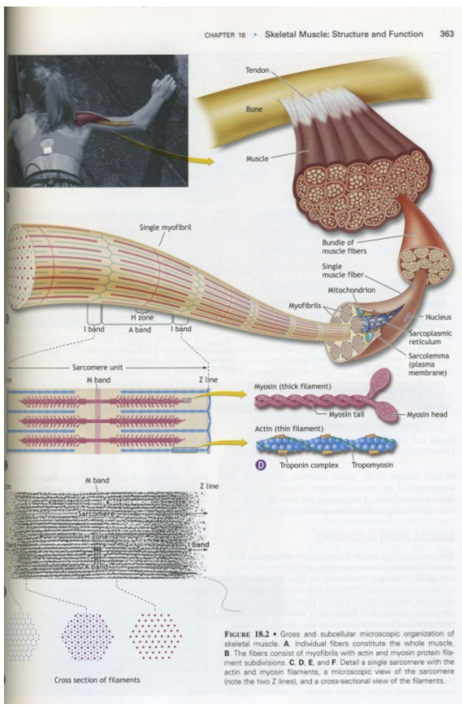
Muskler och träning vid kronisk hjärtsvikt



Åsa Cider

J Cachexia Sarcopenia Muscle (2014) 5:1–3

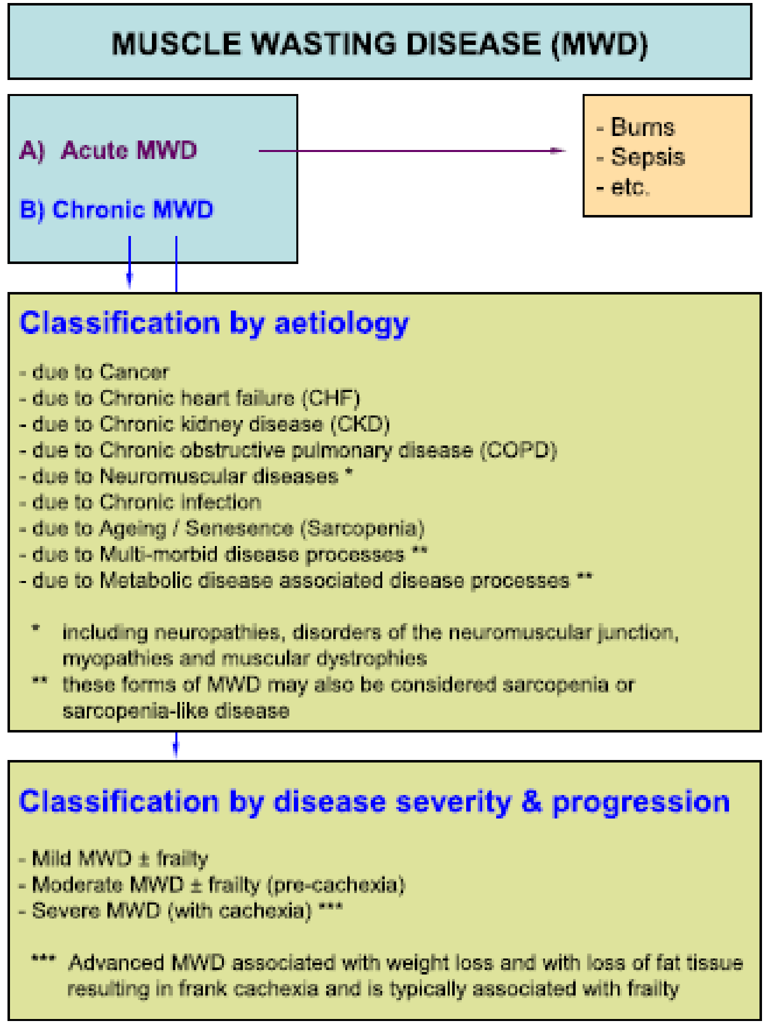


Fig. 1 Framework for the suggested classification of muscle wasting disease by disease etiology and disease progression

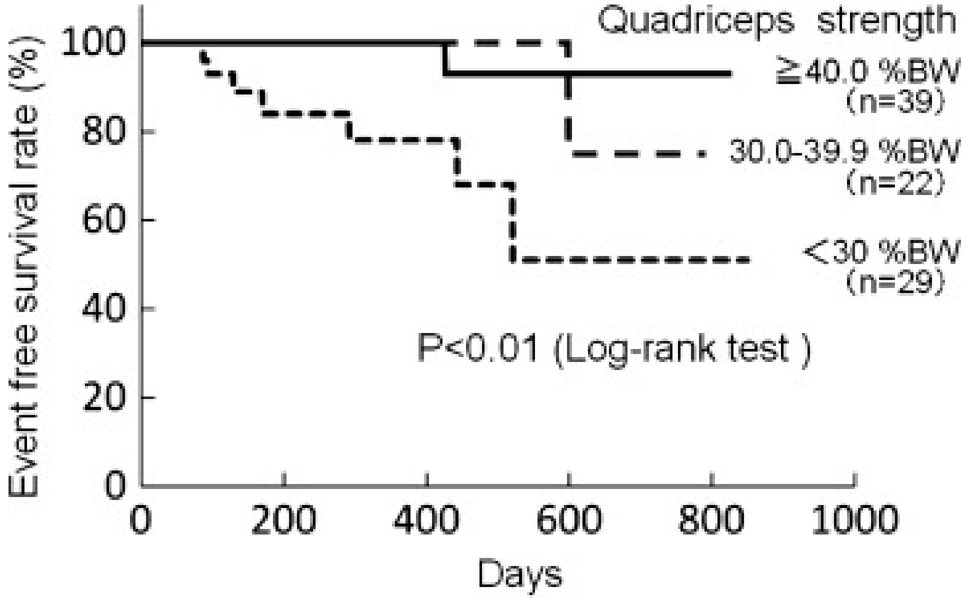


Fig. Kaplan-Meier analysis of the association between quadriceps strength and cardiovascular mortality

