Dénutrition en réanimation

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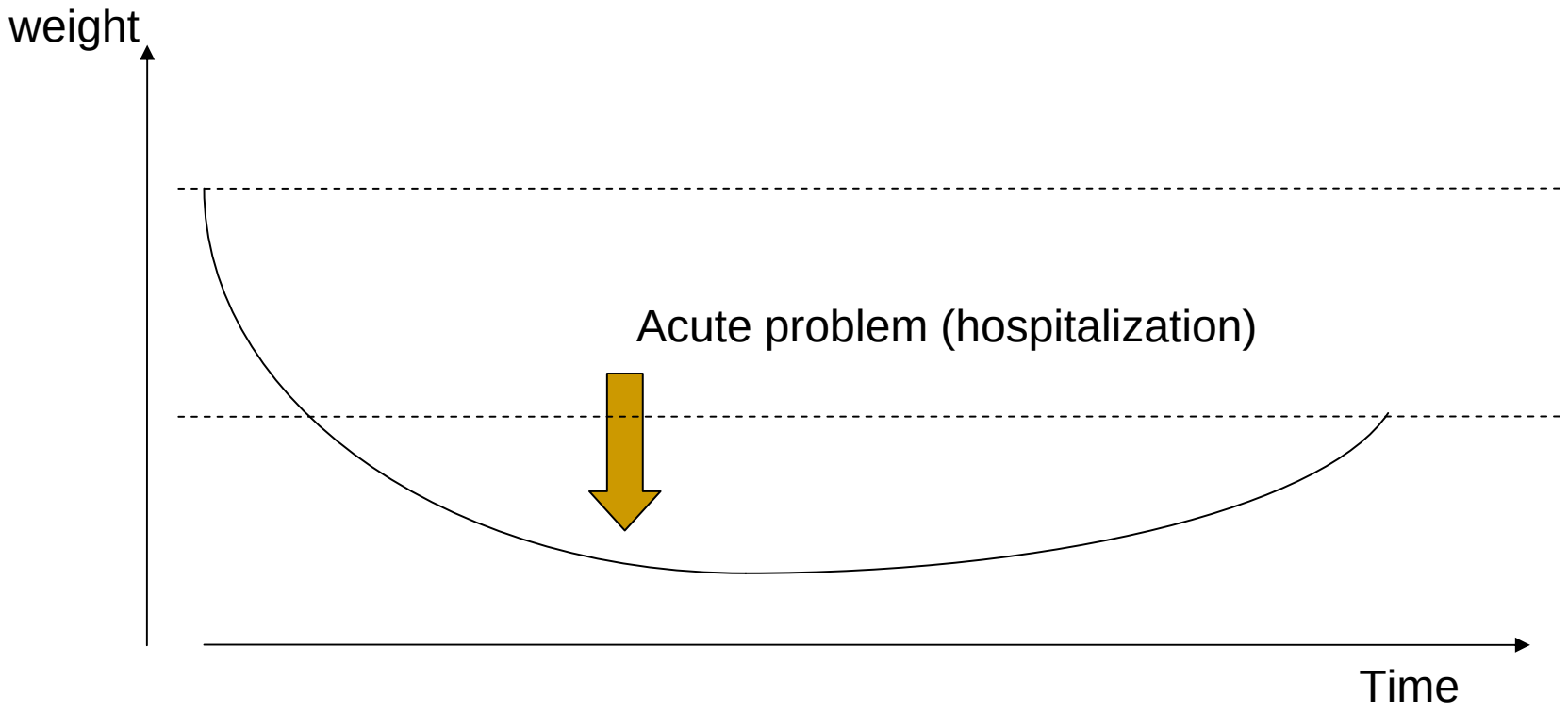
Undernutrition?

Consensus statement of the Academy of nutrition and dietetics and ASPEN
JPEN 2012;36:275

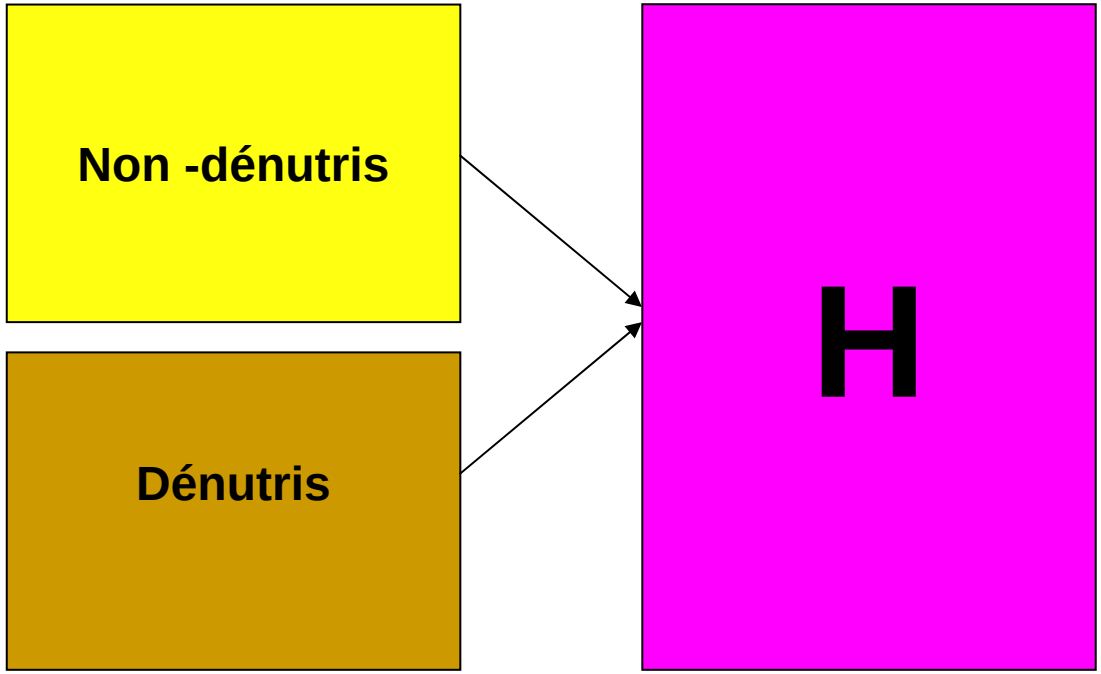
- Prolonged decrease of energy intake
- Weight loss
- Physical findings
  - Loss of subcutaneous fat
  - Muscle loss
  - Fluid accumulation
  - Reduced grip strength

