AZIENDA OSPEDALIERA DI TERNI
UNIVERSITÀ DEGLI STUDI DI PERUGIA
Struttura Complessa Medicina Interna
Malattie dell'Apparato Respiratorio
Direttore: Prof. Giuseppe Schillaci

BPCO: evidenze dell' efficacia del riallenamento

Giuseppe Fiorenzano

ATS/ERS Eur. Respir. J. 2004; 23: 932-946

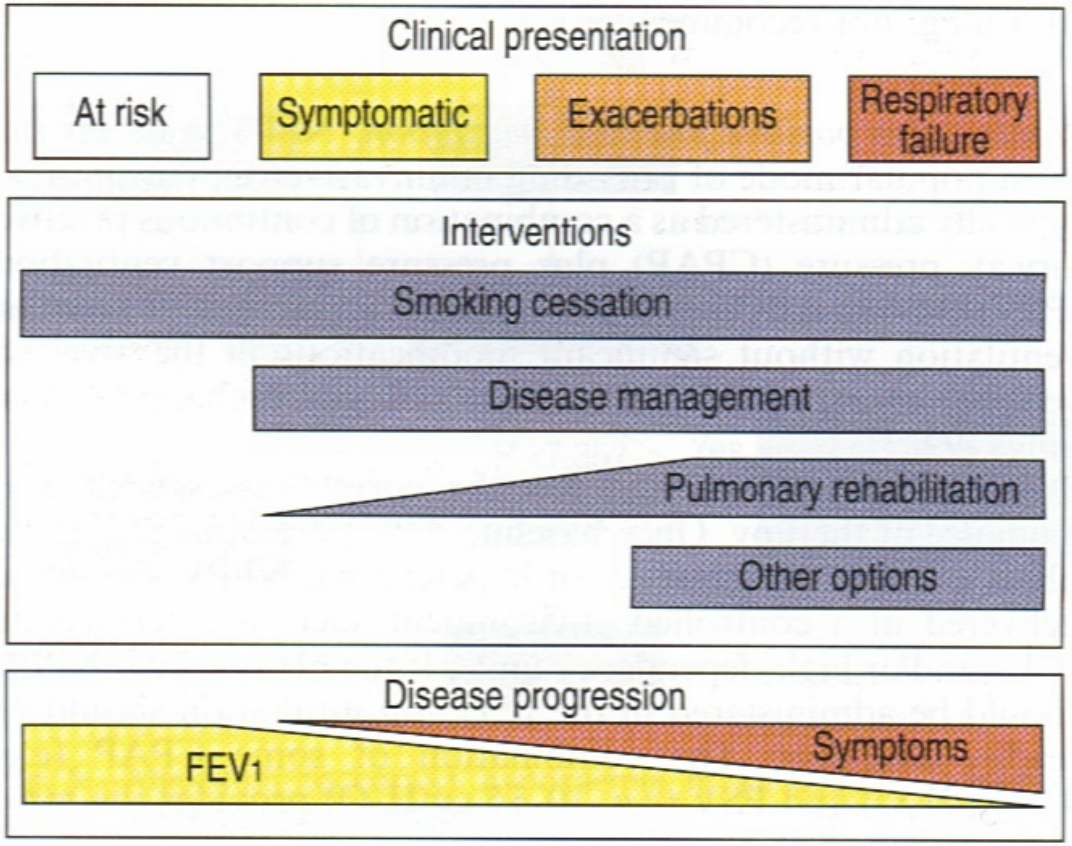


Fig. 7.–Continuum of care for chronic obstructive pulmonary disease (COPD). FEV1: forced expiratory volume in one second.

Strategia globale per la diagnosi, trattamento e prevenzione della BPCO 2015:
Classificazione di gravità spirometrica della BPCO

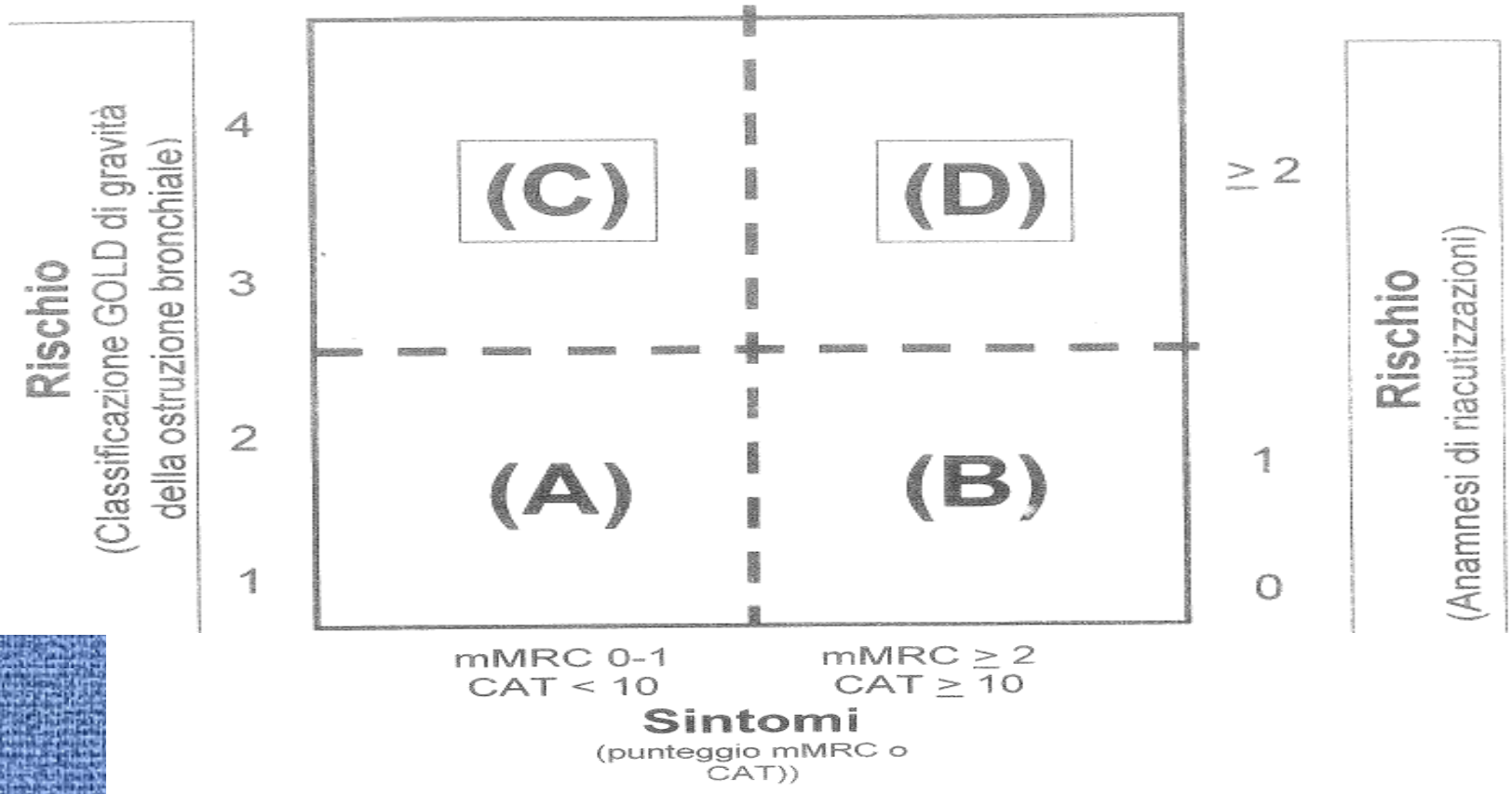
In pazienti con VEMS/CVF < 0.70 dopo broncodilatatore

GOLD 1: Lieve	**VEMS ≥ 80% del teorico**
GOLD 2: Moderata	**50% ≤ VEMS < 80% del teorico**
GOLD 3: Grave	**30% ≤ VEMS < 50% del teorico**
GOLD 4: Molto Grave	**VEMS < 30% del teorico**

Classificazione basata sul valore di VEMS misurato dopo broncodilatore

Valutazione combinata della gravità della BPCO

(modificato Gold 2013)



Nel valutare il rischio, tenere in considerazione il valore più alto fra gravità della ostruzione bronchiale e storia di riacutizzazioni