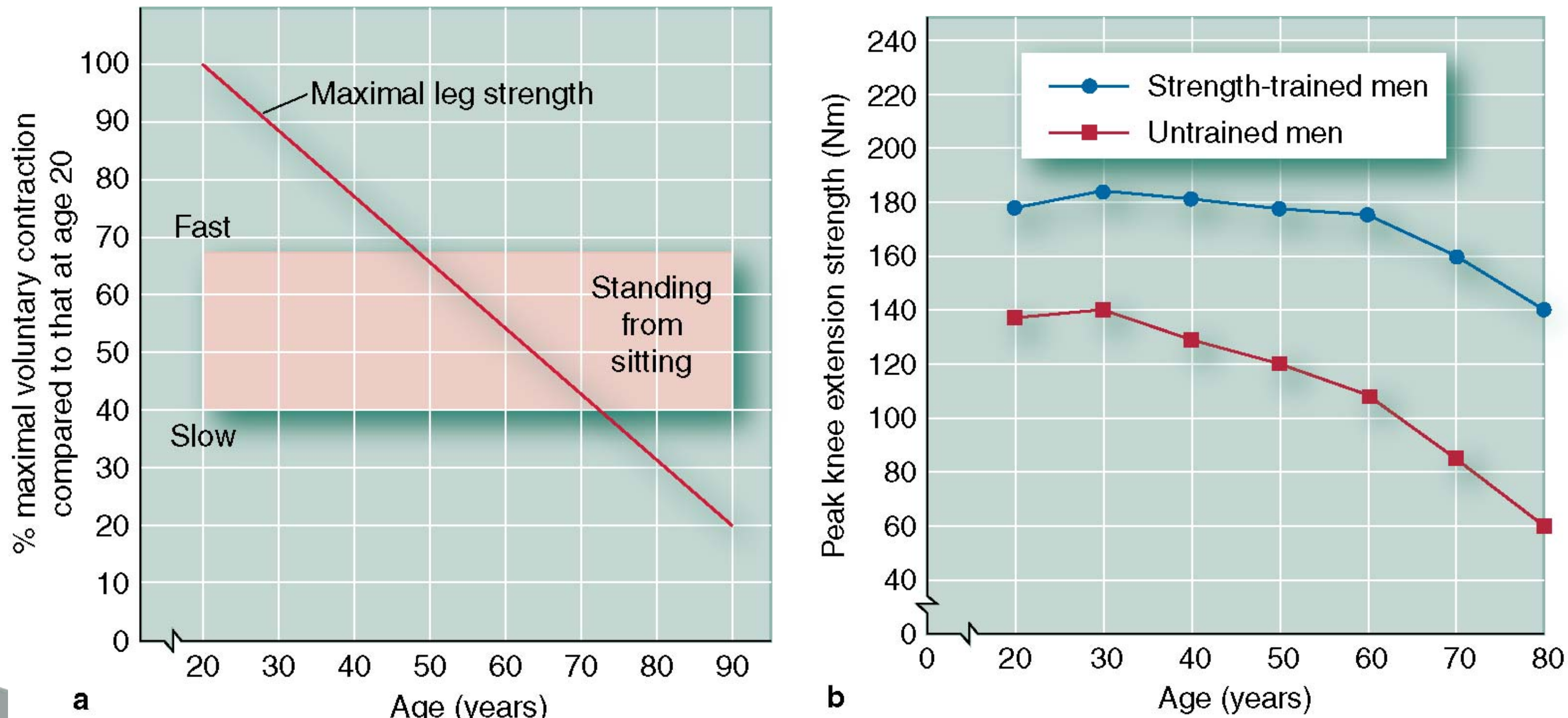
Kentaro Kamiya , Takashi Masuda , Atsuhiko Matsunaga , Yumi Takahashi , Mari Kawano , Misao Ogura , Masahiko Kimu...

Quadriceps Strength of <30%BW Indicates Poor Mortality in Patients With Chronic Heart Failure

Journal of Cardiac Failure, Volume 15, Issue 7, Supplement, 2009, S169

http://dx.doi.org/10.1016/j.cardfail.2009.07.087

(a) Maximal Leg Strength and Speed of Standing and *(b)* Peak Knee Extensor Strength in Strength-Trained and Untrained Men Across the Lifespan



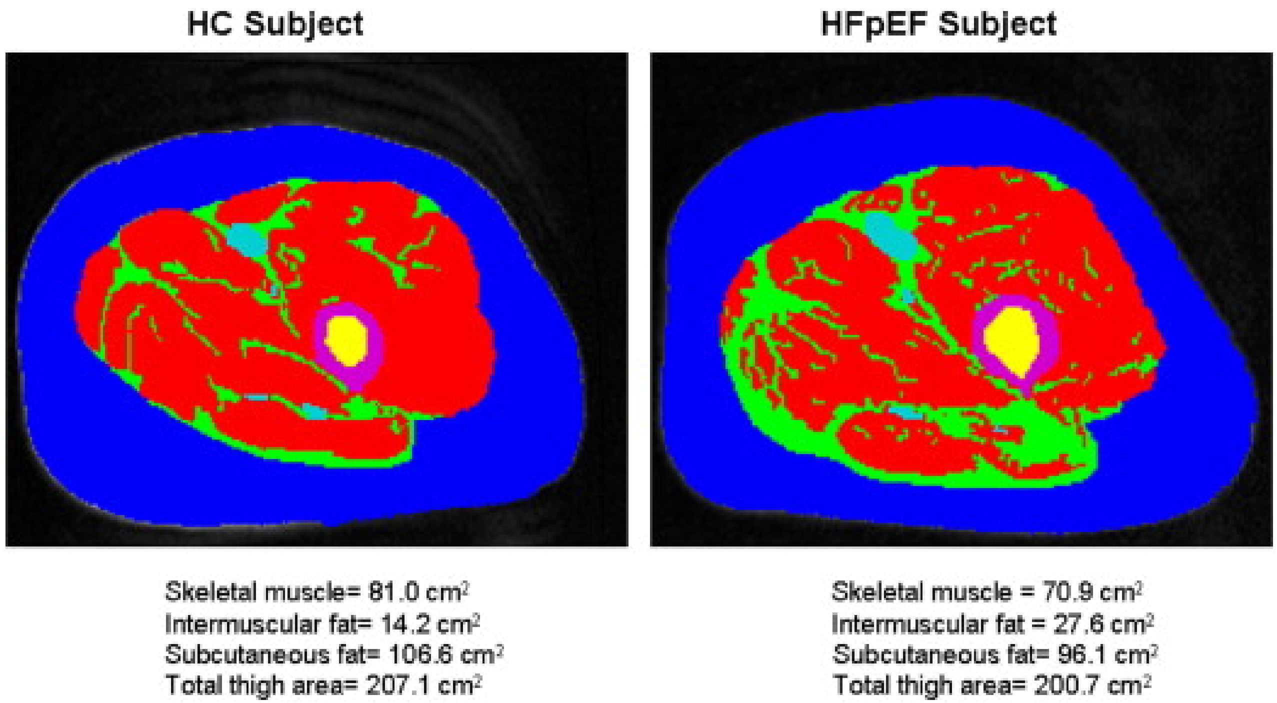


Figure 1 Magnetic resonance imaging axial image of the midthigh in a patient with HFpEF and an HC. Red = SM; green = IMF; blue = SCF; purple = femoral cortex; yellow = femoral medulla. IMF (green) is substantially increased in the patient with ...

Mark J. Haykowsky , Erik J. Kouba , Peter H. Brubaker , Barbara J. Nicklas , Joel Eggebeen , Dalane W. Kitzman

Skeletal Muscle Composition and Its Relation to Exercise Intolerance in Older Patients With Heart Failure and Preserved Ejection Fraction

The American Journal of Cardiology, Volume 113, Issue 7, 2014, 1211 - 1216

http://dx.doi.org/10.1016/j.amjcard.2013.12.031

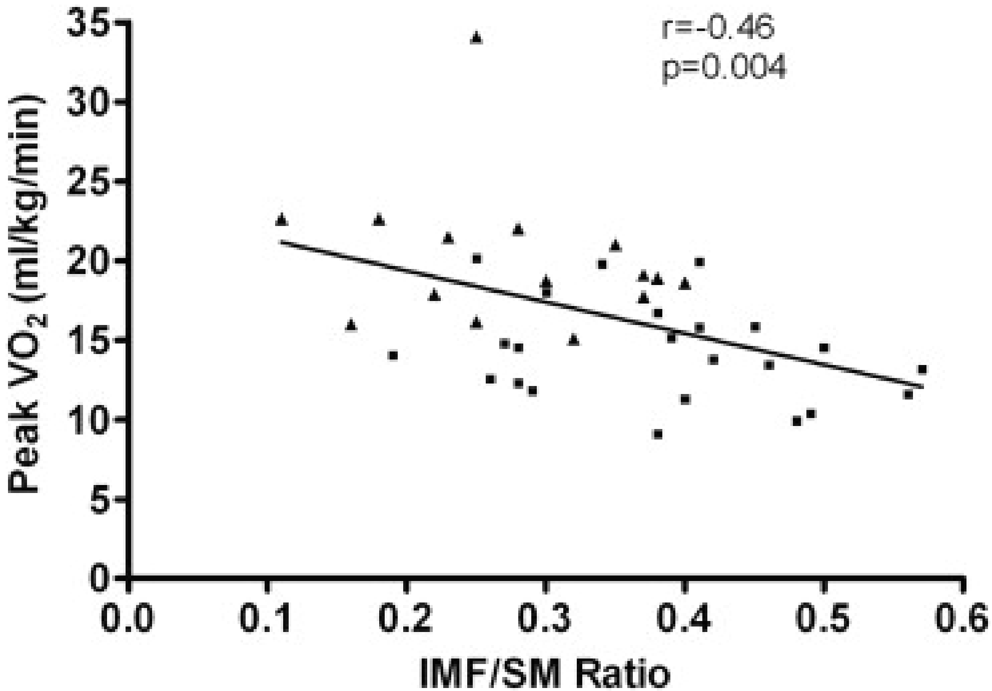


Figure 2 Relation between IMF/SM ratio and peak oxygen uptake in HFpEF and HC groups. Solid squares = HFpEF group; solid triangles = HC group.