History of undernutrition



DENUTRITION – HOPITAL

Situation actuelle

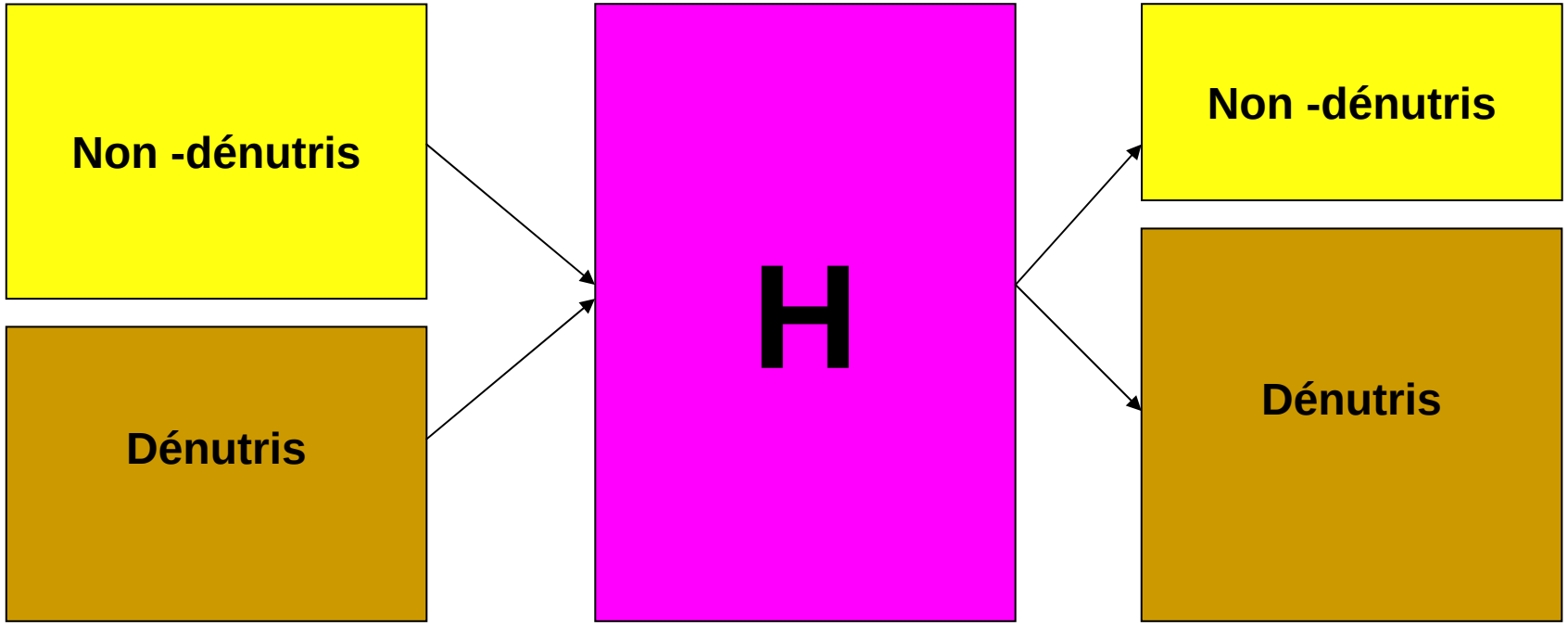


Prevalence of Undernutrition Hospital admission

Kyle	2005	1707	51
Pichard	2004	996	46
Wyszynski	2003	1000	48
Waitzberg	2002	4000	48
Kyle	2001	995	38
Larsson	1993	382	29
Willard	1990	200	32
Coats	1983	228	38
McWhirter	1994	300	45
Robinson	1987	100	56
Reilly	1988	365	59
Bistrian	1980	251	44
		10858	**46**

MALNUTRITION – HOPITAL

Situation actuelle



Sous-nutrition acquise à l'hopital

- Liée à la maladie
  - Prise alimentaire insuffisante
    - Anorexie, altération du goût, nausées/vomissements, dysphagie, troubles de la déglutition, dépression, anxiété
  - Maldigestion-malabsorption
  - Augmentation DER – catabolisme accéléré
- Liée aux traitements – aux examens

General criteria for implementation of nutritional support

- « Classical »
  - Pre-existing severe malnutrition / **nutrition risk**
  - Oral intake matches < 50% of needs
  - Expected delay before recovery of eating > 5-7 days (guidelines ESPEN : 3 days)
- Early enteral nutrition
  - Surgical, trauma, burns

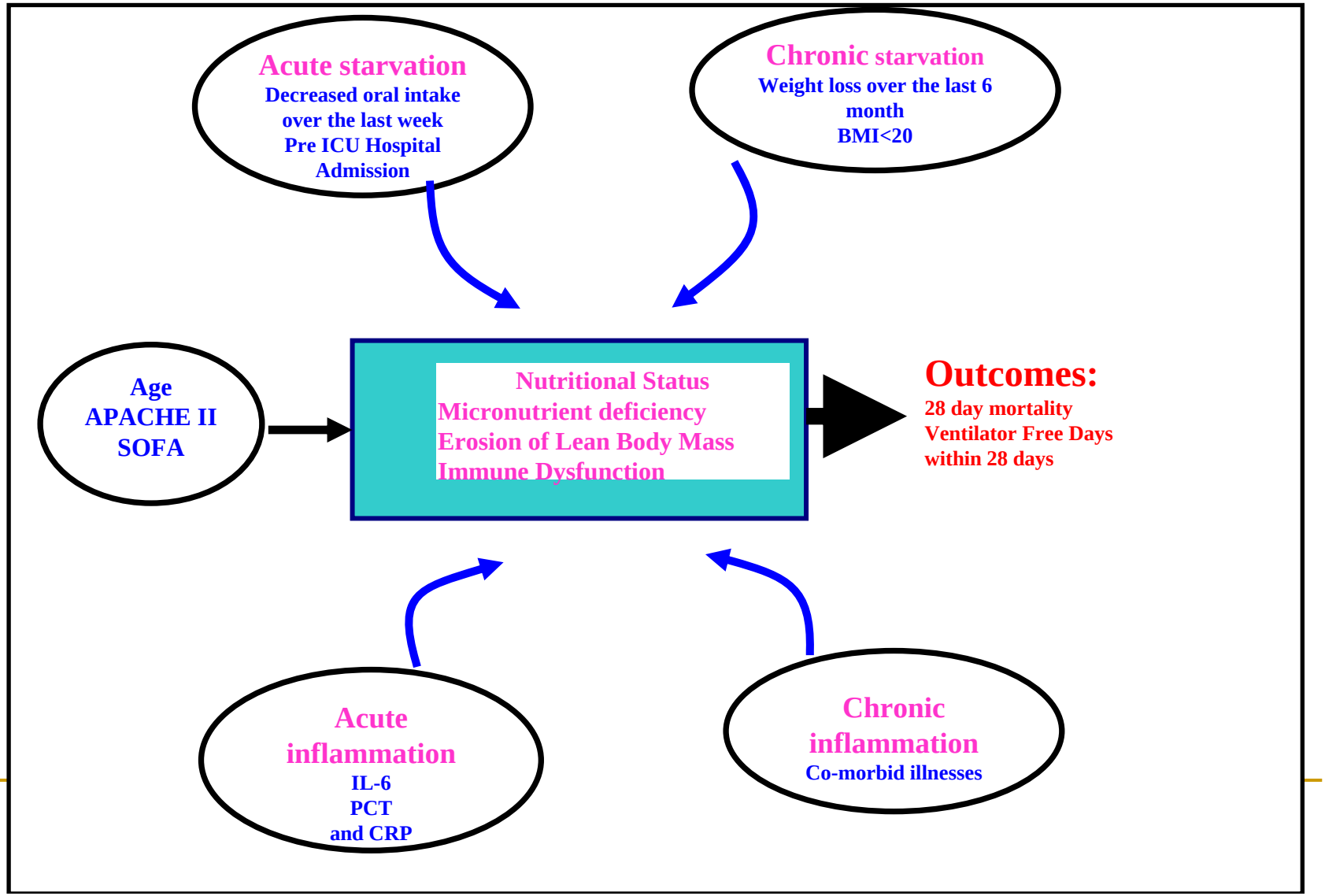
SCORING SYSTEM : The simplest = the best