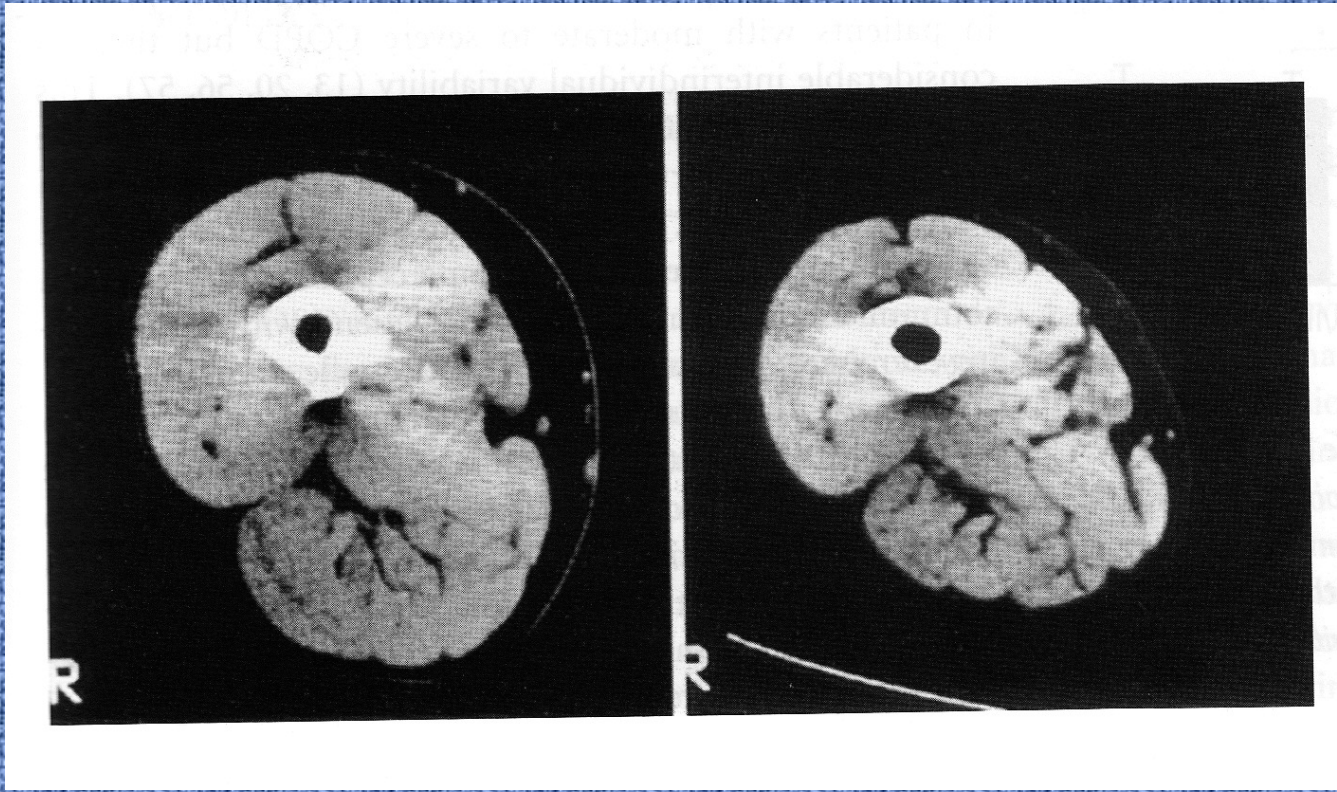
Il paziente rientra quindi in una di queste 4 categorie:

A: Sintomi lievi, basso rischio
B: Sintomi gravi, basso rischio
C: Sintomi lievi, alto rischio
D: Sintomi gravi, alto rischio

Progetto strategico mondiale per la diagnosi, trattamento e prevenzione della BPCO:
Trattamento della BPCO stabile: trattamento non farmacologico

Paziente gruppo	Essenziale	Raccomandato	Secondo le linee guida locali
A	Cessazione del fumo di sigaretta (può includere il trattamento farmacologico)	Attività fisica	Vaccinazione antinfluenzale Vaccinazione antipneumococcica
B, C, D	Cessazione del fumo di sigaretta (può includere il trattamento farmacologico) Rabilitazione respiratoria	Attività fisica	Vaccinazione antinfluenzale Vaccinazione antipneumococcica

Bernard S, et al., Am. J. Respir. Crit. Care Med. 1998; 158: 629-34



ATS/ERS Statement on Pulmonary rehabilitation
Am. J. Respir. Crit. Care Med. 2006; 173: 1390-1413

TABLE 1. PATHOPHYSIOLOGIC ABNORMALITIES IN CHRONIC RESPIRATORY DISEASE AND POSSIBLE MECHANISMS FOR IMPROVEMENT AFTER EXERCISE TRAINING

	Pathophysiologic Abnormality	References	Changes with Exercise Training	References
Body composition	↓ Lower limb muscle cross-sectional area ↓ Fat-free mass and ↓ fat mass ↓ % Fat-free mass and =/↑ fat mass	35	↑ With resistance training ↑ Fat-free mass and fat mass with rehabilitation and nutritional supplementation ↑ Fat-free mass and ↓ fat mass with rehabilitation (resistance + endurance combined Enhanced ↑ fat-free mass with testosterone (1) and anabolic steroids	360, 194, 361
Lower limb muscle fiber type, size	↓ % Fiber type I and myosin heavy chain (advanced disease) ↑ % Fiber type IIX ↓ Fiber cross-sectional area linked to muscle atrophy	40, 158, 362–367	= Fiber-type proportion ↑ Fiber cross-sectional area	362
Capillarization	↓ Capillary contacts to fiber cross-sectional area, especially in patients developing fatigue during exercise	45, 362	↑ Capillary contacts proportional to increase in fiber cross-sectional area	362
Muscle metabolic capacity	↓ Capacity of oxidative enzymes: citrate synthase, 3-hydroxyacyl-CoA dehydrogenase, succimic acid dehydrogenase, cytochrome C oxidase ↑ Cytochrome-c oxidase activity in hypoxemic patients	41, 368, 369	↑ Capacity of oxidative enzymes after endurance training	63
Metabolism at rest/after exercise	Rest: ↓ intracellular pH, ↓ [PCr] and [ATP], ↑ lactate and inosine monophosphate; ↓ glycogen stores in hypoxemic patients; ↓ glycogen stores related to physical activity level; ↓ uncoupling protein-3 content Exercise: rapid decline in muscle intracellular pH, phosphocreatine/inorganic phosphate [PCr/Pi] even in patients with relatively preserved submaximal oxygen delivery	42, 61, 68, 370–373	↓ Lactic acidemia at iso work rate Normalization of decline in intracellular pH and PCr/Pi. Faster PCr-recovery.	31, 61
Inflammatory state	↑ Inflammatory/apoptotic markers may occur in skeletal muscle in subpopulations of wasted COPD	374, 375	No effect shown or not studied	
Redox state	Glutathione levels normal to moderately reduced ↑ Oxidative stress in the skeletal muscle of COPD patients after quadriceps exercise	363, 376–378	↑ Oxidized gluthatione in contrast to observations in healthy subjects. Partially reversed by antioxidant therapy (N-acetyl cysteine)	379

AMERICAN THORACIC SOCIETY DOCUMENTS

An Official American Thoracic Society/European Respiratory Society Statement: Update on Limb Muscle Dysfunction in Chronic Obstructive Pulmonary Disease

François Maltais, Marc Decramer, Richard Casaburi, Esther Barreiro, Yan Burelle, Richard Debigaré, P. N. Richard Dekhuijzen, Frits Franssen, Ghislaine Gayan-Ramirez, Joaquim Gea, Harry R. Gosker, Rik Gosselink, Maurice Hayot, Sabah N. A. Hussain, Wim Janssens, Micheal I. Polkey, Josep Roca, Didier Saey, Annemie M. W. J. Schols, Martijn A. Spruit, Michael Steiner, Tanja Taivassalo, Thierry Troosters, Ioannis Vogiatzis, and Peter D. Wagner; on behalf of the ATS/ERS Ad Hoc Committee on Limb Muscle Dysfunction in COPD

This official statement of the American Thoracic Society (ATS) and the European Respiratory Society (ERS) was approved by the ATS Board of Directors, November 2013, and by the ERS Executive Committee, September 2013

Background: Limb muscle dysfunction is prevalent in chronic obstructive pulmonary disease (COPD) and it has important clinical implications, such as reduced exercise tolerance, quality of life, and even survival. Since the previous American Thoracic Society/European Respiratory Society (ATS/ERS) statement on limb muscle dysfunction, important progress has been made on the characterization of this problem and on our understanding of its pathophysiology and clinical implications.

Purpose: The purpose of this document is to update the 1999 ATS/ERS statement on limb muscle dysfunction in COPD.

Methods: An interdisciplinary committee of experts from the ATS and ERS Pulmonary Rehabilitation and Clinical Problems assemblies determined that the scope of this document should be limited to limb muscles. Committee members conducted focused reviews of the literature on several topics. A librarian also performed a literature search. An ATS methodologist provided advice to the committee, ensuring that the methodological approach was consistent with ATS standards.

Results: We identified important advances in our understanding of the extent and nature of the structural alterations in limb muscles in patients with COPD. Since the last update, landmark studies were published on the mechanisms of development of limb muscle dysfunction in COPD and on the treatment of this condition. We now have a better understanding of the clinical implications of limb muscle dysfunction. Although exercise training is the most potent intervention to address this condition, other therapies, such as neuromuscular electrical stimulation, are emerging. Assessment of limb muscle function can identify patients who are at increased risk of poor clinical outcomes, such as exercise intolerance and premature mortality.

Conclusions: Limb muscle dysfunction is a key systemic consequence of COPD. However, there are still important gaps in our knowledge about the mechanisms of development of this problem. Strategies for early detection and specific treatments for this condition are also needed.