Mark J. Haykowsky , Erik J. Kouba , Peter H. Brubaker , Barbara J. Nicklas , Joel Eggebeen , Dalane W. Kitzman

Skeletal Muscle Composition and Its Relation to Exercise Intolerance in Older Patients With Heart Failure and Preserved Ejection Fraction

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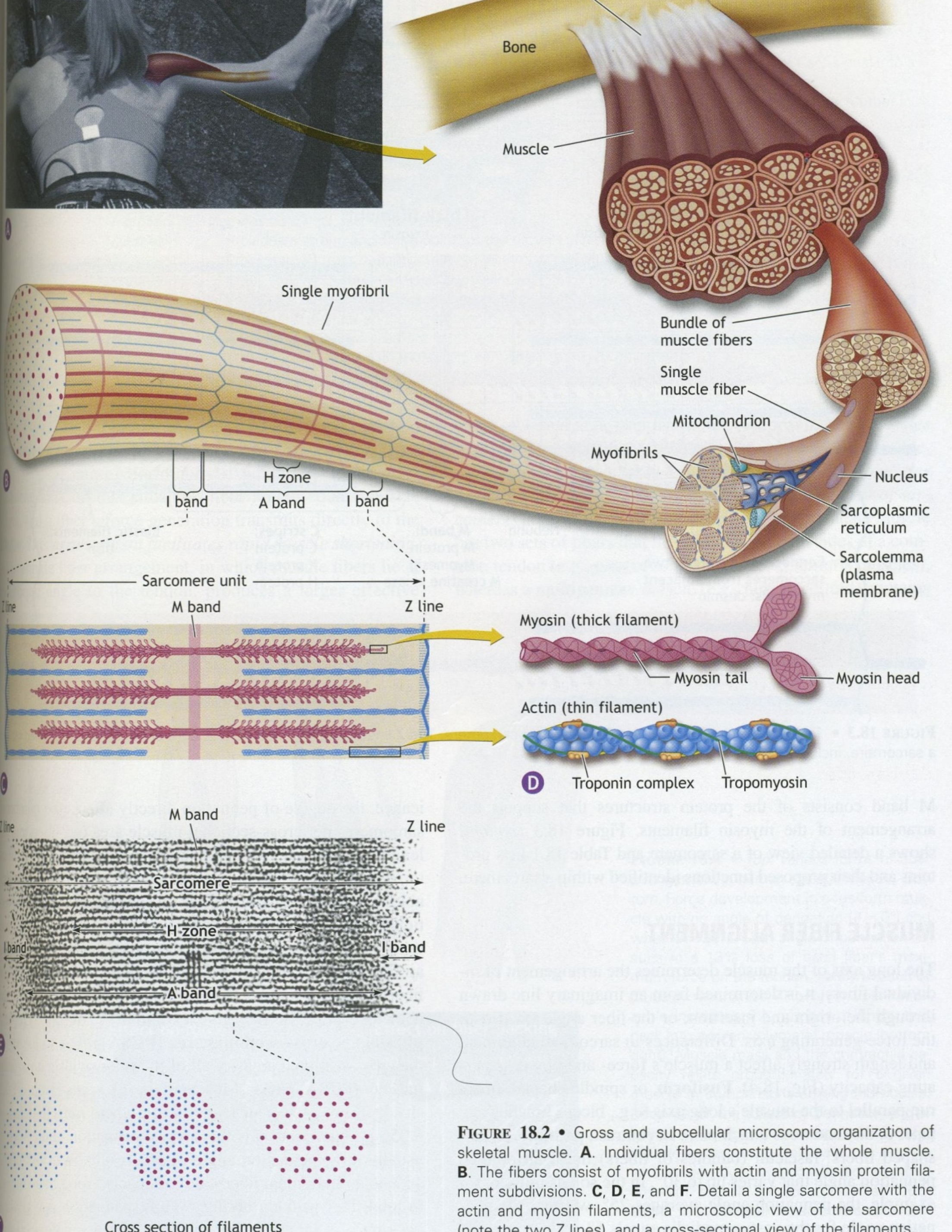


FIGURE 18.2 • Gross and subcellular microscopic organization of skeletal muscle. **A**. Individual fibers constitute the whole muscle. **B**. The fibers consist of myofibrils with actin and myosin protein filament subdivisions. **C**, **D**, **E**, and **F**. Detail a single sarcomere with the actin and myosin filaments, a microscopic view of the sarcomere (note the two Z lines), and a cross-sectional view of the filaments.

Storleksordning, tvärsnitt:

-Hela muskeln: cm

-Bunt av muskelfibrer: mm

-Enskilda muskelfibrer:
µm x 100-1000 (0.1-1 mm)

