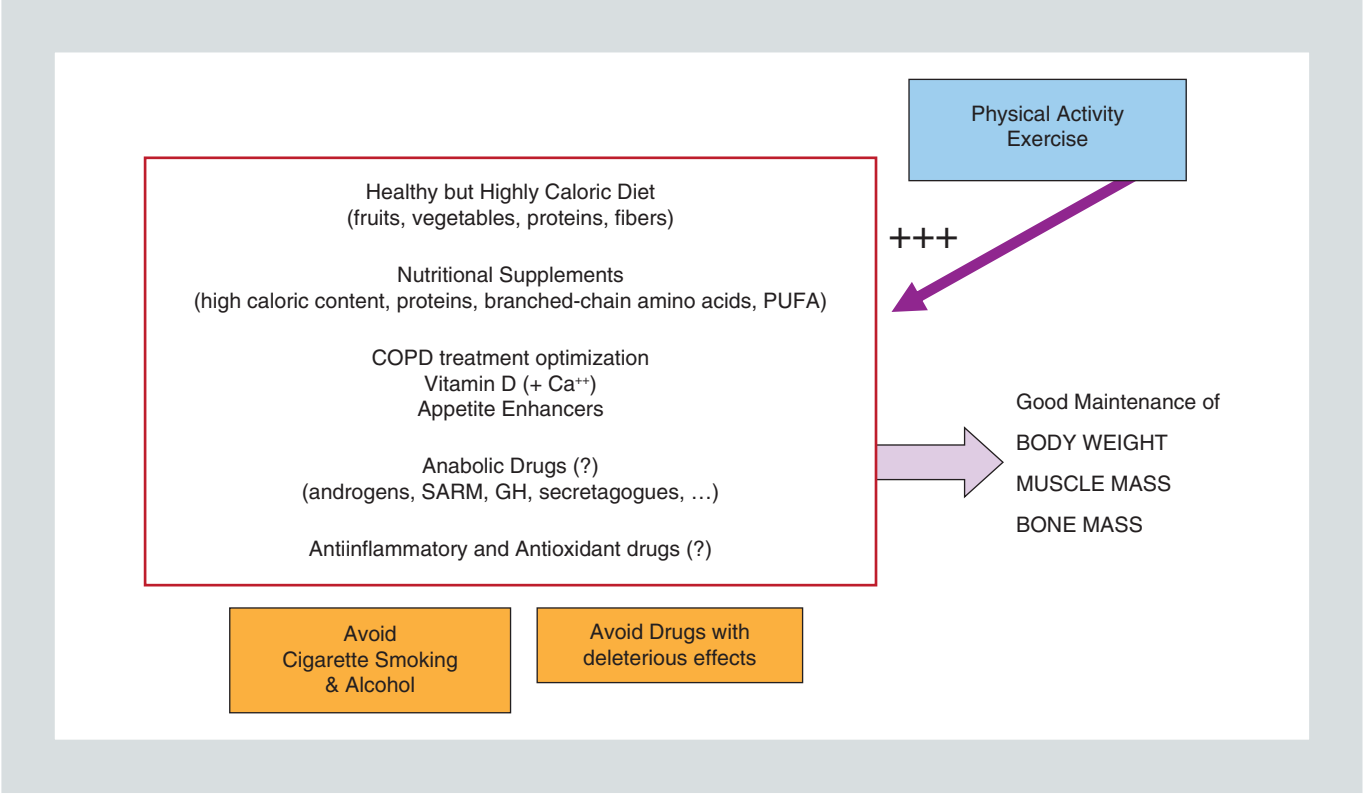

Figure 5. Therapeutic measures that can help in body weight, fat-free mass, and bone mass gain and/or maintenance in COPD patients. GH: growth hormone; PUFA: polyunsaturated fatty acids; SARM: Selective androgen receptor modulators.

the onset and progression of COPD, probably because of its effects on the intestinal microbiome[66].