Clin Nutr 2003;22:331

- **Nutrition risk score (NRS)**
  - Nutritional status
    - Weight loss
    - Food intake
    - BMI
  - Severity of disease
  - Age

- 0-3 for nutritional status
- **0-3 for disease severity**
- + 1 when age > 70
- Sum > 3 : risk of malnutrition

Conceptual Model For Nutrition Risk Assessment in the Critically Ill



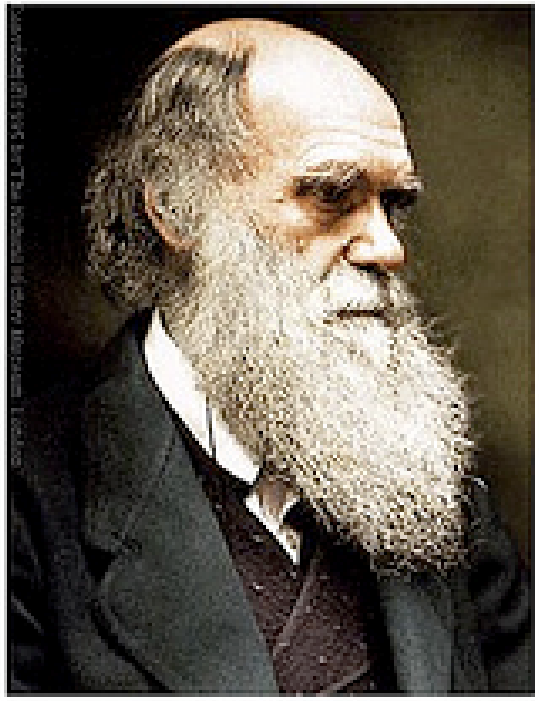
Le stress ultime .. Images de la jungle

Le stress ultime .. Images de la jungle

Le stress ultime .. Images de la jungle

The critical illness

- A continuous challenge to physiology.. Revealing endogenous defences mechanisms

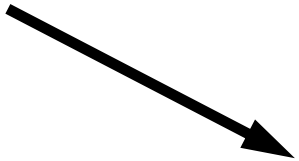


It is not the strongest of the species that survives, nor the most intelligent that survives. It is the one that is the most adaptable to change.