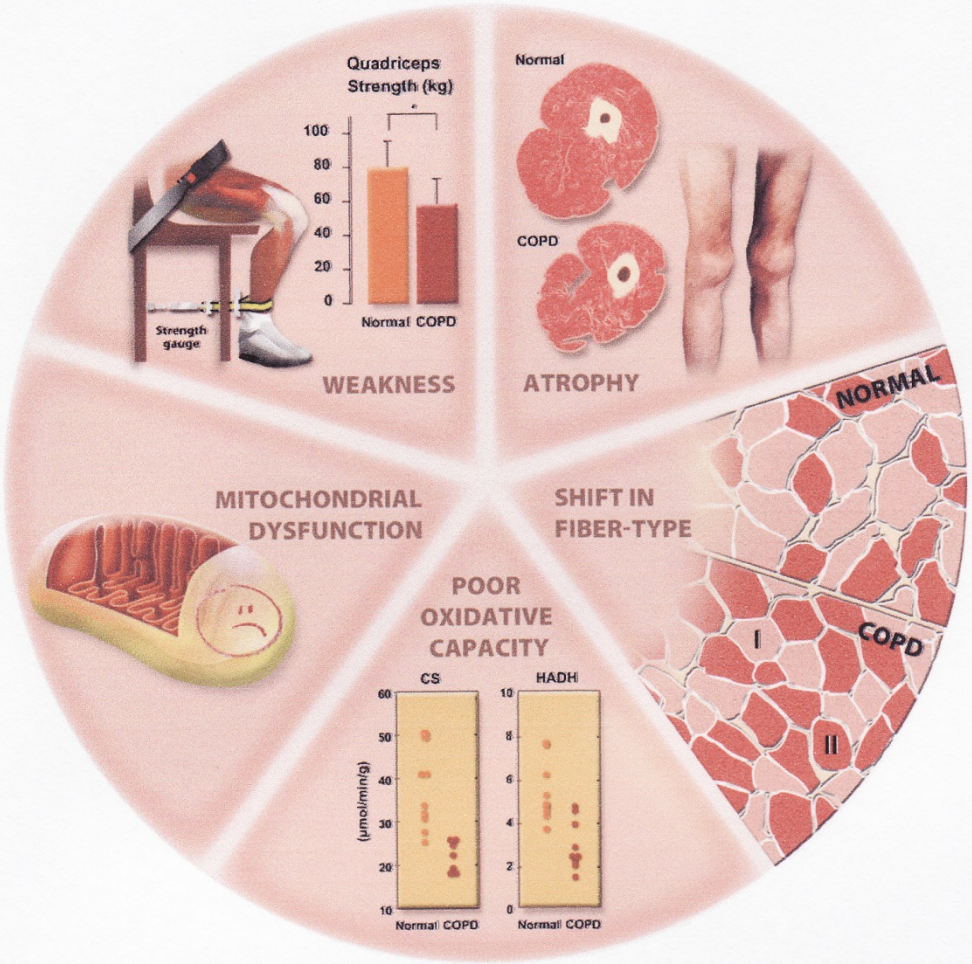
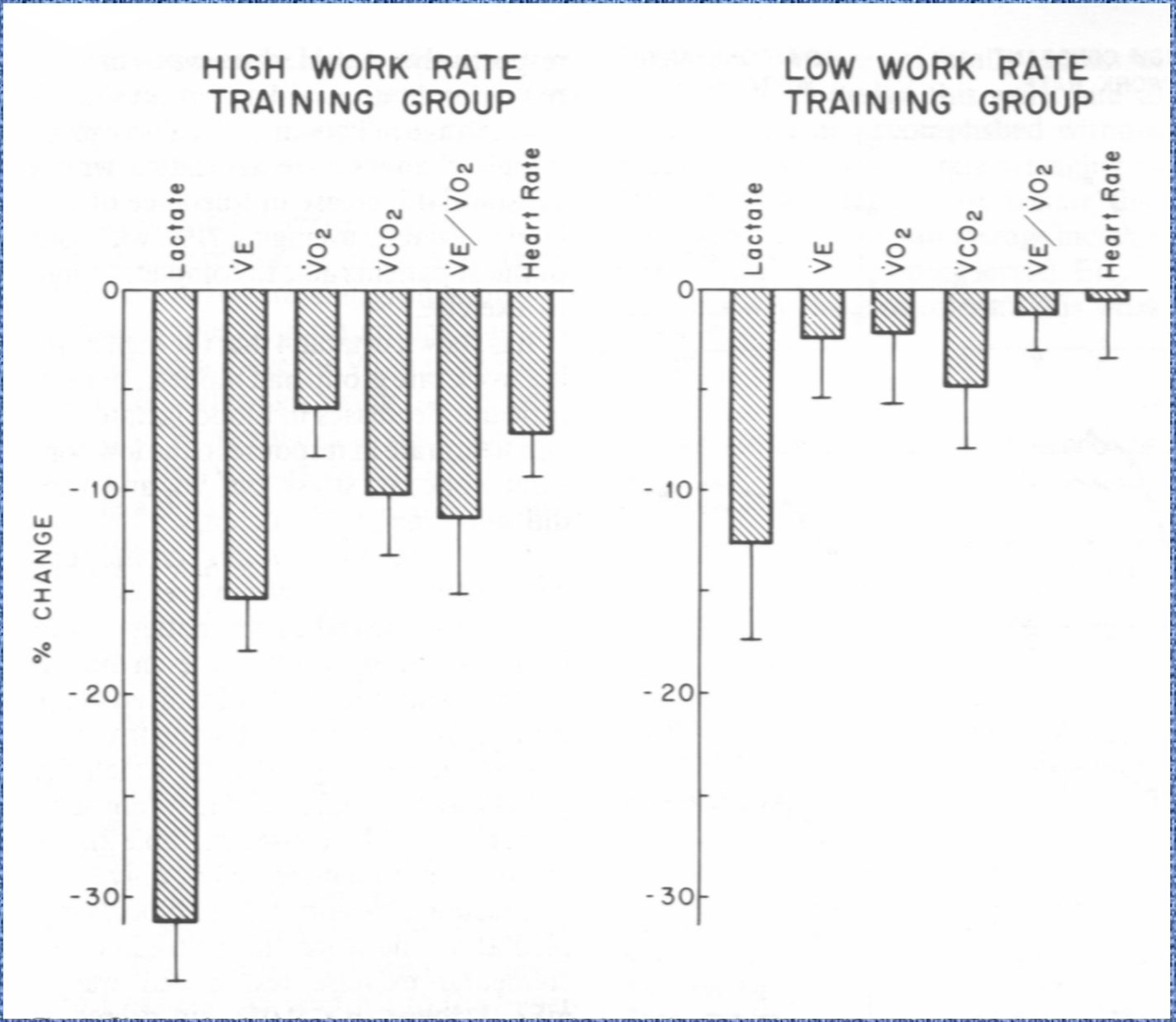


Figure 2. Morphological and structural alterations reported in limb muscles in patients with chronic obstructive pulmonary disease (COPD). CS = citrate synthase; HADH = 3-hydroxyacyl CoA dehydrogenase.

Table 1: Etiologies of Limb Muscle Atrophy, Weakness, and Susceptibility to Fatigue

	Mechanisms Involved
Factors leading to muscle atrophy and weakness	
Disuse	Associated with weakness, atrophy, changes in fiber type distribution, and metabolic alterations (303–306, 310)
Inflammation	Triggering of the muscle proteolysis cascade (102, 116, 322, 325)
Oxidative stress	Triggering of the muscle proteolysis cascade (336, 339, 340) Associated with reduced muscle endurance (222, 227, 229) Protein carbonylation possibly involved in exercise intolerance and weakness (201)
Hypoxemia	Decreased muscle protein synthesis Activation of muscle degradation through hypoxia-inducible factor/von Hippel–Lindau signaling cascade (347–350)
Hypercapnia	Intracellular acidosis/alterations in contractile protein synthesis/degradation (105, 362)
Low levels of anabolic hormones and growth factors	Associated with reduced muscle protein synthesis (371, 372)
Impaired energy balance	Associated with reduced muscle protein synthesis (381, 383)
Corticosteroids	Reduced muscle protein synthesis and enhanced proteolysis through increased myostatin levels and reduced insulin-like growth factor-1 levels (385)
Vitamin D deficiency	Associated with muscle weakness, type II atrophy impaired calcium metabolism (392, 400, 405)
Factors leading to muscle susceptibility to fatigue	
Central fatigue—afferent feedback from limb muscles	Reduced motor output to the contracting muscles (295)
Reduced O_2 delivery (impaired cardiac output, blood flow competition between the respiratory and limb muscle, reduced capillarity)	Changes in muscle metabolism in favor of glycolysis; accumulation of muscle metabolites associated with muscle fatigue
Muscle metabolic alteration (reduced oxidative enzyme activity, reduced mitochondrial function)	Preferential use of glycolysis and accumulation of muscle metabolites associated with muscle fatigue (179, 180, 190, 199, 443)



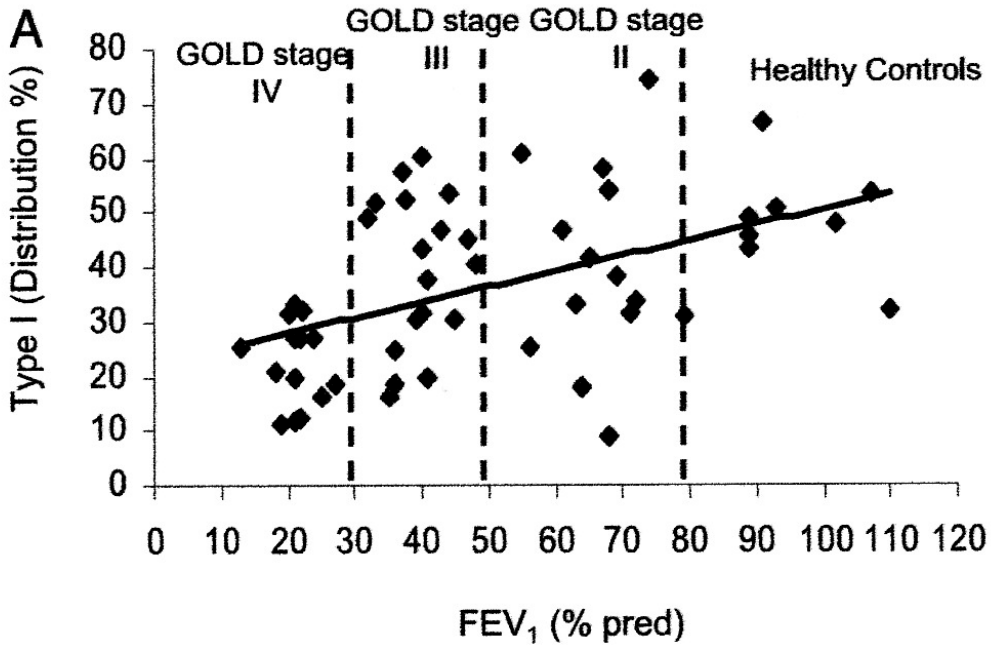
Casaburi et al. ARRD 1991;143:9-18.