Nutritional supplements

Nutritional supplements can be used as an adjunct to an adequate diet to improve its effects. In general, the supplements should provide an appropriate amount of calories, as well as proteins and amino acids that would allow an adequate protein synthesis[11]. Liquid supplements with high-caloric content can help in restoring weight and muscle mass in these patients[67]. It is important to note that the source of amino acids and proteins is also very relevant. In this regard, supplements derived from milk seem to get better amino acid concentrations than those obtained from soybean[68]. Also, proteins from whey are absorbed very efficiently by the bowel, and their amino acid composition allows rapid initiation of protein synthesis[11]. One issue to be taken into account is that COPD patients, especially those with muscle mass loss, have low concentrations of branched-chain amino acids[69], so it would be beneficial to include these in the nutritional supplements. Moreover, it has been shown that their administration also improves patients' muscle metabolism[69]. As happens with anabolic drugs, the combination of dietary supplements with moderate physical activity enhances the nutritional effects of therapy, also being accompanied by clinical and functional improvements[2,32,67].

Finally, it has also been reported that the administration of polyunsaturated fatty acids, such as omega-3, improves the exercise capacity of patients, although its effects on lean body mass are more controversial[70].

In summary, probably the best approach for a nutritional intervention in malnourished COPD patients is to modify their habits in order to achieve a healthy and balanced diet, and administer high-caloric supplements, enriched with proteins, amino acids (especially branched chain amino acids) and polyunsaturated fatty acids as well as vitamin combinations.

Appetite enhancers

Megestrol, an oral progesterone derivative, has been used among other drugs. However, the results in underweight COPD patients have been disappointing[71].

Anabolic drugs

Most anabolic drugs are of a hormonal nature, related to androgens or GH. Testosterone, which can be administered either intramuscularly or transcutaneously, is in the former group. It enhances protein synthesis and decreases protein breakdown, thereby increasing muscle mass[37]. However, testosterone also increases lipolysis, with consequent loss of fat mass, and has other undesirable side effects. Substances with a similar androgenic effect but also important side effects are oxandrolone, which can be administered orally, and nandrolone[36,37]. Unfortunately, in most of the studies performed in COPD patients, these drugs are able to increase muscle mass, but this is not associated to parallel benefits in functional properties[3,36]. More recently, some trials have been performed with selective androgen receptor modulators, which have anabolic actions similar to testosterone but fewer undesirable side effects[3,72]. However, the results are still controversial.

Growth hormone, in turn, has a powerful anabolic effect not only at the skeletal muscle level, by increasing protein synthesis and inhibiting its destruction, but also because it increases the available calcium for building new bone. This hormone can be administered subcutaneously and is able to increase body weight and muscle mass in COPD patients with malnutrition[73]. Unfortunately, this effect is not associated with a clear functional improvement and side effects can become important[3,73]. Therefore, administration of GH secretagogues (substances that promote synthesis and/or release of the endogenous hormone) have been used as an alternative. The best known is ghrelin, which is physiologically synthesized by various tissues (including the lung). It is administered intravenously and its effects are those of GH itself, but ghrelin also stimulates the appetite and has anti-inflammatory properties. The results in underweight COPD patients are controversial. In some cases it improved weight and lean mass with good functional results[74], but in other studies the effects have been much more modest. Another secretagogue is tesamorelin, which is more stable and can be administered subcutaneously. In COPD patients it appears to improve lean muscle mass and strength, with few side effects[75]. However, definitive results are pending.

Many other GH secretagogues, such as capromorelin, examorelin or sermorelin, are currently under study.

COPD treatments

Bronchodilators can reduce respiratory symptoms and work of breathing, which in turn would allow the patient to increase the level of physical activity. Something similar would happen with the instrumental reduction of pulmonary hyperinflation through endoscopic or surgical techniques. Moreover, an appropriate treatment of COPD exacerbations may prevent increases in systemic inflammation and reduce the period of severe gas exchange alterations and physical inactivity.

Vitamin D supplementation

Previous sections have alluded to the common vitamin D deficit showed by COPD patients. It is therefore appropriate to administer supplements of this vitamin along with calcium, and try to increase the hours of outdoor patient activity.