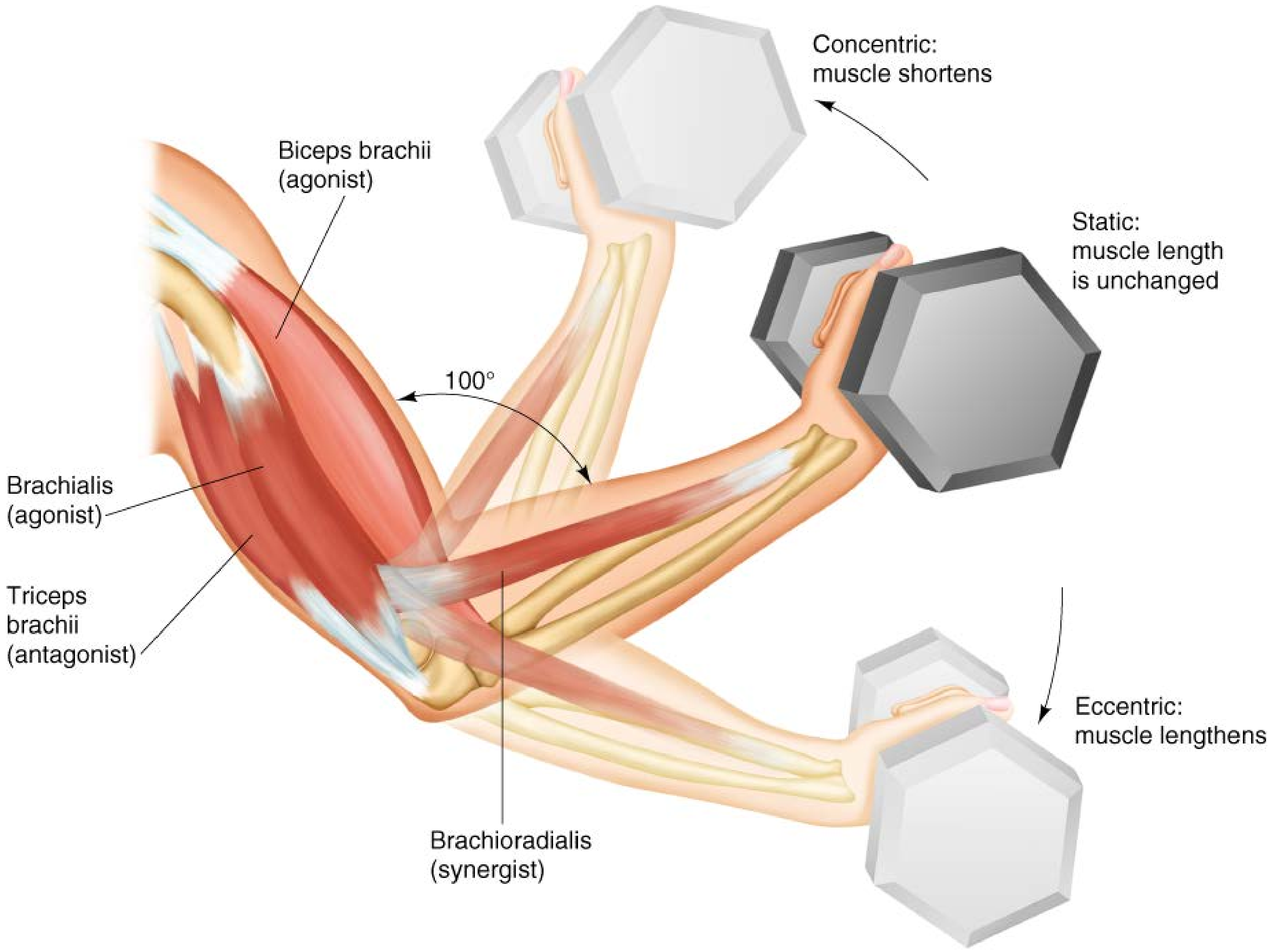
-Myofibriller: µm

-Myofilament (aktin, myosin):
nm (nanometer)

(1nm= 0,000 000 001 m)

GÖTEBORGS UNIVERSITET

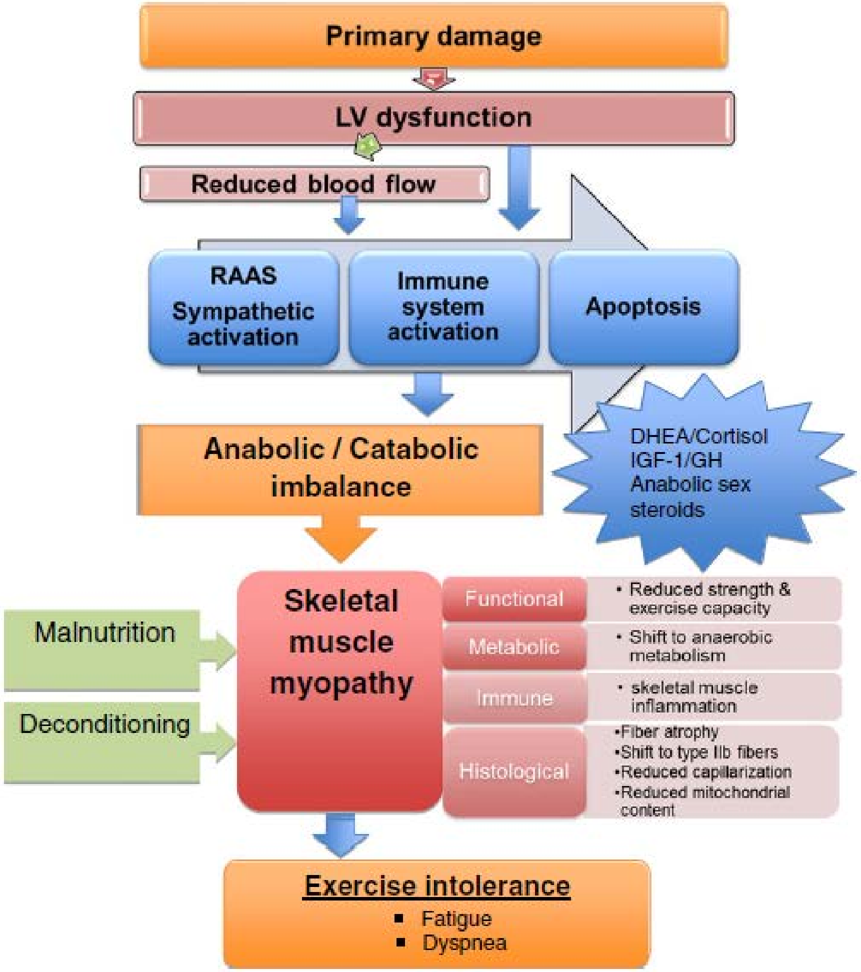
TYPES OF MUSCLE ACTION



Defintioner

- •Inaktivitets hyporofi (atrofi)
- •Sarcopeni
- •Cachexia

Fig. 1 Mechanisms leading from primary myocardial dysfunction to skeletal muscle myopathy and exercise intolerance in CHF



Skeletal muscle loss: cachexia, sarcopenia, and inactivity[1–3]

William J Evans

TABLE 1

Comparison of the metabolic consequences of inactivity/sarcopenia to cachexia

Metabolic condition	Inactivity/ sarcopenia	Cachexia
Muscle protein synthesis	Increased	Increased
Muscle protein degradation	No change	Increased
Muscle mass, strength, and function	Decreased	Decreased
Fat mass	Increased	Decreased
Basal metabolic rate and total energy expenditure	Decreased	Increased
Inflammation	No change	Increased
Insulin resistance	Increased	Increased

Am J Clin Nutr 2010;91 (suppl) 1123S-7S

Skeletal myopathy in patients with chronic heart failure: significance of anabolic-androgenic hormones