Chest 2011; 140: 744-752

CHEST Original Research
PULMONARY REHABILITATION

Effect of Pulmonary Rehabilitation on Peripheral Muscle Fiber Remodeling in Patients With COPD in GOLD Stages II to IV

Ioannis Vogiatzis, PhD; Gerasimos Terzis, PhD; Grigoris Stratakos, MD, FCCP; Evgenia Cherouveim, MSc; Dimitris Athanasopoulos, PhD; Stauroula Spetsioti, MSc; Ioannis Nasis, MSc; Panagiota Manta, MD; Charis Roussos, MD, PhD, FCCP; and Spyros Zakynthinos, MD, PhD



Chest 2011; 140: 744-752

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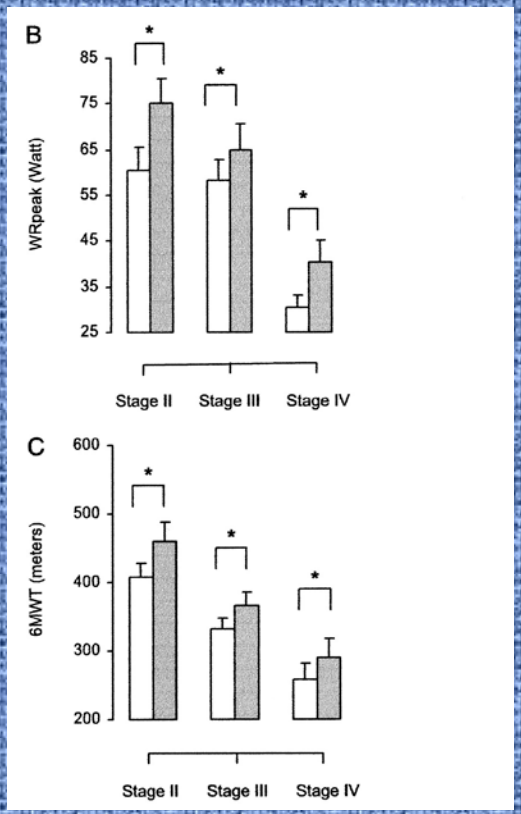

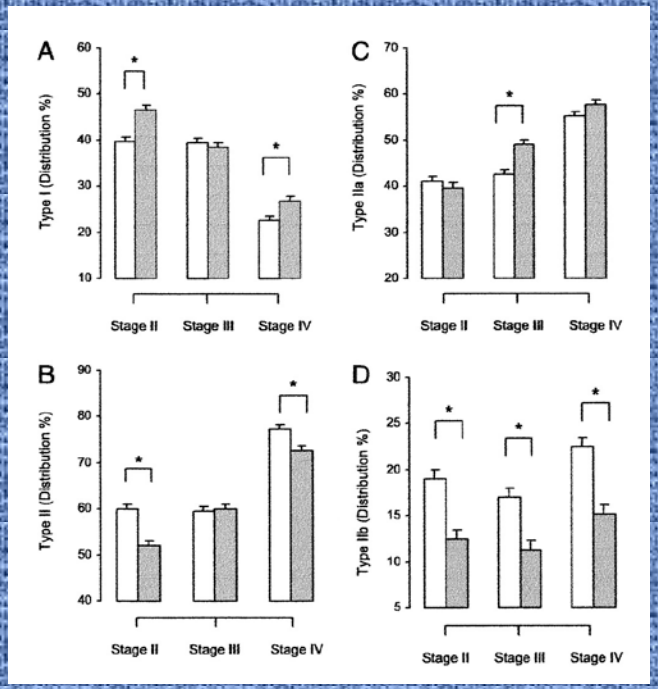

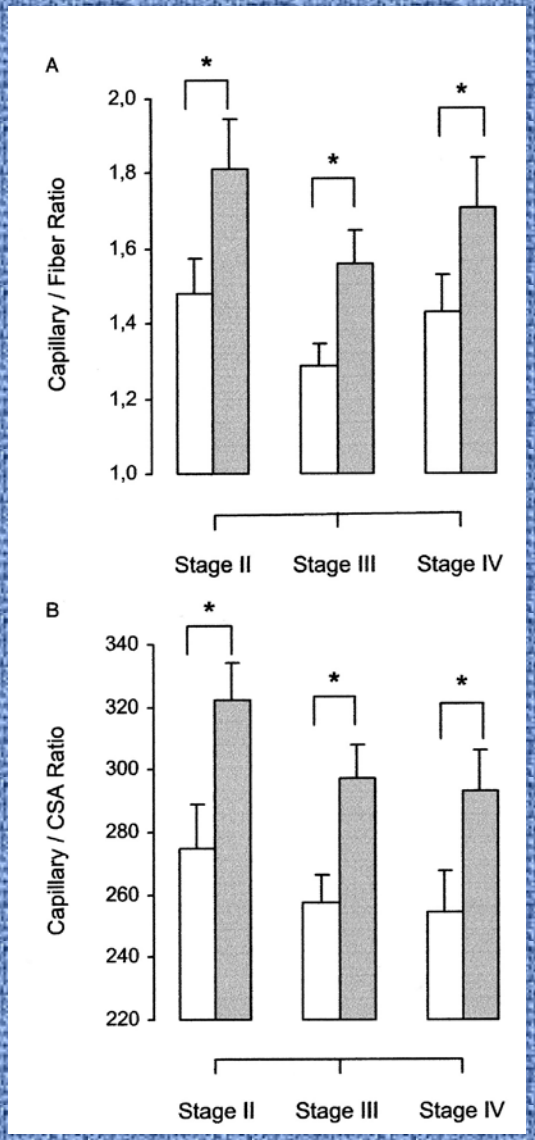

American Thoracic Society Documents

An Official American Thoracic Society/European Respiratory Society Statement: Key Concepts and Advances in Pulmonary Rehabilitation

Martijn A. Spruit, Sally J. Singh, Chris Garvey, Richard ZuWallack, Linda Nici, Carolyn Rochester, Kylie Hill, Anne E. Holland, Suzanne C. Lareau, William D.-C. Man, Fabio Pitta, Louise Sewell, Jonathan Raskin, Jean Bourbeau, Rebecca Crouch, Frits M. E. Franssen, Richard Casaburi, Jan H. Vercoulen, Ioannis Vogiatzis, Rik Gosselink, Enrico M. Clini, Tanja W. Effing, François Maltais, Job van der Palen, Thierry Troosters, Daisy J. A. Janssen, Eileen Collins, Judith Garcia-Aymerich, Dina Brooks, Bonnie F. Fahy, Milo A. Puhan, Martine Hoogendoorn, Rachel Garrod, Annemie M. W. J. Schols, Brian Carlin, Roberto Benzo, Paula Meek, Mike Morgan, Maureen P. M. H. Rutten-van Mölken, Andrew L. Ries, Barry Make, Roger S. Goldstein, Claire A. Dowson, Jan L. Brozek, Claudio F. Donner, and Emiel F. M. Wouters; on behalf of the ATS/ERS Task Force on Pulmonary Rehabilitation

This official statement of the American Thoracic Society (ATS) and the European Respiratory Society (ERS) was approved by the ATS Board of Directors, June 2013, and by the ERS Scientific and Executive Committees in January 2013 and February 2013, respectively