Anti-inflammatory and antioxidant drugs

Since inflammation and oxidative stress are involved in the pathogenesis of COPD, different drugs have been tested to neutralize these phenomena. However, only very few studies have focused on the effects of these drugs on weight and fat-free mass loss. Infliximab, for instance, which has been used to counterbalance the effects of tumour necrosis factor-α, did not show clear effects on either the lung disease or systemic inflammation, having important side effects[76]. Moreover, although one study evidenced an improvement in exercise capacity, no changes have been observed in BMI or FFMI when this drug was used in underweight COPD patients[76]. Other drugs such as tocilizumab (anti IL-6), anakinra or canakinumab (both anti-IL1) either have not been tested in these patients or have not shown significant changes in nutritional parameters[77]. Regarding antioxidants, ascorbate administration appears to reduce systemic oxidative stress, improving muscle function, although no effects have been reported on patients' nutritional status[78]. Similar effects have been obtained with N-acetylcysteine and other antioxidants, both in animal models and COPD patients[79].

Other therapeutic strategies

It has recently been suggested that inhibitors of NF-kB and MAPK could be useful in treating cachexia by reducing oxidation and protein catabolism and autophagy. However, there is still a lack of studies in underweight COPD patients[80].

OBESITY AND COPD

It has already been mentioned that a significant percentage of COPD patients (20-30%) are obese, as defined by a BMI > 30 kg/m^2 [6,13,81,82]. This proportion reaches two thirds of patients if those with overweight (BMI 25.0-29.9 kg/m^2) are added[6,81,82], being clearly higher than that observed in the general population[6]. Obesity can also be diagnosed based on body composition, with the thresholds for

fat mass index (FMI) established at ≥ 6.6 kg/m^2 in men and ≥ 9.5 kg/m^2 in women, and those for the percentage of body fat obesity in ≥ 25% for men and ≥ 35% for women[81]. Obesity is not necessarily associated with maintenance of muscle mass, since relatively often this tissue is replaced by fat, especially in women. Hence the need to determine not only anthropometric variables but different body compartments. For obesity associated muscle wasting, some authors use the term "sarcopenic obesity". Paradoxically, mild-to-moderate obesity appears to be associated with lower mortality in COPD patients[83]. The most likely explanation is that, like in other critical circumstances, energy reserves linked to obesity partially counterbalance the increased catabolism that is characteristic of severe acute illness.

The causes of obesity associated with COPD do not differ substantially from those affecting the general population of the Western world, but are probably more accentuated: a low level of physical activity, inappropriate diet (it has been shown that COPD patients with abdominal obesity consume a high amount of calories but low quantities of proteins and micronutrients), and other unhealthy habits[6]. Obesity can also be caused by hormonal changes linked to COPD, its comorbidities, and/or treatments (i.e. steroids)[60]. The consequences of obesity derive largely from cardiovascular and metabolic complications and the potential association with a hypoventilation syndrome[84,85]. In addition, obese patients have a higher level of dyspnea than patients with normal BMI[6,82].

To date there is a lack of consensus for recommendations in the management of obesity in COPD patients. However, it seems reasonable to suggest a slow and moderate weight reduction and prevention of cardiovascular and metabolic complications. For this, it should be recommended to improve the quality of diet and increase physical activity, if necessary with a relatively low-intensity training program[86]. With these simple measures, most patients will reduce weight but maintain muscle mass, therefore improving exercise capacity and health-related quality of life[82,86]. Surgical or endoscopic treatments may be necessary to achieve weight reduction in extreme obesity. Although there are no specific studies in COPD, it has been reported that surgical weight reduction may improve spirometric values[87] and ventilation/perfusion relationships[88], reducing bronchial hyperactivity[89], cardiovascular morbidity and mortality[90], and systemic inflammome[91] in morbid obesity. However, it should also be taken into account that the presence of abnormal spirometric values increases the risk of bariatric surgery[92].

CONCLUSIONS

Nutritional disorders are frequently found in COPD patients, being diagnosed through anthropometry and determination of different body components. Low body weight and muscle mass result in muscle dysfunction, impairment in exercise capacity, and worse outcomes, including increased mortality. These nutritional abnormalities are caused by the interaction of different factors such as smoking, low physical activity, systemic inflammation, and an imbalance between caloric intake and energy expenditure, which favour protein catabolism. The treatment of underweight

patients includes improvement of lifestyle habits, dietary supplements and, in some particular cases, the use of anabolic drugs. Obesity is also very prevalent in COPD patients, being associated with cardiovascular and metabolic comorbidities. However, moderate obesity seems to have a protective role against mortality. Therefore, its management must be cautious and should include improvements in lifestyle habits.

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CONFLICT OF INTEREST

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