- Charles Darwin

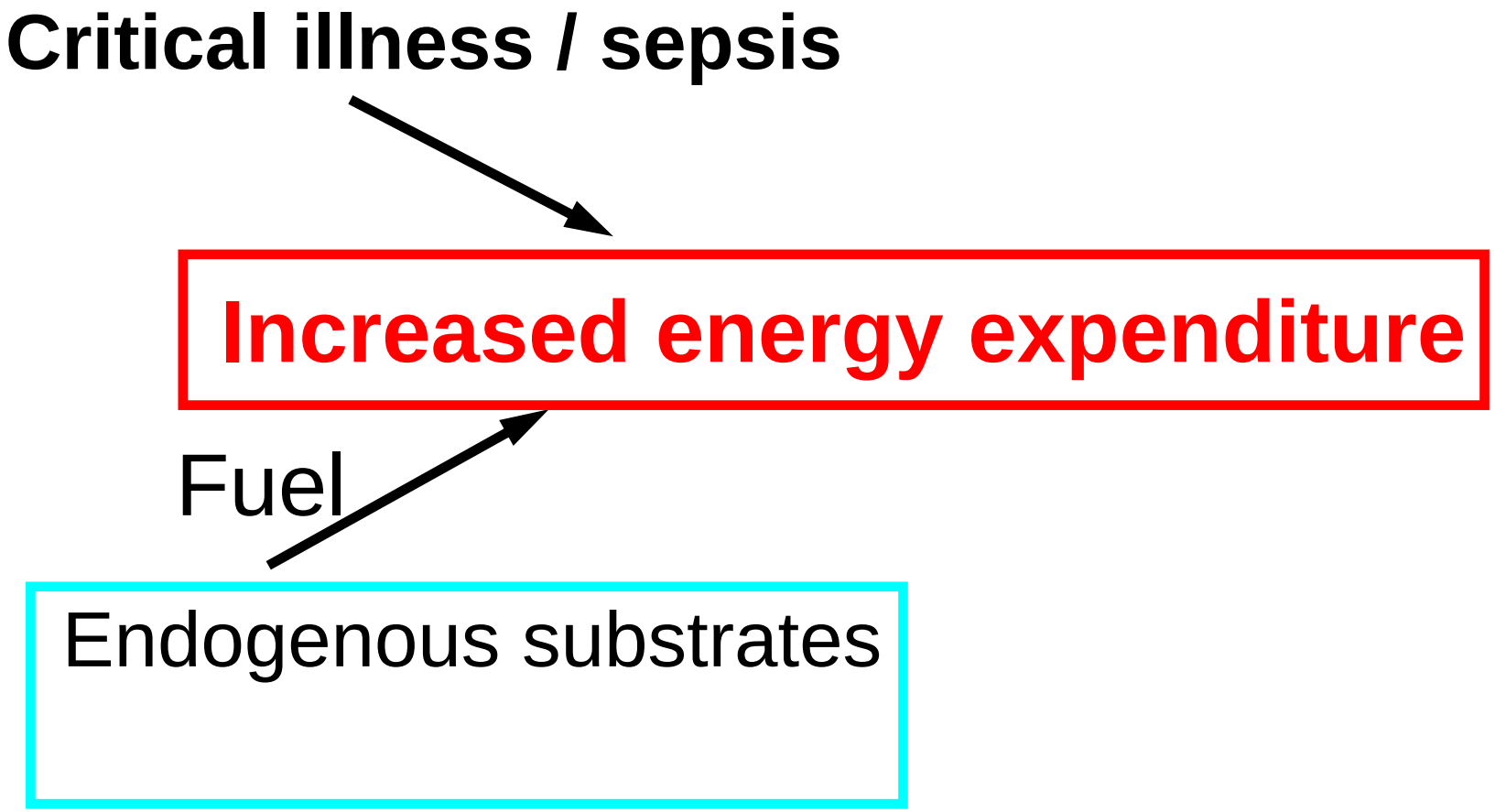
Energy balance in ICU patient

Critical illness / sepsis

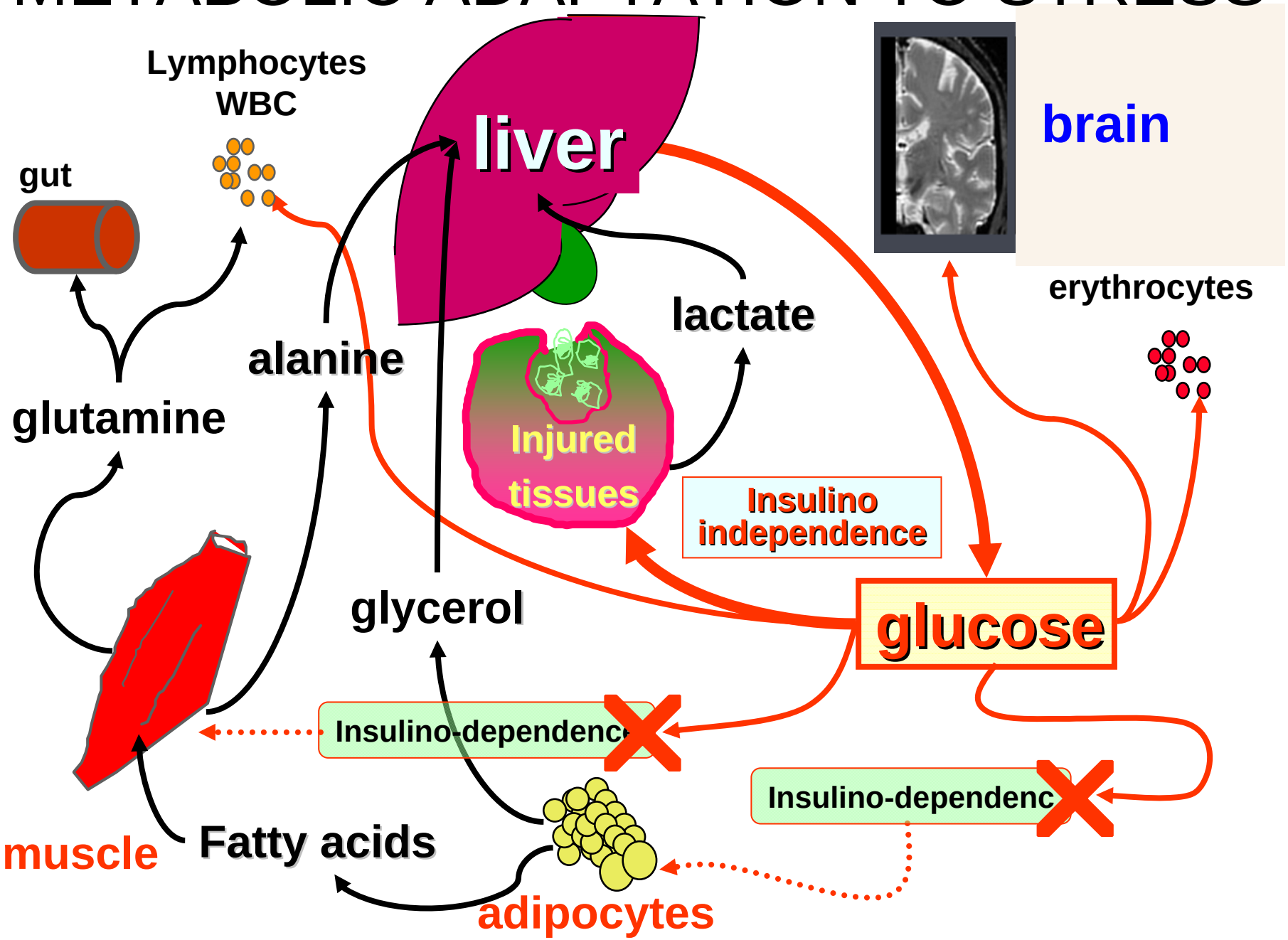


Increased energy expenditure

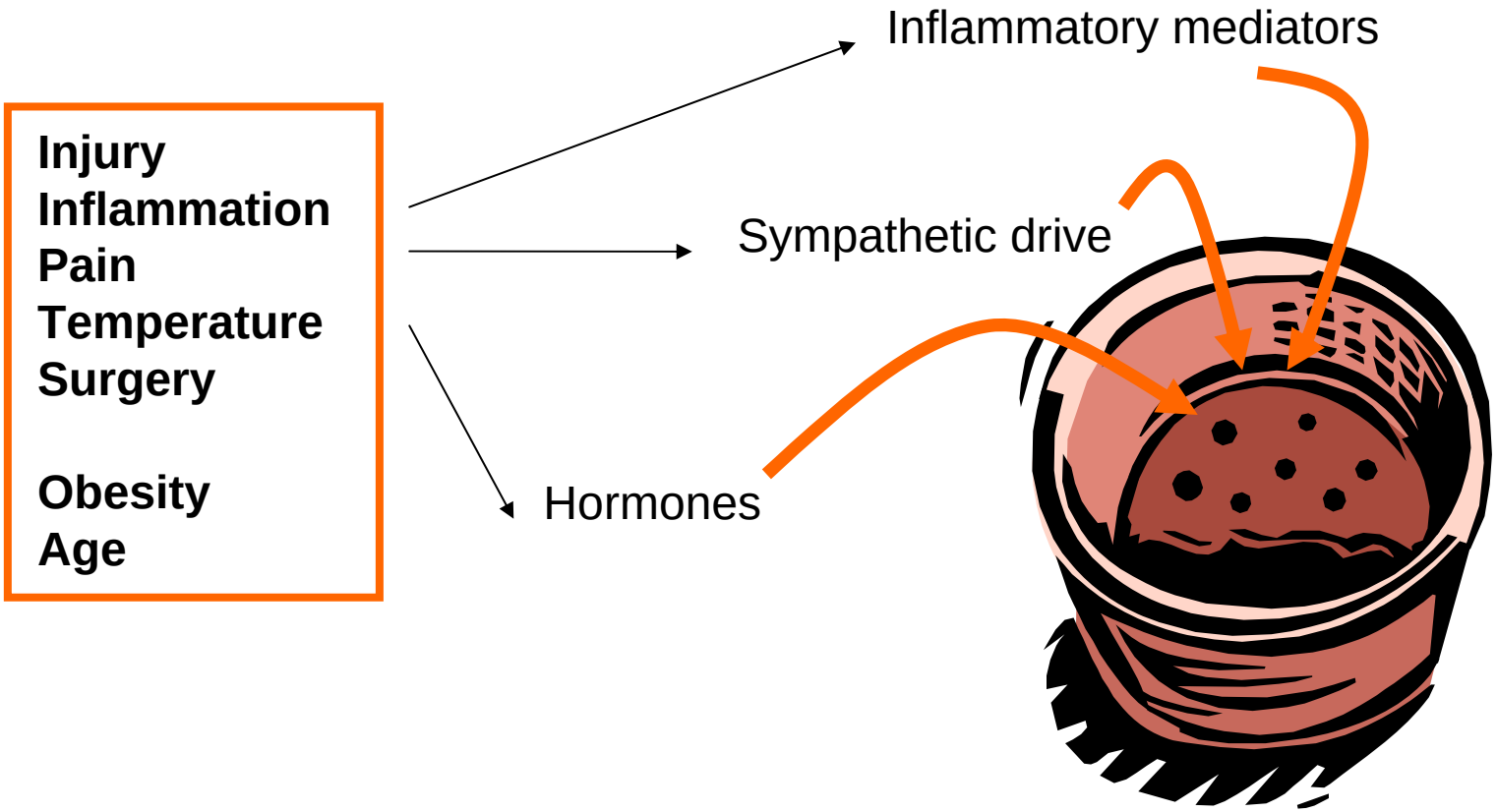
Energy balance in ICU patient



METABOLIC ADAPTATION TO STRESS



After any insult, insulin resistance occurs and is non-specific!



What is insulin resistance ?