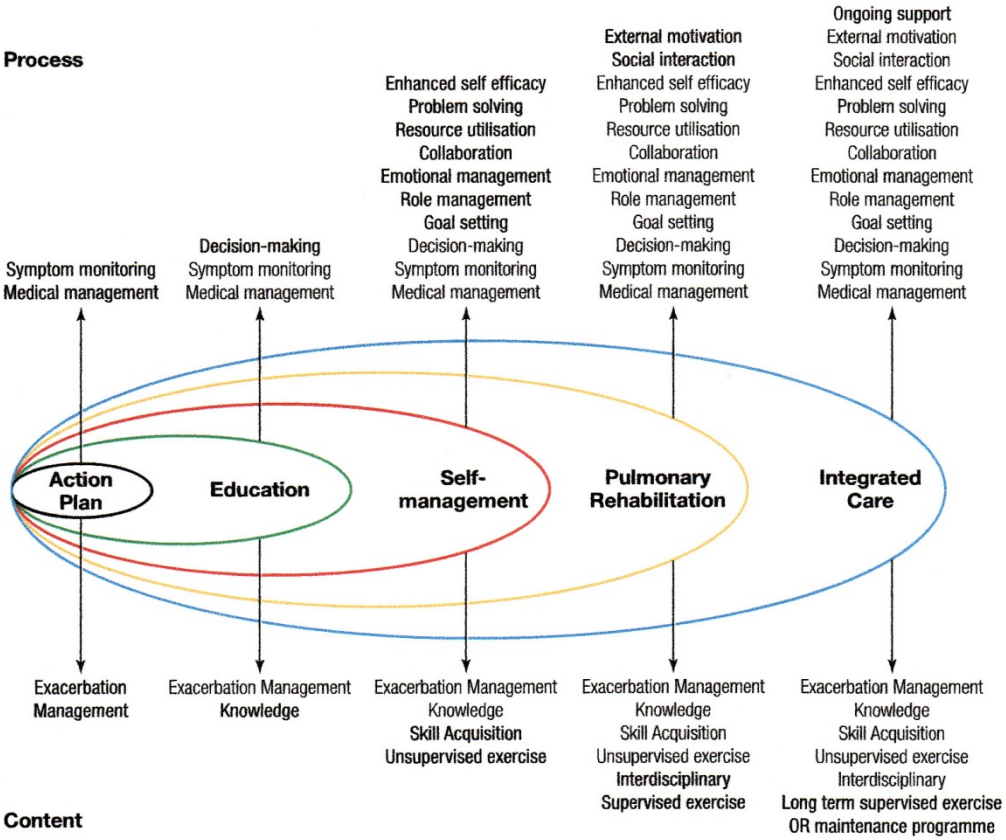

Figure 1. A spectrum of support for chronic obstructive pulmonary disease. Reprinted by permission from Reference 3.

American Thoracic Society Documents

An Official American Thoracic Society/European Respiratory Society Statement: Key Concepts and Advances in Pulmonary Rehabilitation

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THIS OFFICIAL STATEMENT OF THE AMERICAN THORACIC SOCIETY (ATS) AND THE EUROPEAN RESPIRATORY SOCIETY (ERS) WAS APPROVED BY THE ATS BOARD OF DIRECTORS, JUNE 2013, AND BY THE ERS SCIENTIFIC AND EXECUTIVE COMMITTEES IN JANUARY 2013 AND FEBRUARY 2013, RESPECTIVELY

CONTENTS

Am J Respir Crit Care Med Vol 188, Iss. 8, pp e13–e64, Oct 15, 2013

DOI: 10.1164/rccm.201309-1634ST
Internet address: www.atsjournals.org

Lung transplation

- **leads to normalisation of breathing pattern**
- **leads to improved exercise tolerance**
  - **40-60 % of healthy subjects**
    - **peripheral factors impairing adequate oxygen supply to exercising muscles (e.g. microcirculatory or cellular level)?**

      (Ross et al. 1993, Biring et. al. *J. Appl. Physiol.* 84(6): 1967–1975, 1998)
- **studies about effect of exercise training on exercise performance still need to be done**

Pulmonary rehabilitation for chronic obstructive pulmonary disease (Review)

McCarthy B, Casey D, Devane D, Murphy K, Murphy E, Lacasse Y

THE COCHRANE COLLABORATION®

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Pulmonary rehabilitation for chronic obstructive pulmonary disease (Review)
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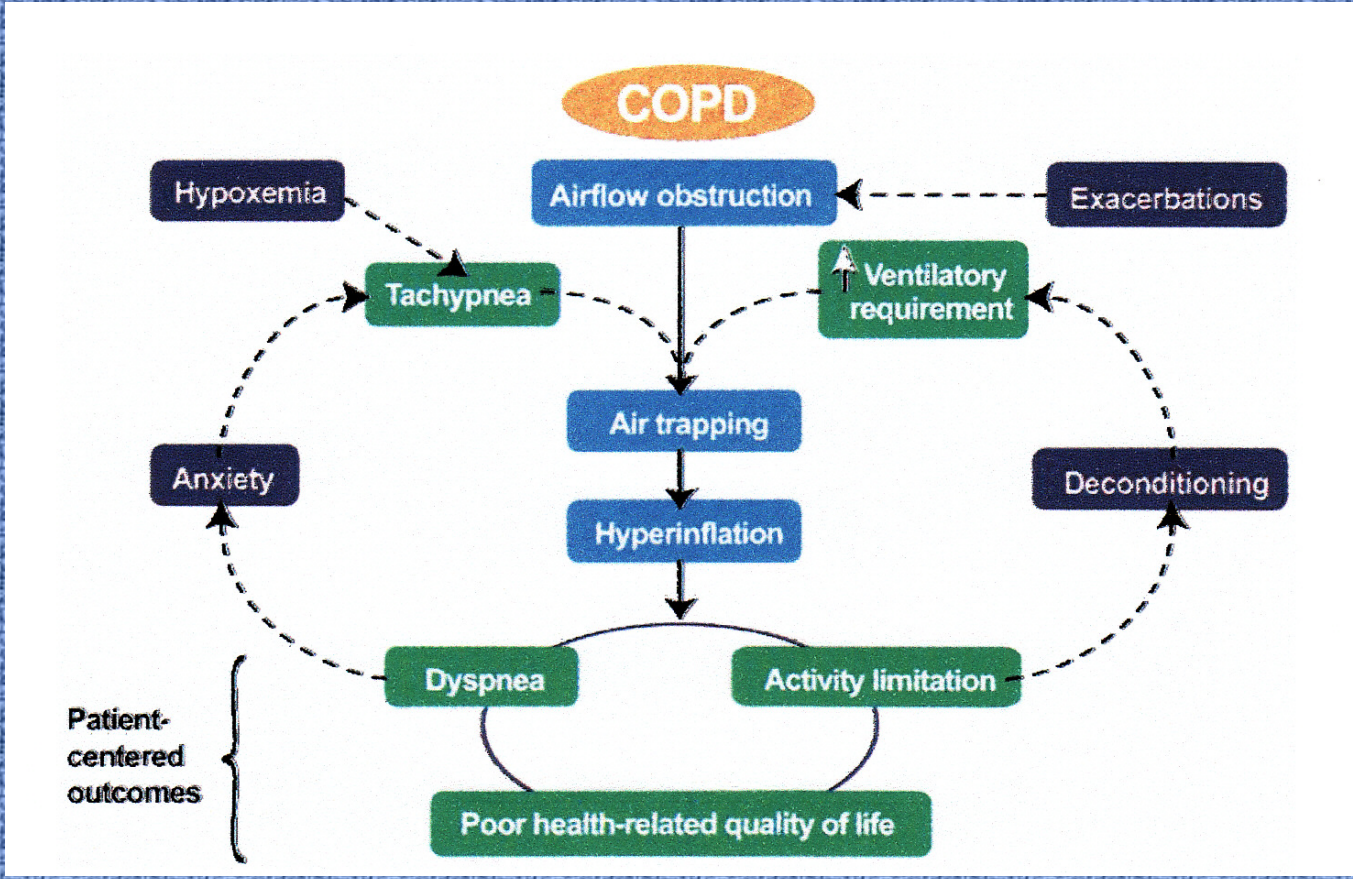
- **65 studi relativi a 3822 pazienti**
- **Efficacia nel migliorare la dispnea, qualità della vita, capacità di esercizio**
- **Non necessari ulteriori studi per confermare l' efficacia della riabilitazione**
- **Ricerche indicate per identificare le componenti essenziali della riabilitazione, la sede e la durata ideale, l' intensità richiesta e la durata dell' effetto**

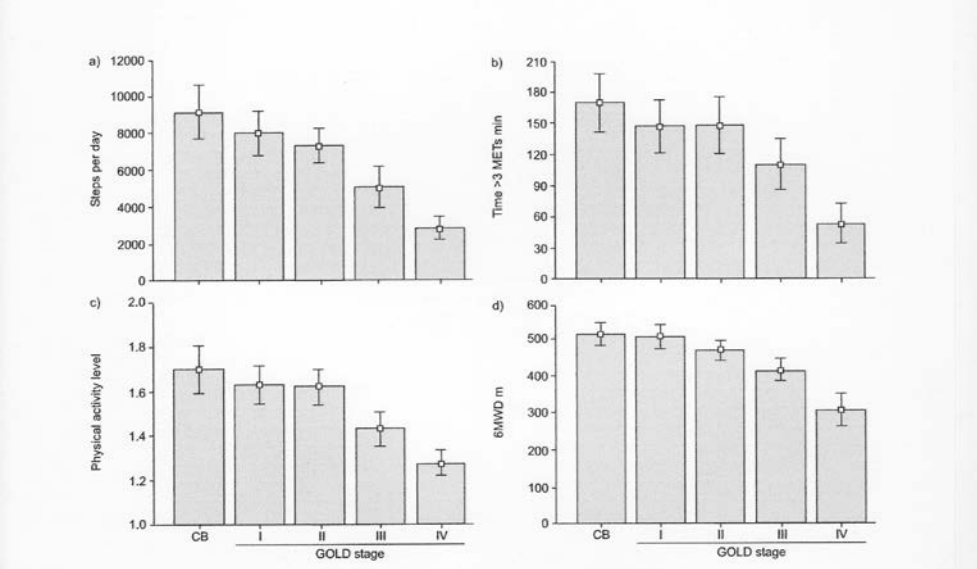
Cooper CB, Am. J. Med.2006; 119: S21-S31