Krystian Josiak · Ewa A. Jankowska ·

...hexia Sarcopenia Muscle

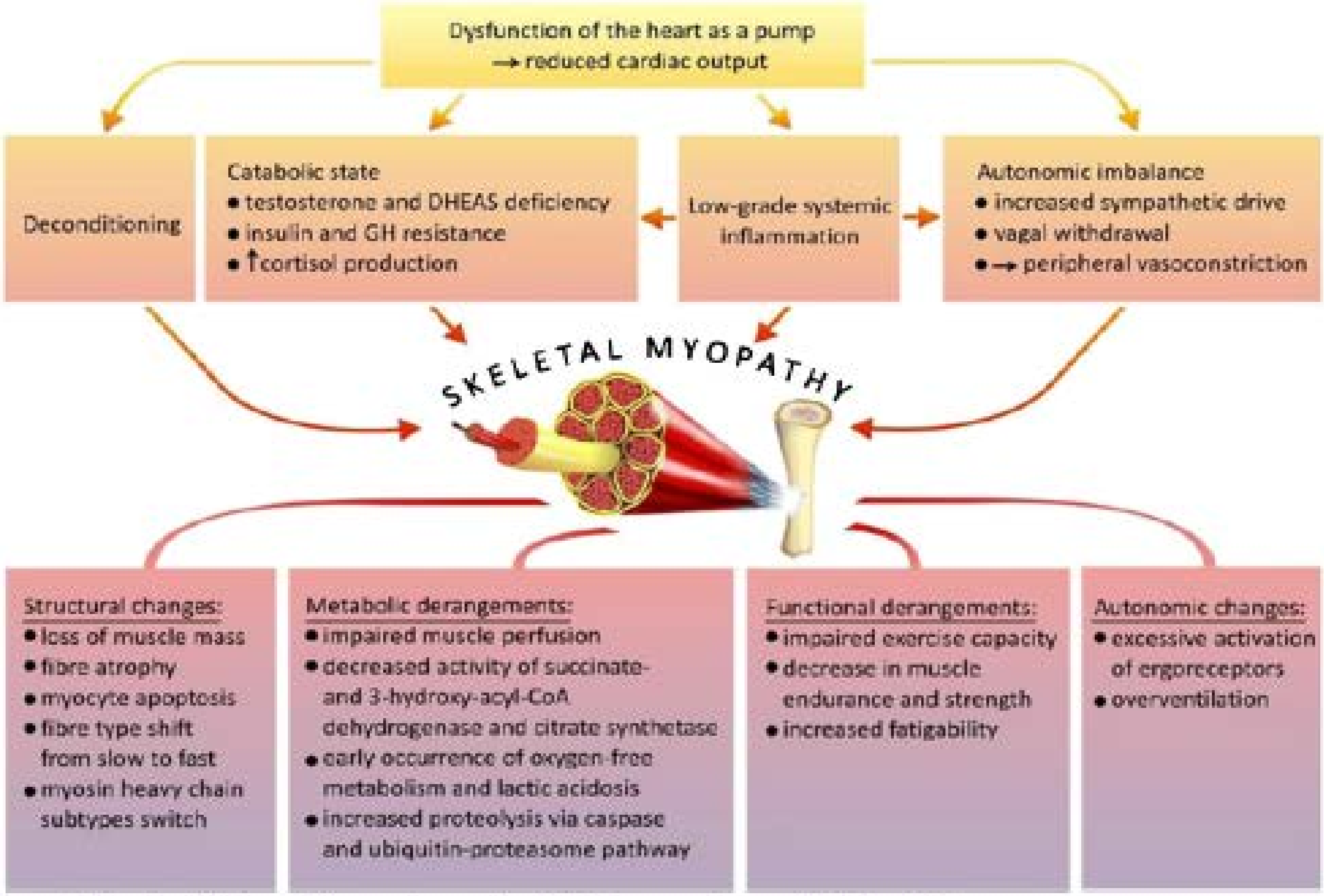


Fig. 1 The muscle hypothesis in heart failure: pathogenesis of skeletal myopathy—modified from [10]

J Cachexia Sarcopenia Muscle
DOI 10.1007/s13539-014-0152-z

Skeletal myopathy in patients with chronic heart failure: significance of anabolic-androgenic hormones

J Cachexia Sarcopen

Krystian Josiak · Ewa A. Jankowska ·

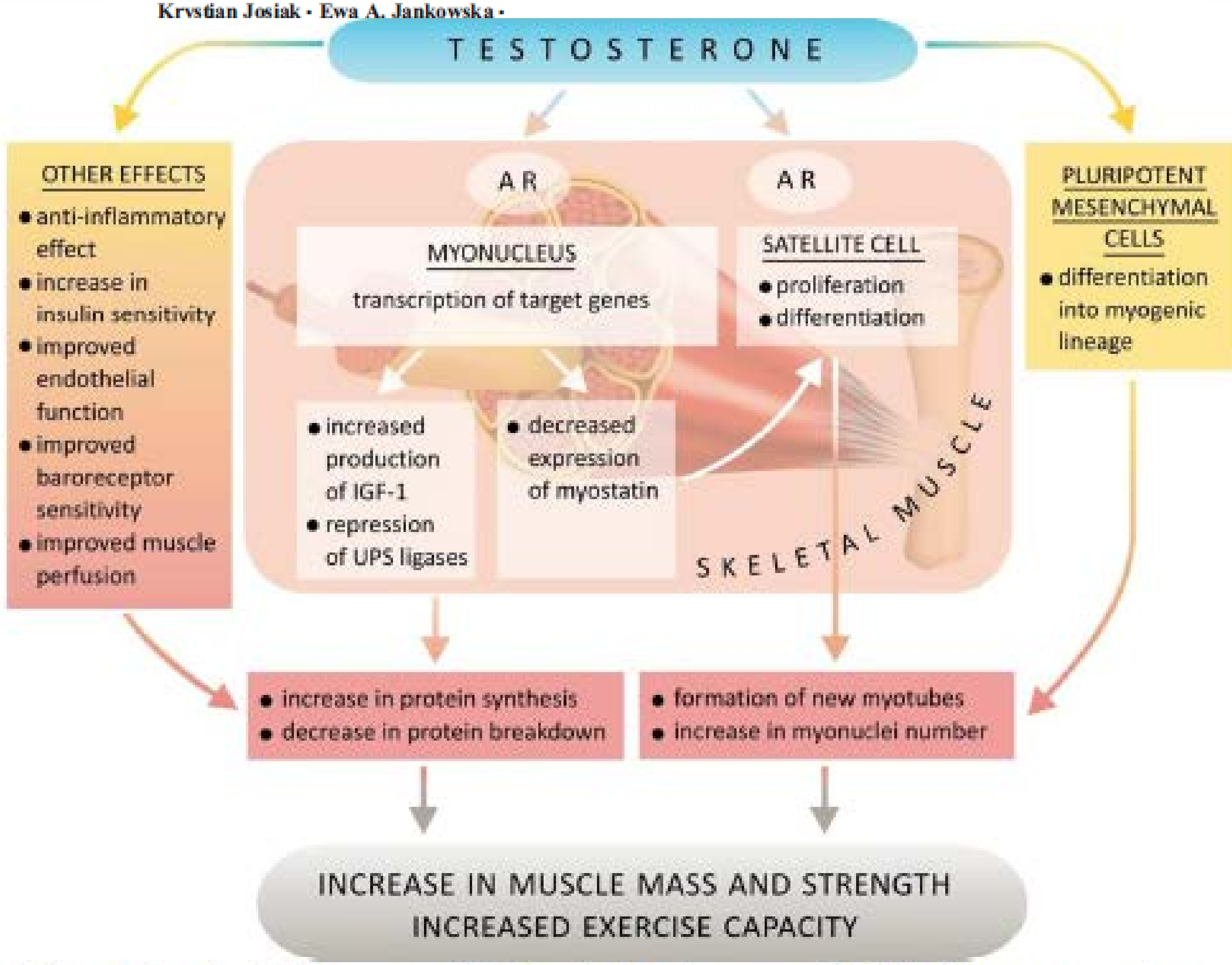


Fig. 2 Mechanisms of action of anabolic hormones on skeletal muscles. *AR* androgen receptor, *UPS* ubiquitin-proteasome system)