Li and Messina Trends Endocrinol Metab 2009:20:429

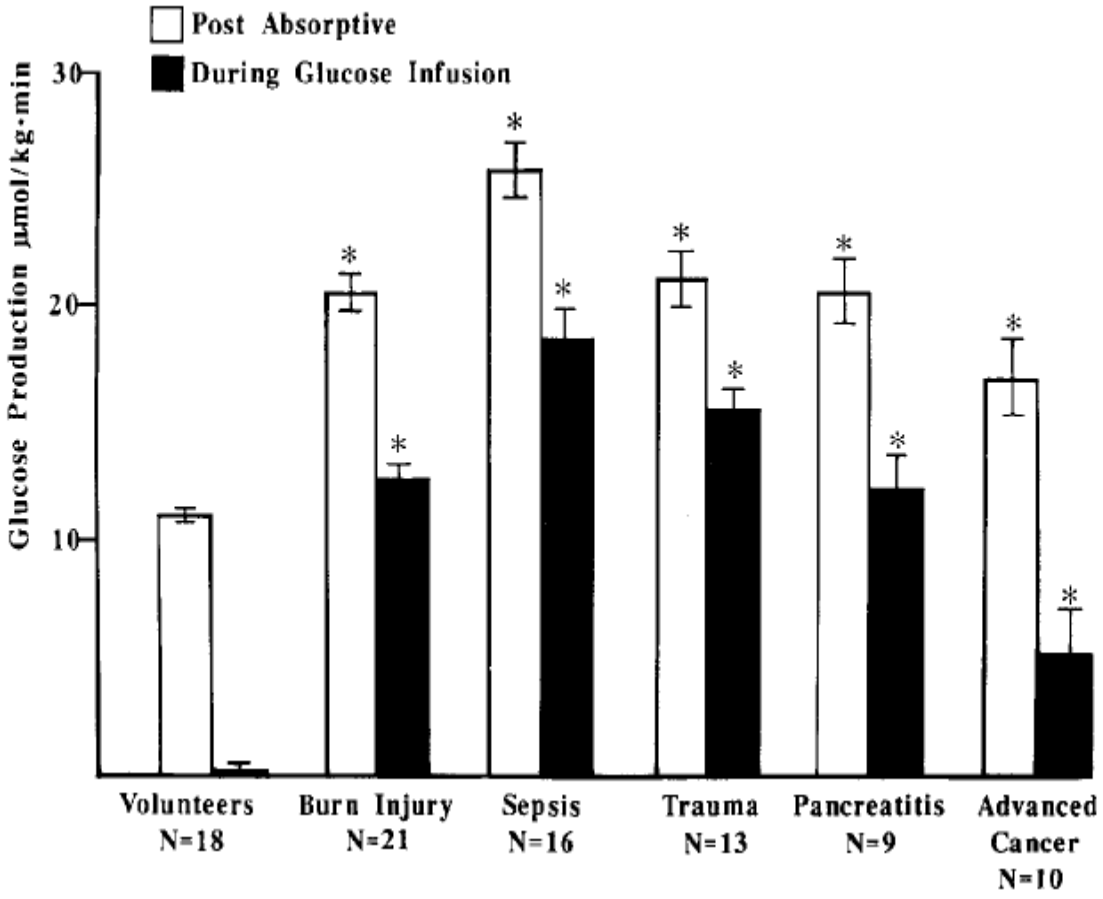
One practical definition of insulin resistance is the inability of insulin to adequately stimulate glucose uptake into skeletal muscle or to inhibit gluconeogenesis in the liver.

Insulin resistance can occur in chronic diseases (months, years) or in critically ill patients (minutes, hours).

Consequences of the adaptive response (chronological order)