Eur Respir J 2009; 33: 262–272
DOI: 10.1183/09031936.00024608

Physical activity in patients with COPD

H. Watz*, B. Waschki*, T. Meyer# and H. Magnussen*



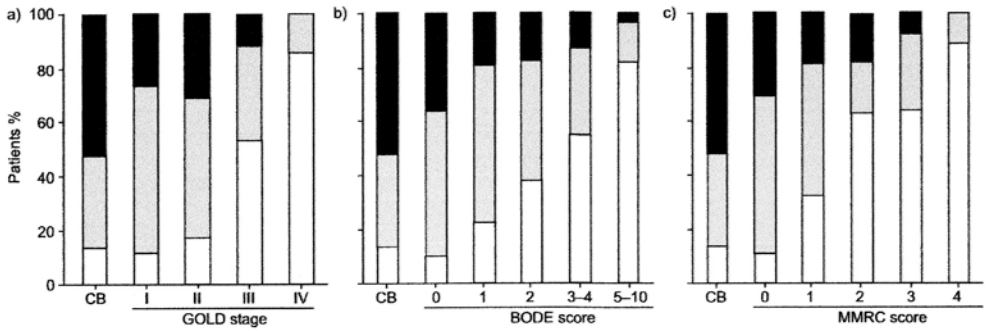


FIGURE 5. Percentage of patients who were active (physical activity level >1.70; ■), predominantly sedentary (physical activity level 1.40–1.69; ▒) or very inactive (physical activity level <1.40; □) according to a) Global Initiative for Chronic Obstructive Lung Disease (GOLD) stages, b) BODE (body mass index, airway obstruction, dyspnoea, exercise capacity) score and c) the modified Medical Research Council (MMRC) dyspnoea scale. CB: chronic bronchitis.

An official European Respiratory Society statement on physical activity in COPD

Henrik Watz[1], Fabio Pitta, Carolyn L. Rochester, Judith Garcia-Aymerich, Richard ZuWallack, Thierry Troosters, Anouk W. Vaes, Milo A. Puhan, Melissa Jehn, Michael I. Polkey, Ioannis Vogiatzis, Enrico M. Clini, Michael Toth, Elena Gimeno-Santos, Benjamin Waschki, Cristobal Esteban, Maurice Hayot, Richard Casaburi, Janos Porszasz, Edward McAuley, Sally J. Singh, Daniel Langer, Emiel F.M. Wouters, Helgo Magnussen and Martijn A. Spruit[1]

Affiliations: For a full list of the authors' affiliations please refer to the Acknowledgements. [1]Task Force co-chairs.

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ABSTRACT This European Respiratory Society (ERS) statement provides a comprehensive overview on physical activity in patients with chronic obstructive pulmonary disease (COPD). A multidisciplinary Task Force of experts representing the ERS Scientific Group 01.02 "Rehabilitation and Chronic Care" determined the overall scope of this statement through consensus. Focused literature reviews were conducted in key topic areas and the final content of this Statement was agreed upon by all members.

The current knowledge regarding physical activity in COPD is presented, including the definition of physical activity, the consequences of physical inactivity on lung function decline and COPD incidence, physical activity assessment, prevalence of physical inactivity in COPD, clinical correlates of physical activity, effects of physical inactivity on hospitalisations and mortality, and treatment strategies to improve physical activity in patients with COPD.

This Task Force identified multiple major areas of research that need to be addressed further in the coming years. These include, but are not limited to, the disease-modifying potential c
activity, and to further understand how improvements in exercise capacity, dyspn
following interventions may translate into increased physical activity.

The Task Force recommends that this ERS statement should be reviewed periodically (

@ERSpublications
An official ERS statement providing a comprehensive overview on physical acti
COPD http://ow.ly/C6v78

Received: March 11 2014 | Accepted after revision: July 29 2014 | First published online: Oct

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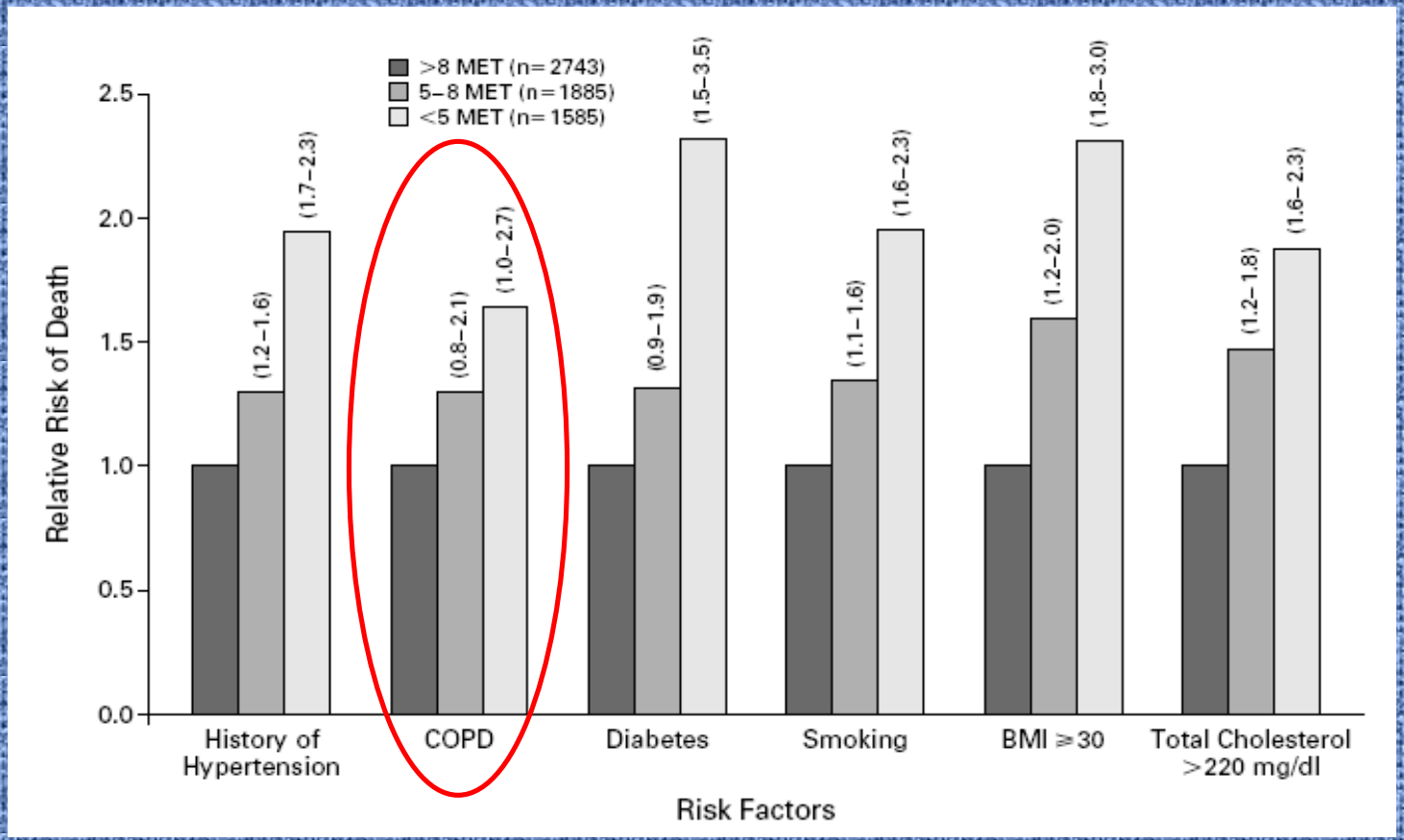
partially funded by Point-One funding from AgentschapNL, Dutch Ministry of Economic af
Benjamin Waschki's contribution to this work was partially funded by the German Center for Lu
The Task Force co-chairs are grateful to the ERS for funding this ERS statement.

Conflict of interest: Disclosures can be found alongside the online version of this article at erj.e

EXERCISE CAPACITY AND MORTALITY AMONG MEN REFERRED FOR EXERCISE TESTING

JONATHAN MYERS, PH.D., MANISH PRAKASH, M.D., VICTOR FROELICHER, M.D., DAT DO, M.D., SARA PARTINGTON, B.SC., AND J. EDWIN ATWOOD, M.D.



N Engl J Med 2002

Invited Review

J Appl Physiol 98: 1154–1162, 2005;
doi:10.1152/japplphysiol.00164.2004.

The anti-inflammatory effect of exercise

Anne Marie W. Petersen and Bente Klarlund Pedersen

Centre of Inflammation and Metabolism at The Copenhagen Muscle Research Centre and The Department of Infectious Diseases, Rigshospitalet, University of Copenhagen, Copenhagen, Denmark

Petersen, Anne Marie W., and Bente Klarlund Pedersen. The anti-inflammatory effect of exercise. *J Appl Physiol* 98: 1154–1162, 2005; doi:10.1152/japplphysiol.00164.2004.—Regular exercise offers protection against all-cause mortality, primarily by protection against cardiovascular disease and Type 2 diabetes mellitus. The latter disorders have been associated with chronic low-grade systemic inflammation reflected by a two- to threefold elevated level of several cytokines. Adipose tissue contributes to the production of TNF-α, which is reflected by elevated levels of soluble TNF-α receptors, IL-6, IL-1 receptor antagonist, and C-reactive protein. We suggest that TNF-α rather than IL-6 is the driver behind insulin resistance and dyslipidemia and that IL-6 is a marker of the metabolic syndrome, rather than a cause. During exercise, IL-6 is produced by muscle fibers via a TNF-independent pathway. IL-6 stimulates the appearance in the circulation of other anti-inflammatory cytokines such as IL-1ra and IL-10 and inhibits the production of the proinflammatory cytokine TNF-α. In addition, IL-6 enhances lipid turnover, stimulating lipolysis as well as fat oxidation. We suggest that regular exercise induces suppression of TNF-α and thereby offers protection against TNF-α-induced insulin resistance. Recently, IL-6 was introduced as the first myokine, defined as a cytokine that is produced and released by contracting skeletal muscle fibers, exerting its effects in other organs of the body. Here we suggest that myokines may be involved in mediating the health-beneficial effects of exercise and that these in particular are involved in the protection against chronic diseases associated with low-grade inflammation such as diabetes and cardiovascular diseases.

cytokines; atherosclerosis; diabetes; aging; physical activity

Clinical SCIENCE

www.clinsci.org

Clinical Science (2010) 118, 565–572 (Printed in Great Britain) doi:10.1042/CS20090458

HYPOTHESIS

Does physical inactivity cause chronic obstructive pulmonary disease?