Sahlgrenska akademin

J Cachexia Sarcopenia Muscle
DOI 10.1007/s13539-014-0152-z

Hypothalamus And Growth

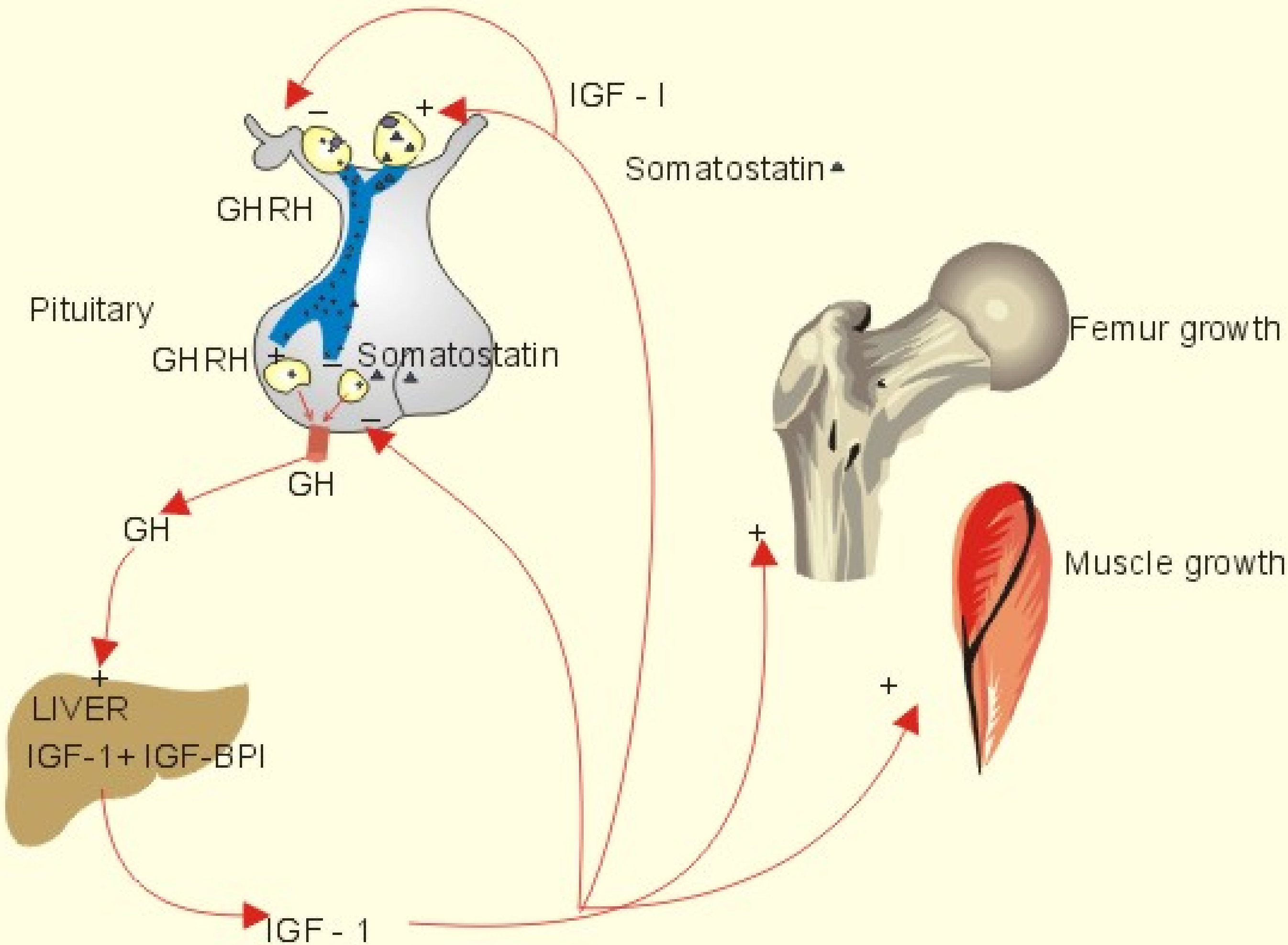


Fig. 30-1

KMc

Effects of the interaction between GH, IGF-1, FFAs and insulin on adipose tissue, liver and skeletal muscle

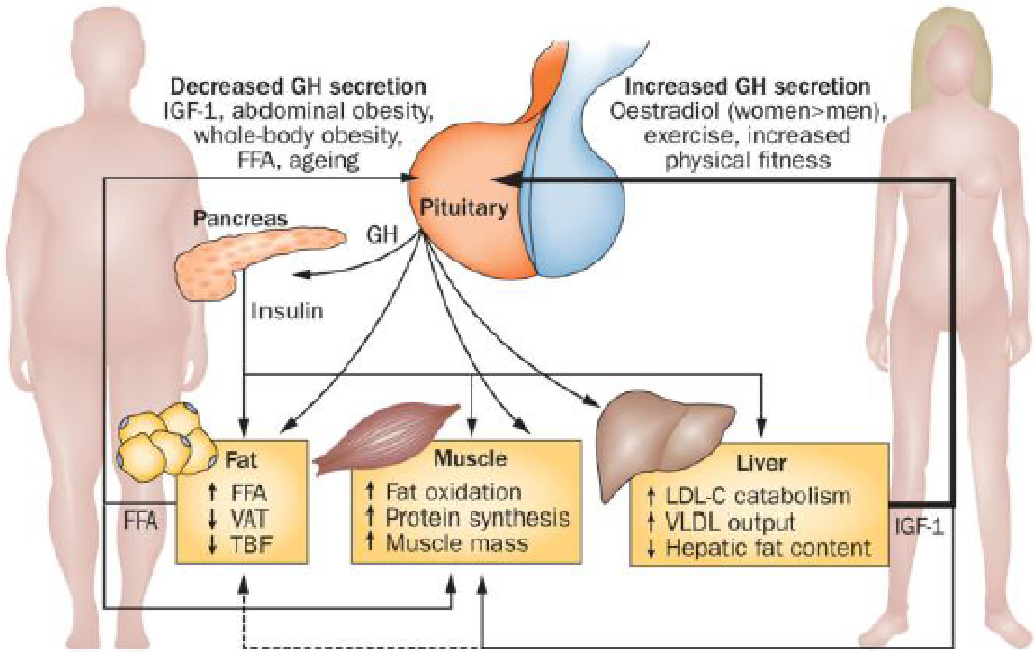


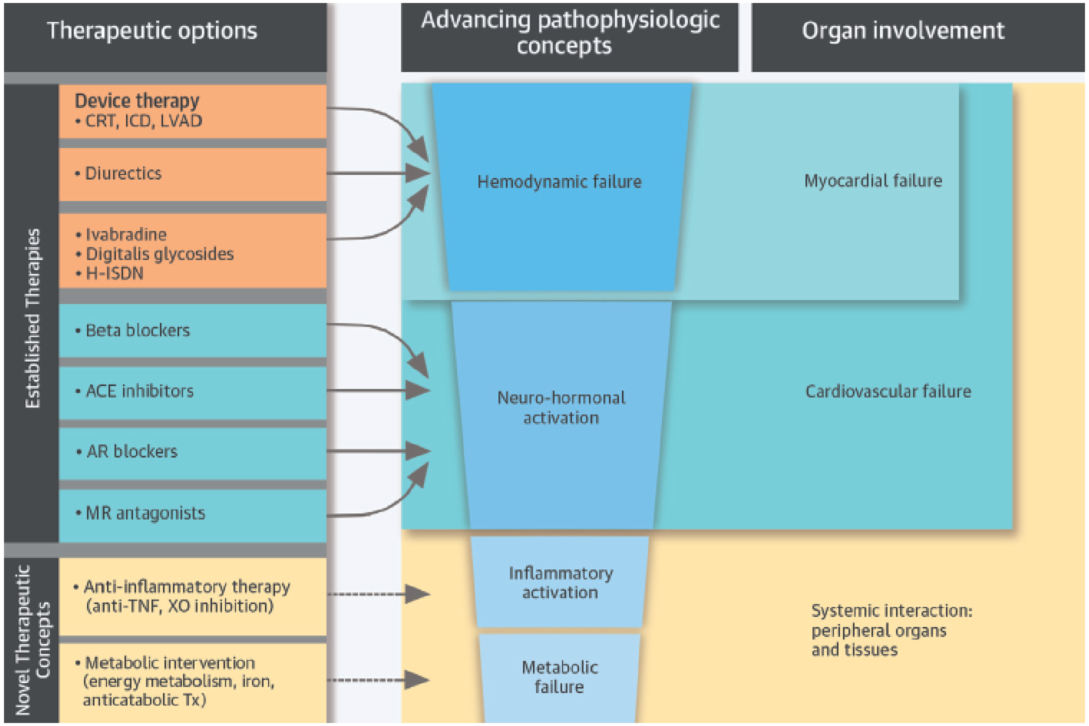
Figure 1

Berryman, D. E. *et al.* (2013) The GH/IGF-1 axis in obesity: pathophysiology and therapeutic considerations *Nat. Rev. Endocrinol.* doi:10.1038/nrendo.2013.64

Metabolic Impairment in Heart Failure

The Myocardial and Systemic Perspective

Wolfram Doehner, MD, PhD,* Michael Frenneaux, MD,† Stefan D. Anker, MD, PhD‡



CENTRAL ILLUSTRATION Evolving Paradigm of HF Pathophysiology

Advancing complexity of heart failure (HF) pathophysiology from a mere hemodynamic disorder to an increasingly systemic involvement of neurohormonal, immune, and metabolic pathways. Current therapeutic concepts focus exclusively on hemodynamic failure and neuroendocrine activation **(left column)**. Novel therapies are warranted to target crucial components of HF pathophysiology, such as metabolic failure and inflammatory activation. ACE = angiotensin-converting enzyme; AT Rec = angiotensin receptor; CRT = cardiac resynchronization therapy; H-ISDN = hydralazine and isosorbide dinitrate; ICD = implantable cardioverter-defibrillator; LVAD = left ventricular assist device; MR = mineralocorticoid receptor; TNF = tumor necrosis factor; Tx = therapy; XO = xanthine oxidase.