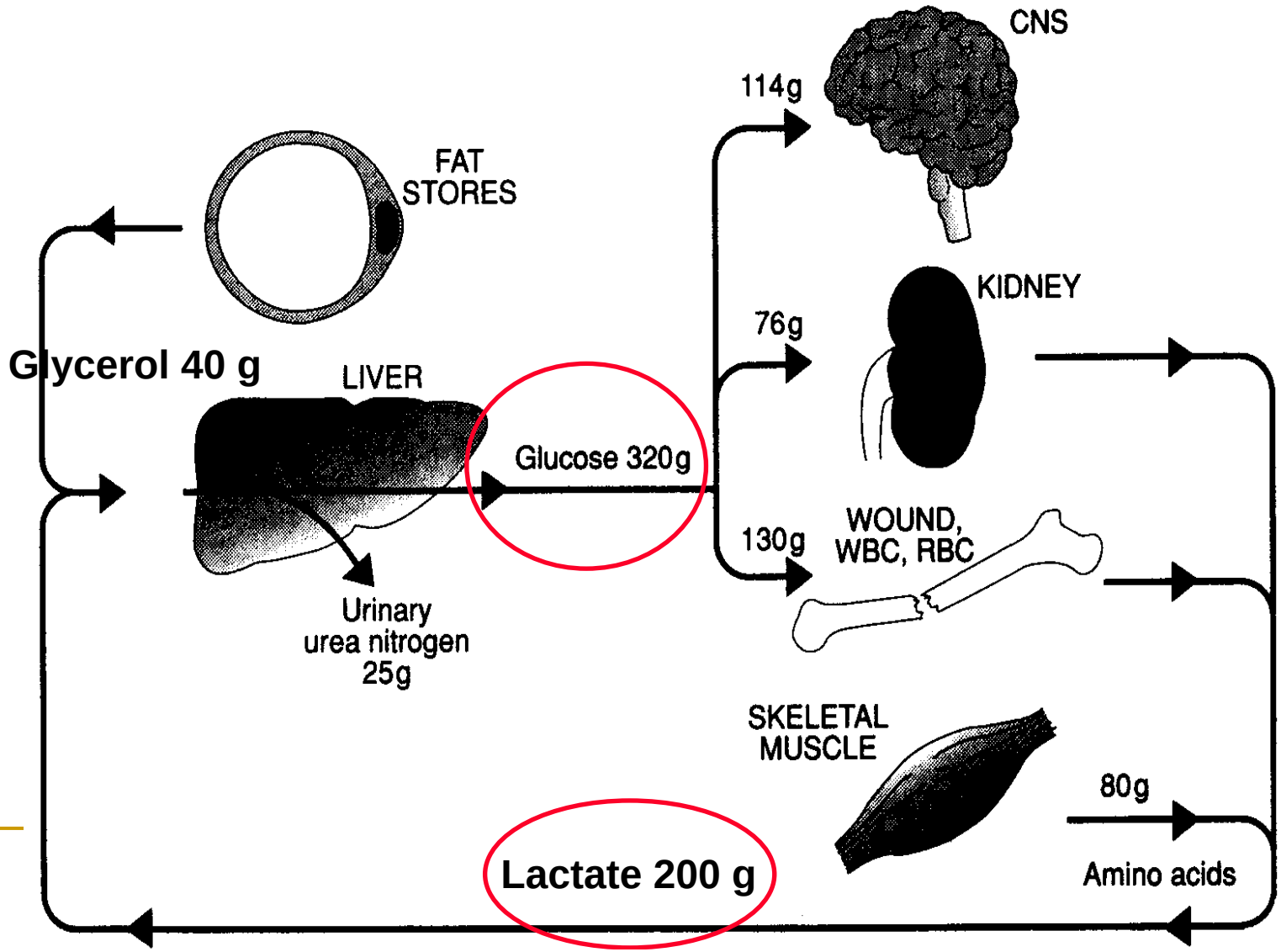
- Stress hyperglycemia
- Endogenous mobilisation of energy substrates
- Differences in the utilisation of energy substrates
- Loss of lean body mass

Rate of basal glucose production and endogenous production during glucose infusion in various conditions



Wolfe RR, Eur J Clin Nutr 1999

Glucose flow is increased after injury

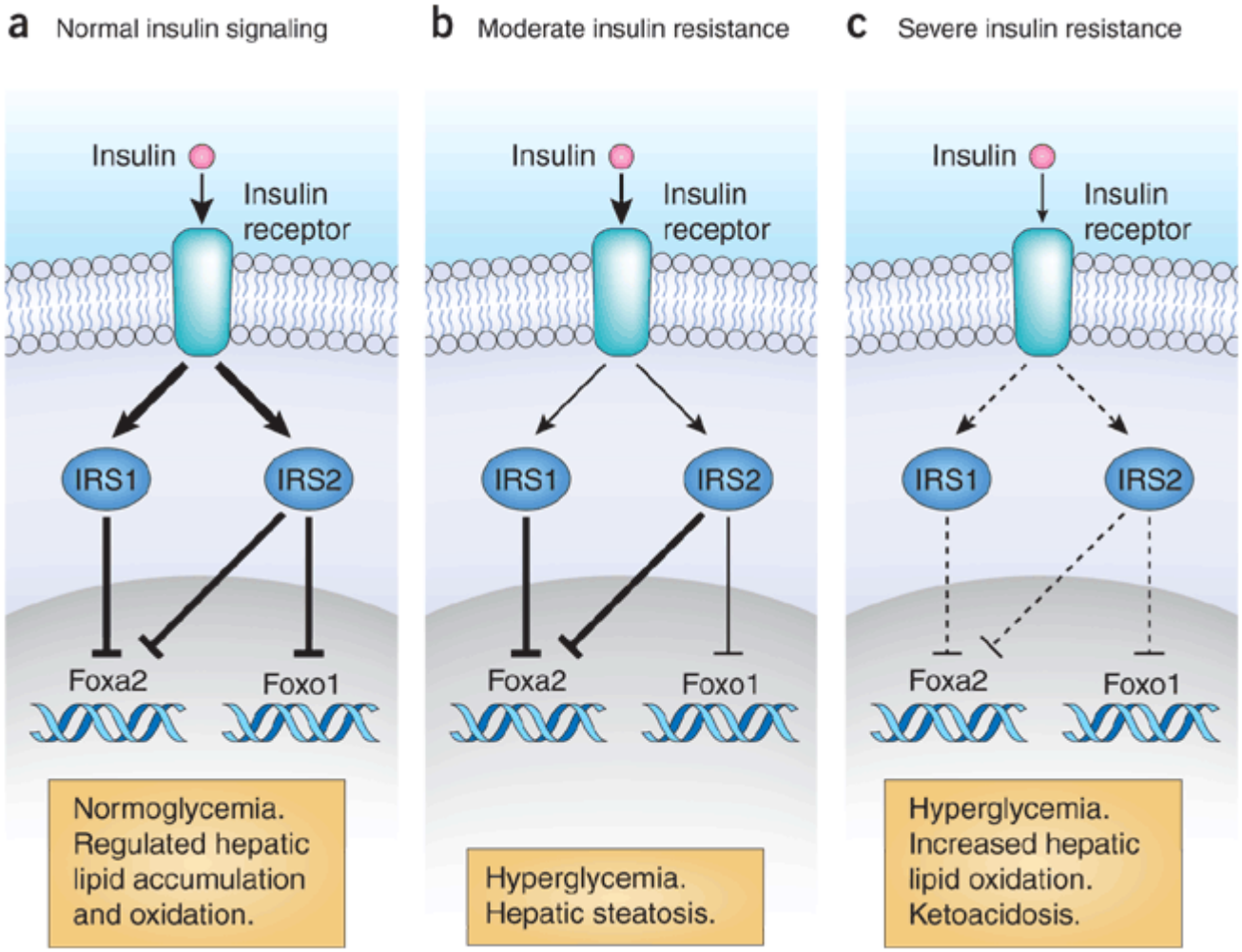


Critically ill patients are able to match their REE

Tappy L et al Crit Care Med 1998; 26: 860

3 day starvation

Resting metabolic rate	**1824 kcal/ day**
Glycemia	**7.3 mmol/L**
Endogenous glucose production	**360 g/ day (1360 kcal/d)**
Net glucose oxidation	**28% (512 kcal/ day)**
Net fat oxidation	**46% (840 kcal /day)**
Net protein oxidation	**26% (470 kcal/ day)**
Net protein balance	**-117 g/ day**