Nicholas S. HOPKINSON and Michael I. POLKEY

National Heart and Lung Institute, Imperial College, Royal Brompton Hospital, London SW3 6NP, U.K.

ABSTRACT

COPD (chronic obstructive pulmonary disease) is the most common pulmonary disease and is the only common cause of death in which mortality is presently rising. It is caused by the inhalation of smoke, which leads to oxidative stress and inflammation both in the lungs and systemically. Reduced physical activity is a well-recognized consequence of the condition, but we argue here that inactivity is itself an early cause of lung function decline and symptoms. This hypothesis is supported by data from population studies that link activity levels to decline in spirometric indices, both in smokers and non-smokers. In addition, smokers with low physical activity levels are more likely to be diagnosed subsequently with COPD. Physical exercise reduces oxidative stress, has an anti-inflammatory effect and reduces the frequency of upper respiratory tract infections, providing a number of mechanisms by which it could attenuate the harmful effects of smoking. There is sufficient evidence to justify population trials of lifestyle interventions aimed at improving physical activity levels and reducing lung function decline in people diagnosed with early COPD through spirometry screening.

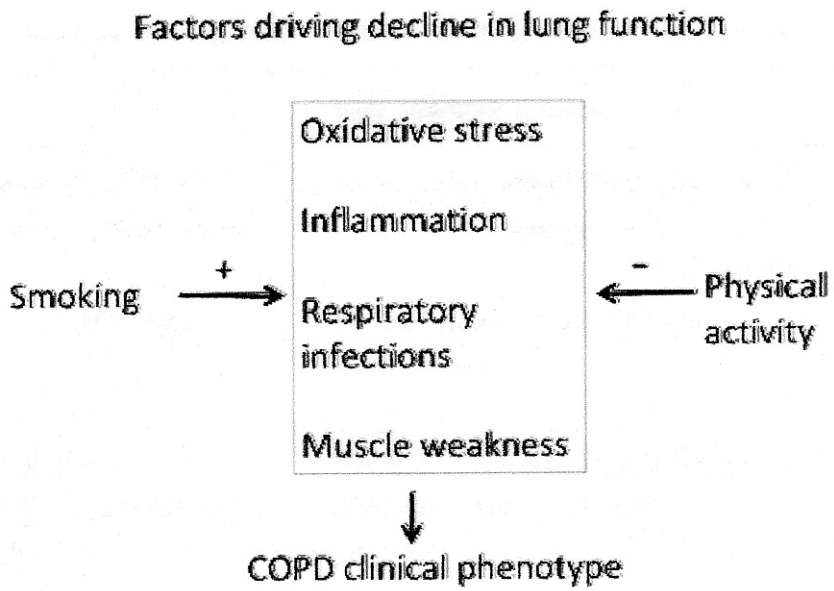


Figure 1 Mechanisms by which physical activity might reduce the impact of smoking on the lungs

Am. J. Respir. Crit. Care Med. 2003; 168: 494-499

Delaying Decline in Pulmonary Function with Physical Activity

A 25-Year Follow-up

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TABLE 3. MEAN ANNUAL DECLINE IN $FEV_{0.75}$ (95% CONFIDENCE INTERVAL) DURING 20 YEARS BY SMOKING CATEGORY AND PHYSICAL ACTIVITY THROUGHOUT 20 YEARS

	Smoking Category											
	All			Never			Quit‡			Continuous		
Tertile of Physical Activity*	n	Decline† (*ml/yr*)	p Value	n	Decline (*ml/yr*)	p Value	n	Decline (*ml/yr*)	p Value	n	Decline (*ml/yr*)	p Value
Low	98	−45.2 (−50.1, −40.3)	Reference	17	−35.4 (−47.0, −23.8)	Reference	47	−46.0 (−53.0, −38.9)	Reference	34	−55.6 (−63.8, −47.4)	Reference
Middle	109	−39.9 (−44.6, −35.3)	0.083	38	−27.7 (−35.4, −19.9)	0.233	50	−44.1 (−50.9, −37.4)	0.831	21	−46.0 (−56.5, −35.6)	0.258
High	68	−34.8 (−40.6, −29.0)	0.009	15	−24.1 (−36.5, −11.6)	0.063	31	−36.1 (−44.8, −27.5)	0.124	22	−44.7 (−54.8, −34.5)	0.162
p for trend	275		0.006	70		0.060	128		0.144	77		0.139

* Tertile limits for physical activity the same as in Table 1.

† In analysis of covariance, the p value for tertile of physical activity was 0.029, for smoking habits was < 0.001, for age was 0.059, for baseline $FEV_{0.75}$ was 0.006, and for tertile of physical activity* smoking habits was 0.895.

‡ Including past smokers at the baseline and smokers who gave up smoking during the 20 years of follow-up.

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Regular Physical Activity Modifies Smoking-related Lung Function Decline and Reduces Risk of Chronic Obstructive Pulmonary Disease

A Population-based Cohort Study

Judith Garcia-Aymerich[1], Peter Lange[2,3], Marta Benet[1], Peter Schnohr[2], and Josep M Antó[1,4]

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TABLE 2. AVERAGE ANNUAL CHANGE IN FEV_1 (ML/YR*) IN THE LOW PHYSICAL ACTIVITY GROUP, AND ADDITIONAL RELATIVE CHANGE† (95% CI) IN THE MODERATE AND HIGH PHYSICAL ACTIVITY GROUPS, ACCORDING TO SMOKING EXPOSURE (LINEAR REGRESSION MODEL‡)

		All Subjects (n = 6,619)§		Never-Smokers (n = 1,572)§		Former Smokers (n = 1,393)§		Active Smokers (n = 3,654)§	
	n§	Coefficient (95% CI)	*P* Value	Coefficient (95% CI)	*P* Value	Coefficient (95% CI)	*P* Value	Coefficient (95% CI)	*P* Value
Physical activity									
Low (reference)	1,035	−17.9		−5.4		−9.9		−20.3	
Moderate	2,418	1.6 (−1.1 to 4.3)	0.237	0.3 (−4.7 to 5.3)	0.899	−2.0 (−8.7 to 4.6)	0.550	2.6 (−1.0 to 6.2)	0.159
High	3,166	3.0 (0.4 to 5.6)	0.026	0.0 (−5.0 to 5.1)	0.988	−1.4 (−7.8 to 5.1)	0.672	4.8 (1.3 to 8.3)	0.008
P for linear trend			0.021		0.960		0.852		0.006

Definition of abbreviation: 95% CI = 95% confidence interval.

* Adjusted mean values based on the linear regression equations. Negative values represent decline.

† Coefficient (and 95% CI) from the linear regression model. Positive values mean yearly gain in milliliters compared with the low physical activity group.

‡ Multivariate models adjusted for sex, age, education, body mass index, weight change during follow-up, ischemic heart disease, dyspnea, sputum, smoking status, smoking duration, alcohol consumption, and FEV_1 at baseline. The complete model (with all covariates) in all subjects is included in the online supplement (*see* Table E1). The model for active smokers is additionally adjusted for tobacco consumption during follow-up (pack-years).

§ Numbers do not add to the total number of subjects due to missing values in some of the variables included in the multivariate models.

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AT A GLANCE COMMENTARY

Scientific Knowledge on the Subject

There are no known modifiable factors—apart from smoking—that may reduce lung function decline. The role of physical activity on COPD development is not known.

What This Study Adds to the Field

Regular physical activity may reduce lung function decline and risk of developing COPD among active smokers.