JACC VOL. 64, NO. 13, 2014

Akut effekt

Största möjliga kraftinsats under en period mellan 10 s och 2-3 min

Typ I, IIa och IIx får arbeta-

CP och ATP lagren ger energi

Blodådrorna helt öppna mellan kontraktionerna.

Hjärtfrekvens och andningsfrekvens ökar snabbt

Mycket mjölksyra bildas

Kontinuerligt arbete under 3-15 minuter

- Typ I och Typ IIa fibrer
- ATP framställt såväl aerobt som anaerobt

Långvarigt arbete >15 minuter

Typ I fibrer och vid kortare arbete också typ IIa fibrer.

Hjärtats minutvolym strax under det maximala

Received 25 April 2000, accepted in final form 10 January

Pu, Charles T., Meredith T. Johnson, Daniel E. Forman, Jeffrey M. Hausdorff, Ronenn Roubenoff, Mona Foldvari, Roger A. Fielding, and Maria A. Fiatarone Singh. Randomized trial of progressive resistance training to counteract the myopathy of chronic heart failure. *J Appl Physiol* 90: 2341–2350, 2001.—Chronic heart failure (CHF) is characterized by a skeletal muscle myopathy not optimally addressed by current treatment paradigms or aerobic exercise. Sixteen older women with CHF were compared with 80 age-matched peers without CHF and randomized to progressive resistance training or control stretching exercises for 10 wk. Women with CHF had significantly lower muscle strength ($P < 0.0001$) but comparable aerobic capacity to women without CHF. Exercise training was well tolerated and resulted in no changes in resting cardiac indexes in CHF patients. Strength improved by an average of 43.4 ± 8.8% in resistance trainers vs. −1.7 ± 2.8% in controls ($P = 0.001$), muscle endurance by 299 ± 66% vs. 1 ± 3% ($P = 0.001$), and 6-min walk distance by 49 ± 14 m (13%) vs. −3 ± 19 m (−3%) ($P = 0.03$). Increases in type I fiber area (9.5 ± 16%) and citrate synthase activity (35 ± 21%) in skeletal muscle were independently predictive of improved 6-min walk distance ($r^2 = 0.78$; $P = 0.0024$). High-intensity progressive resistance training improves impaired skeletal muscle characteristics and overall exercise performance in older women with CHF. These gains are largely explained by skeletal muscle and not resting cardiac adaptations.

Scand J Med Sci Sports 2014: 24: e1–e10
doi: 10.1111/sms.12123

SCANDINAVIAN JOURNAL OF
MEDICINE & SCIENCE
IN SPORTS

Review

Responsiveness of muscle size and strength to physical training in very elderly people: A systematic review

Stewart et al.

SMD = 0.2; medium, SMD = 0.5; and large, SMD = 0.8.

V. H. Stewart[1], D. H. Saunders[2], C. A. Greig[1]

	Experimental			Control				Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Fixed, 95% CI
Fiatarone et al. 1994	0.9	8.5	25	−0.4	9.68	26	11.5%	1.30 [−3.69, 6.29]
Goodpaster et al. 2008	−2.8	4.22	22	−3.9	4.02	20	46.2%	1.10 [−1.39, 3.59]
Sipilä et al. 1995	1.5	2.36	12	−2.4	3.78	11	42.3%	3.90 [1.30, 6.50]
Total (95% CI)			**59**			**57**	**100.0%**	**2.31 [0.62, 4.00]**

Heterogeneity: Chi² = 2.50, df = 2 (P = 0.29); I² = 20%
Test for overall effect: Z = 2.67 (P = 0.008)

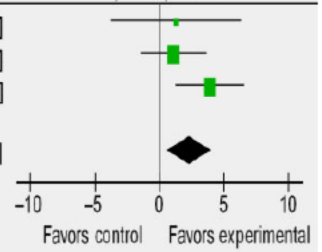


Fig. 2. Thigh muscle cross sectional area. Forest plot illustrating the mean difference (MD) in thigh muscle cross-sectional area between the intervention and control group of the three appropriate studies. The forest plot illustrates the MD of each individual study, shown as filled square symbols centered on the MD with extending horizontal lines indicating the 95% confidence intervals (CI). The different sized boxes represent the weight given to the study based on its standard deviations and number of participants. The overall MD is presented as a filled diamond whose extremities show the 95% CI.

	Experimental			Control				Standard Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Fixed, 95% CI
Fiatarone et al. 1994	4.9	3	25	0.1	3.06	26	37.8%	1.56 [0.93, 2.19]
Goodpaster et al. 2008	−1.07	18.76	22	−15.4	22.36	20	38.8%	0.68 [0.06, 1.31]
Machado et al. 2010	350	579.18	13	10	16.5482	13	23.4%	0.80 [−0.00, 1.61]
Total (95% CI)			**60**			**59**	**100.0%**	**1.04 [0.65, 1.43]**

Heterogeneity: Chi² = 4.16, df = 2 (P = 0.12); I² = 52%
Test for overall effect: Z = 5.25 (P < 0.00001)