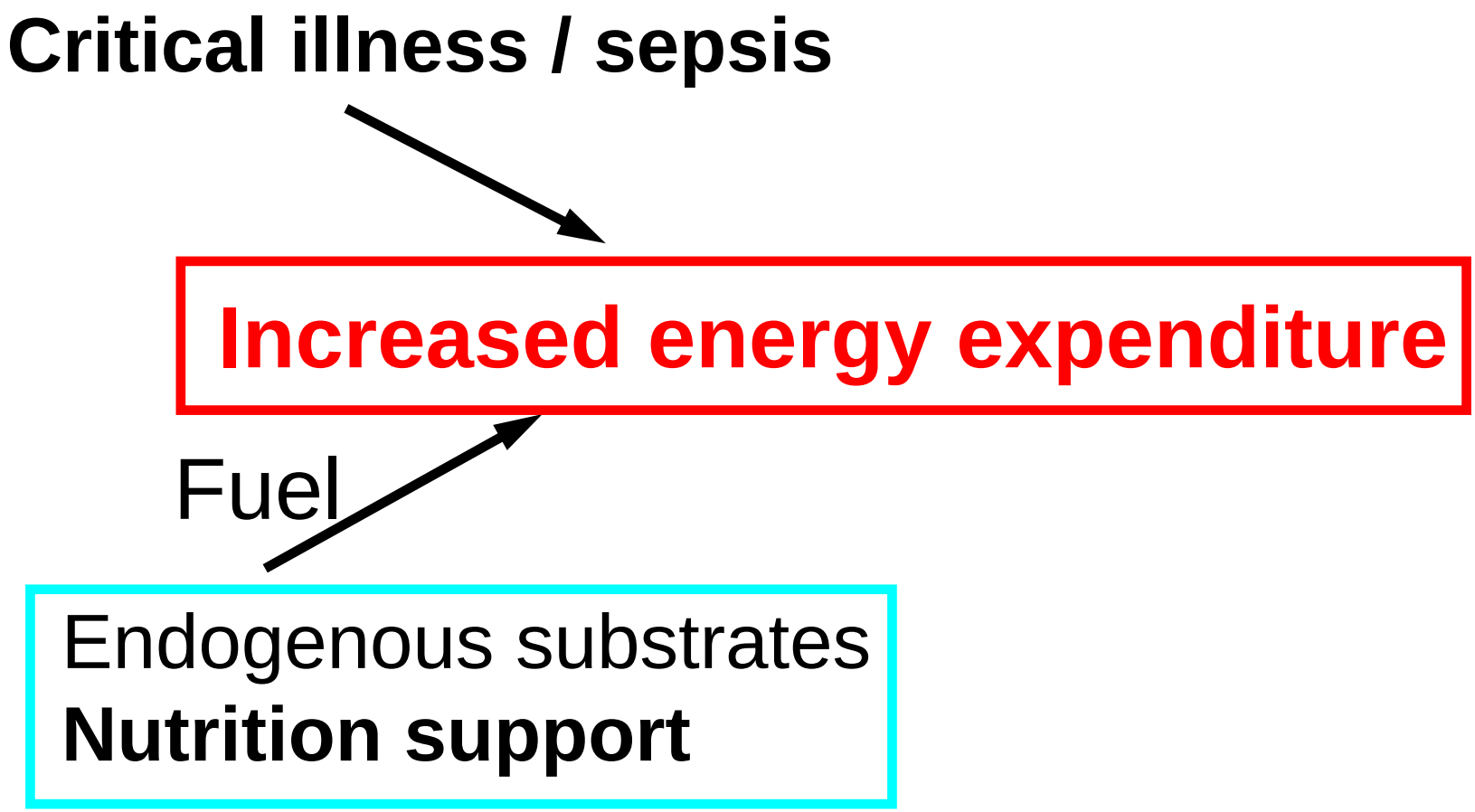


(a) Under normal conditions, the insulin pathway regulates Foxo1 and Foxa2, resulting in regulated glycemia and a balance of hepatic lipid accumulation and oxidation. **(b)** In moderate insulin resistance, reduced activity of the insulin pathway results in activation of Foxo1, leading to elevated gluconeogenesis and hyperglycemia. However, Foxa2 is more sensitive to insulin and therefore is still repressed, resulting in reduced lipid oxidation and hepatic steatosis. **(c)** In severe insulin resistance, the insulin pathway is barely active, and the constitutive activation of Foxo1 and Foxa2 result in elevated gluconeogenesis, hyperglycemia and high levels of lipid oxidation, leading to ketoacidosis.