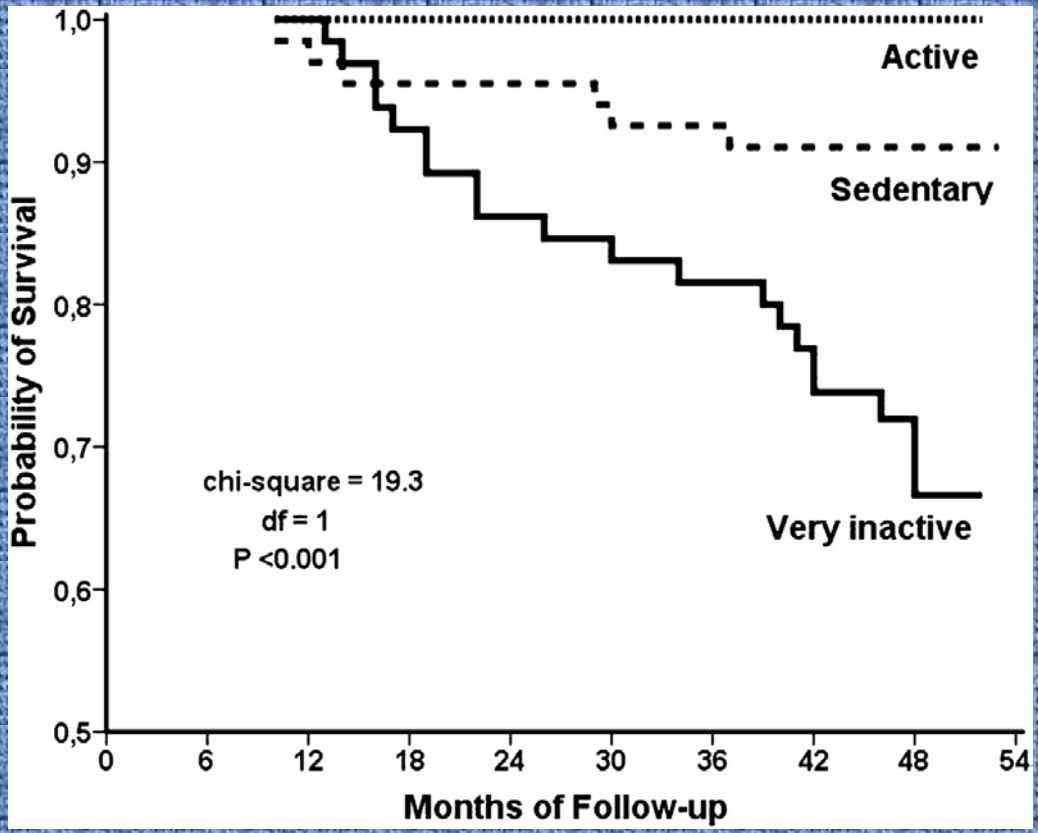
Waschki B et al., Chest 2011; 140: 331-342

CHEST Original Research
COPD

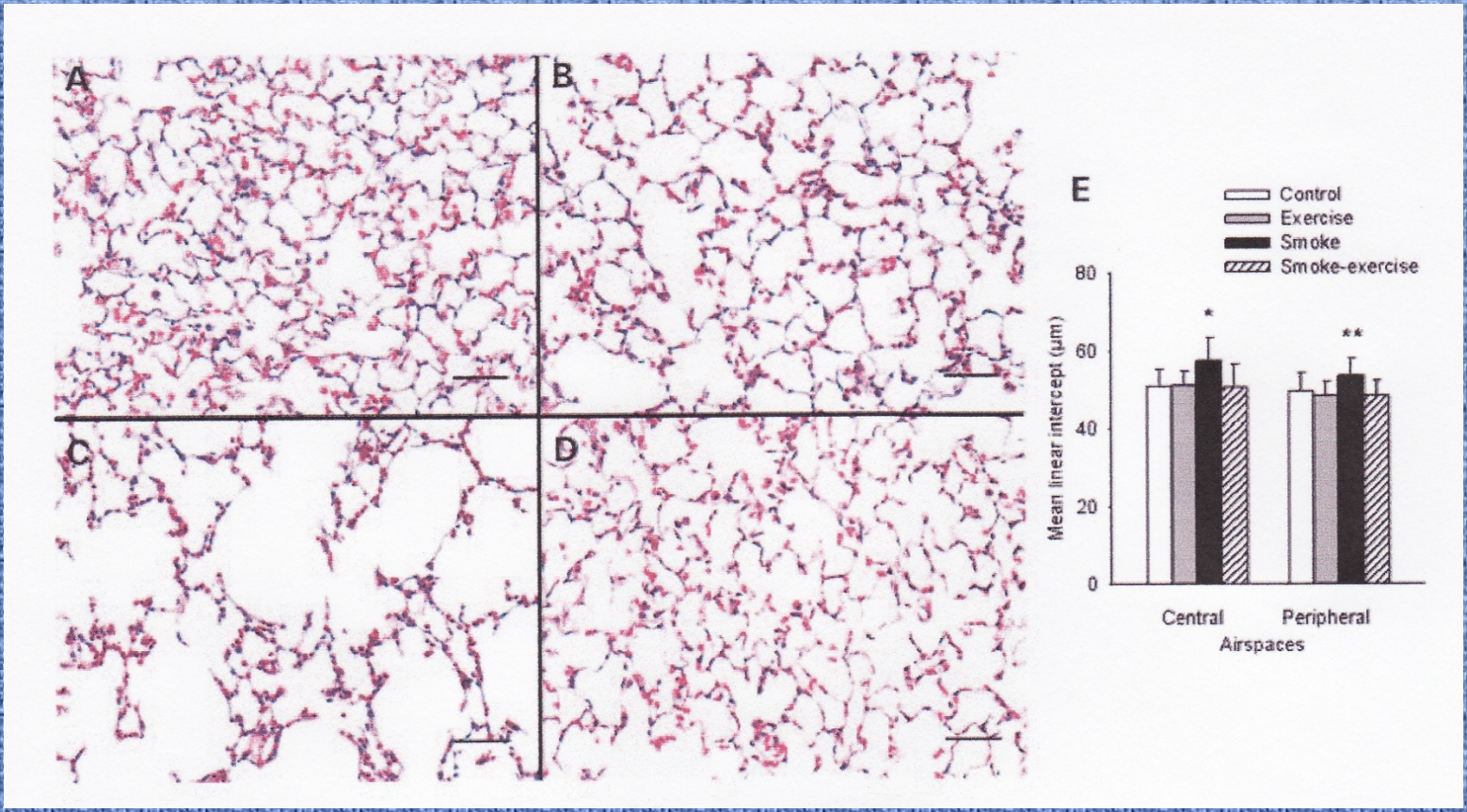
Physical Activity Is the Strongest Predictor of All-Cause Mortality in Patients With COPD

A Prospective Cohort Study

Benjamin Waschki, MD; Anne Kirsten, MD; Olaf Holz, PhD; Kai-Christian Müller, PhD; Thorsten Meyer, PhD; Henrik Watz, MD; and Helgo Magnussen, MD



Aerobic exercise attenuates pulmonary injury induced by exposure to cigarette smoking.
Toledo AC et al ERJ 2012



ATS/ERS Eur. Respir. J. 2004; 23: 932-946

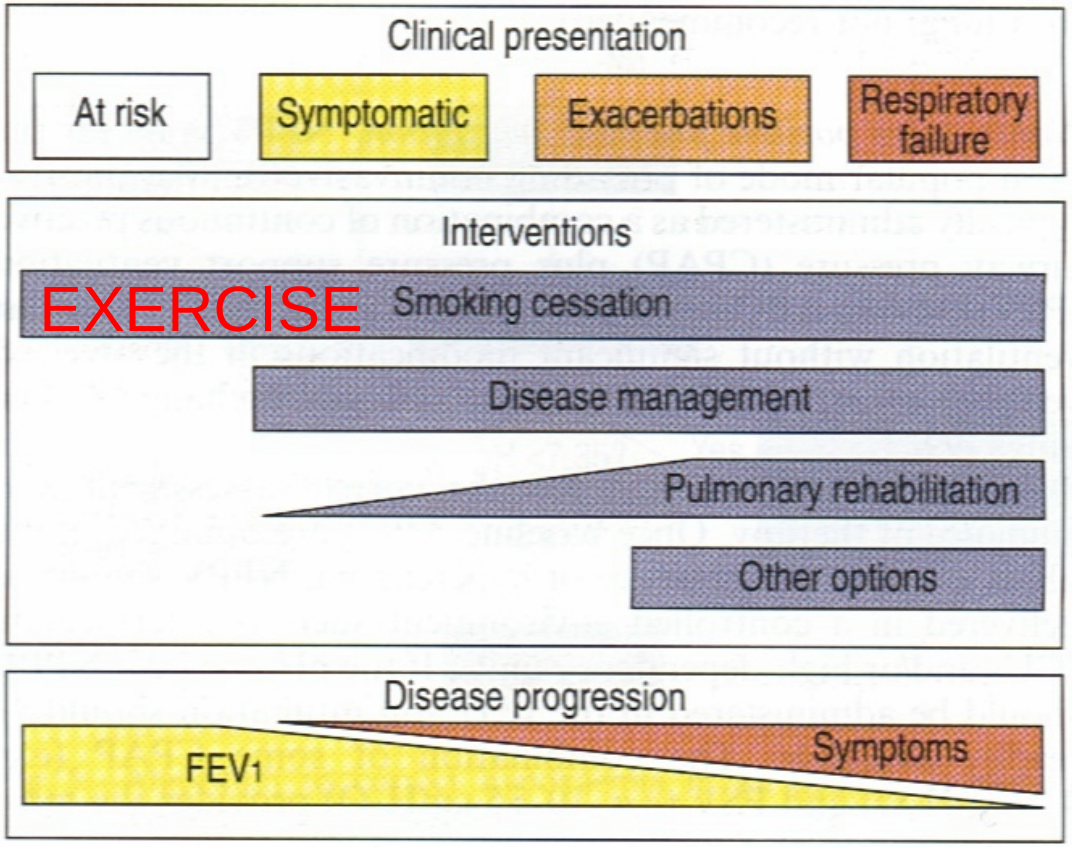


Fig. 7.–Continuum of care for chronic obstructive pulmonary disease (COPD). FEV1: forced expiratory volume in one second.

Dalla Riabilitazione ……alla Sport-Terapia?