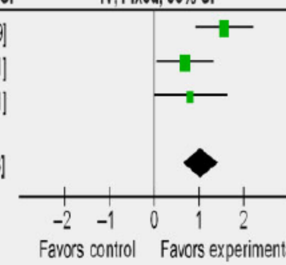
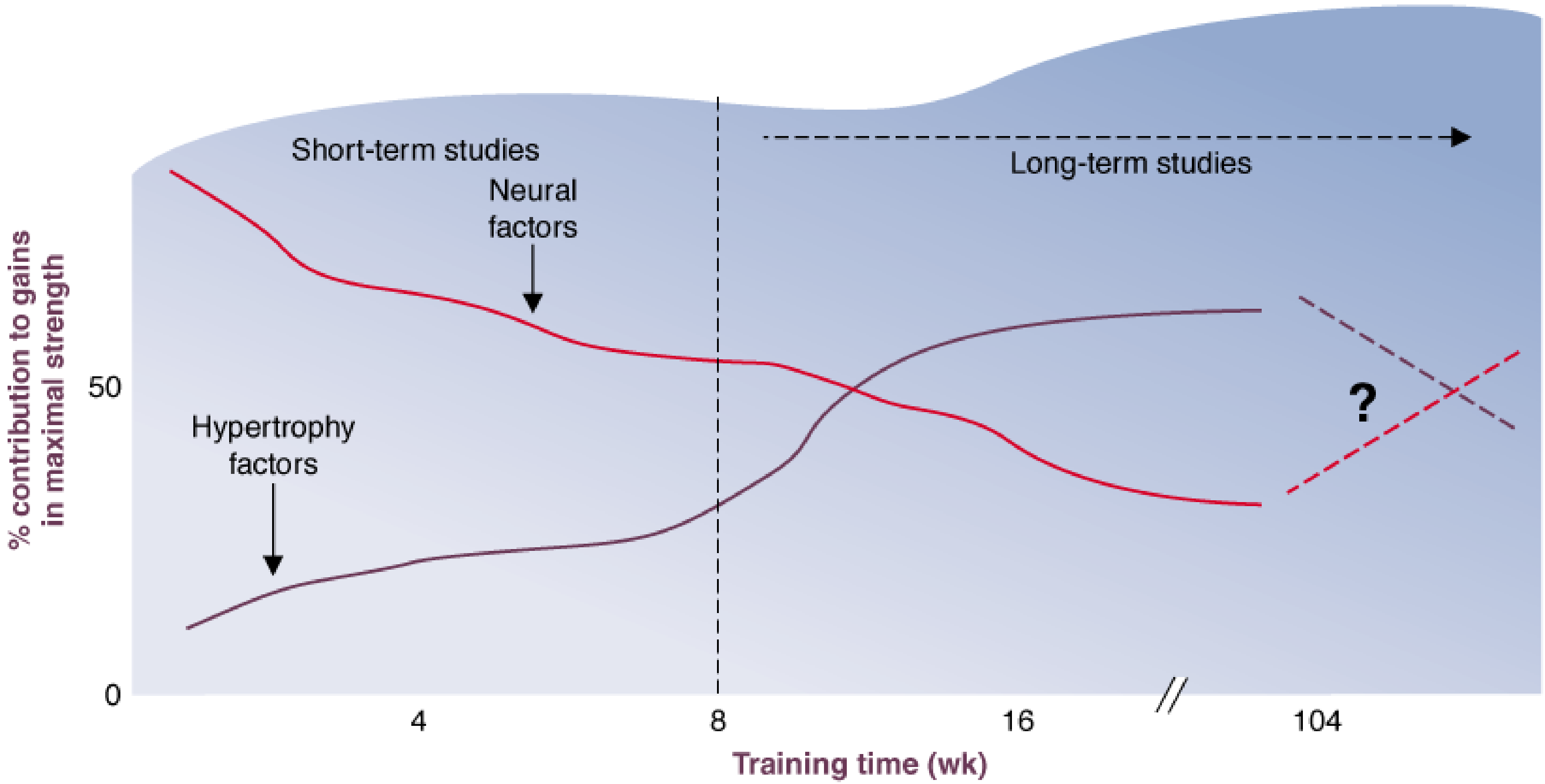
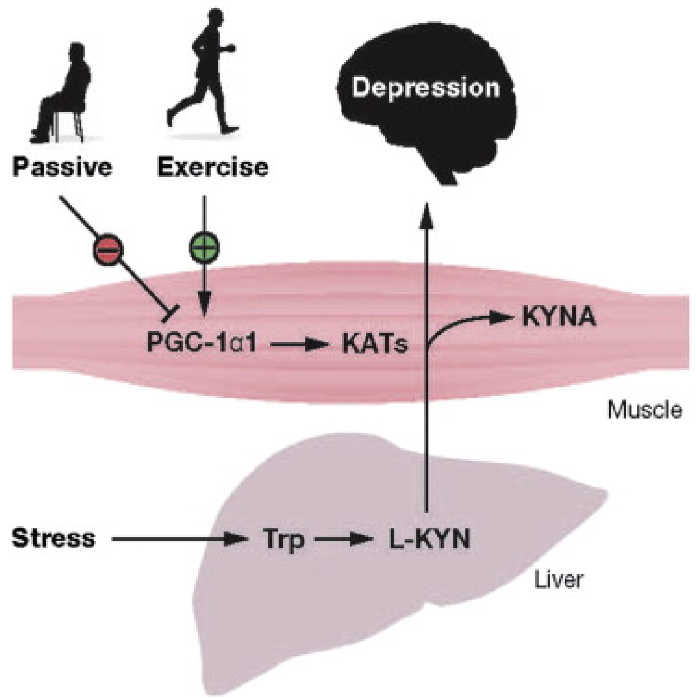


Fig. 4. Muscle strength. Forest plot illustrating the standardized mean difference in muscle strength between the intervention and control group of the three appropriate studies.

MODEL OF NEURAL AND HYPERTROPHIC FACTORS





Leandro Z. Agudelo , Teresa Femenía , Funda Orhan , Margareta Porsmyr-Palmertz , Michel Goiny , Vicente Martinez-R...

Skeletal Muscle PGC-1α1 Modulates Kynurenine Metabolism and Mediates Resilience to Stress-Induced Depression

Cell, Volume 159, Issue 1, 2014, 33 - 45

http://dx.doi.org/10.1016/j.cell.2014.07.051

Curr Heart Fail Rep (2010) 7:52–58
DOI 10.1007/s11897-010-0013-9

Electrical Muscle Stimulation for Chronic Heart Failure: An Alternative Tool for Exercise Training?

Prithwish Banerjee

Abstract Conventional exercise training has been shown conclusively to improve exercise capacity, quality of life, and even reduce mortality in chronic heart failure. Unfortunately, not all heart failure patients are suitable for conventional exercise programs for various reasons. The exciting new technique of electrical muscle stimulation (EMS) of large groups of muscles has been shown to produce a physiologic response consistent with cardiovascular exercise at mild to moderate intensities by increasing peak oxygen consumption, carbon dioxide production, ventilatory capacity, and heart rate. Additionally, there is improvement in muscle strength. The handful of small studies that exist of home-based EMS training of leg muscles in heart failure show that EMS produces similar benefits to conventional exercise in improving exercise capacity, making EMS an alternative to aerobic exercise training in those that cannot undertake conventional exercise. The improvement seen in leg muscle strength promises also to improve mobility in this sedentary population.

MAIN RESULTS: One hundred and twenty one trials with 6700 participants were included. In most trials, PRT was performed two to three times per week and at a high intensity. PRT resulted in a small but significant improvement in physical ability (33 trials, 2172 participants; SMD 0.14, 95% CI 0.05 to 0.22). Functional limitation measures also showed improvements: e.g. there was a modest improvement in gait speed (24 trials, 1179 participants, MD 0.08 m/s, 95% CI 0.04 to 0.12); and a moderate to large effect for getting out of a chair (11 trials, 384 participants, SMD -0.94, 95% CI -1.49 to -0.38). PRT had a large positive effect on muscle strength (73 trials, 3059 participants, SMD 0.84, 95% CI 0.67 to 1.00). Participants with osteoarthritis reported a reduction in pain following PRT(6 trials, 503 participants, SMD -0.30, 95% CI -0.48 to -0.13). There was no evidence from 10 other trials (587 participants) that PRT had an effect on bodily pain. Adverse events were poorly recorded but adverse events related to musculoskeletal complaints, such as joint pain and muscle soreness, were reported in many of the studies that prospectively defined and monitored these events. Serious adverse events were rare, and no serious events were reported to be directly related to the exercise programme.
AUTHORS' CONCLUSIONS: This review provides evidence that PRT is an effective intervention for improving physical functioning in older people, including improving strength and the performance of some simple and complex activities. However, some caution is needed with transferring these exercises for use with clinical populations because adverse events are not adequately reported.

1. Cochrane Database Syst Rev. 2009 Jul 8;(3):CD002759. doi: 10.1002/14651858.CD002759.pub2.

Progressive resistance strength training for improving physical function in older adults.

Liu CJ(1), Latham NK.

Konklusion

- Muskelfunktion markant nedsatt vid åldrande och vid kronisk hjärtsvikt
- Muskulär motståndsträning kan motverka sarcopeni och hypotrofi vilket i sin tur kan förbättra maximal syreupptagningsförmåga