In summary

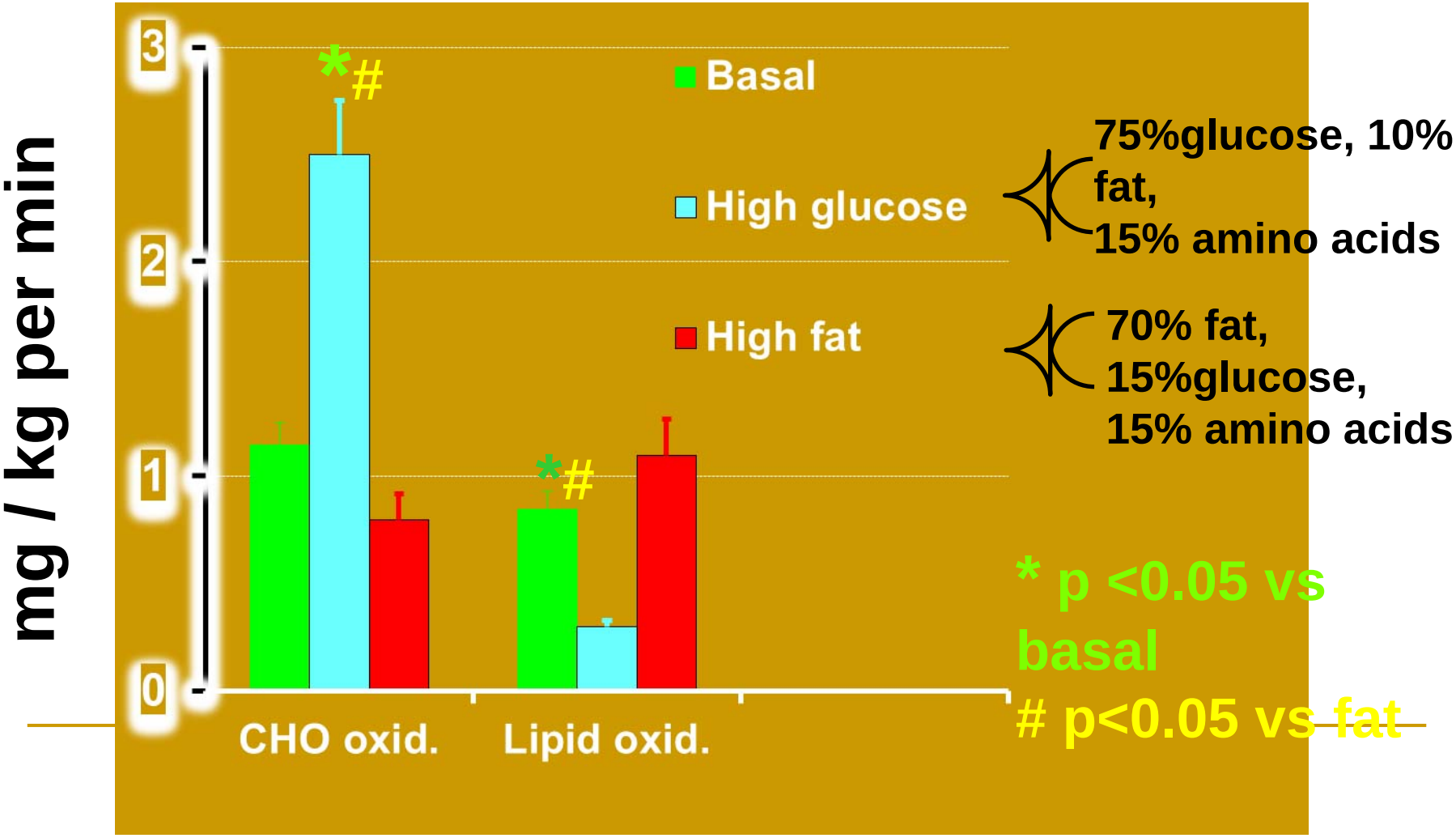
- Carbohydrates are the preferred fuel during the acute phase
- Lipids are less efficiently used than carbohydrates and exert several other effects
- Proteins breakdown is increased, probably account for 15-20% of the energy.

Energy balance in ICU patient

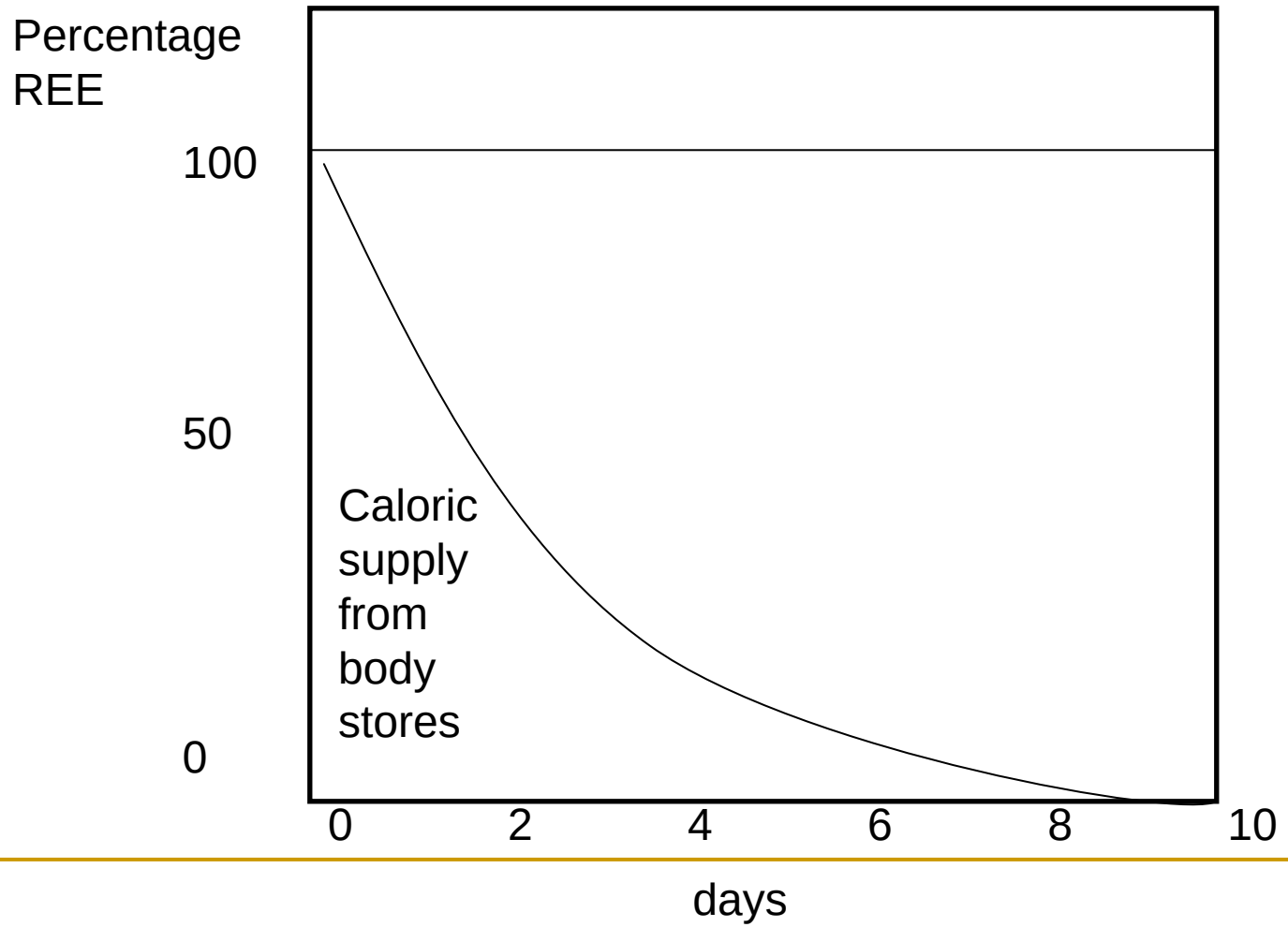


CHO vs lipids for isocaloric TPN
CHO and lipid net oxidation

Tappy L et al, CCM 1998; 26:860



How many calories should be prescribed?
How does the body match the increased REE?



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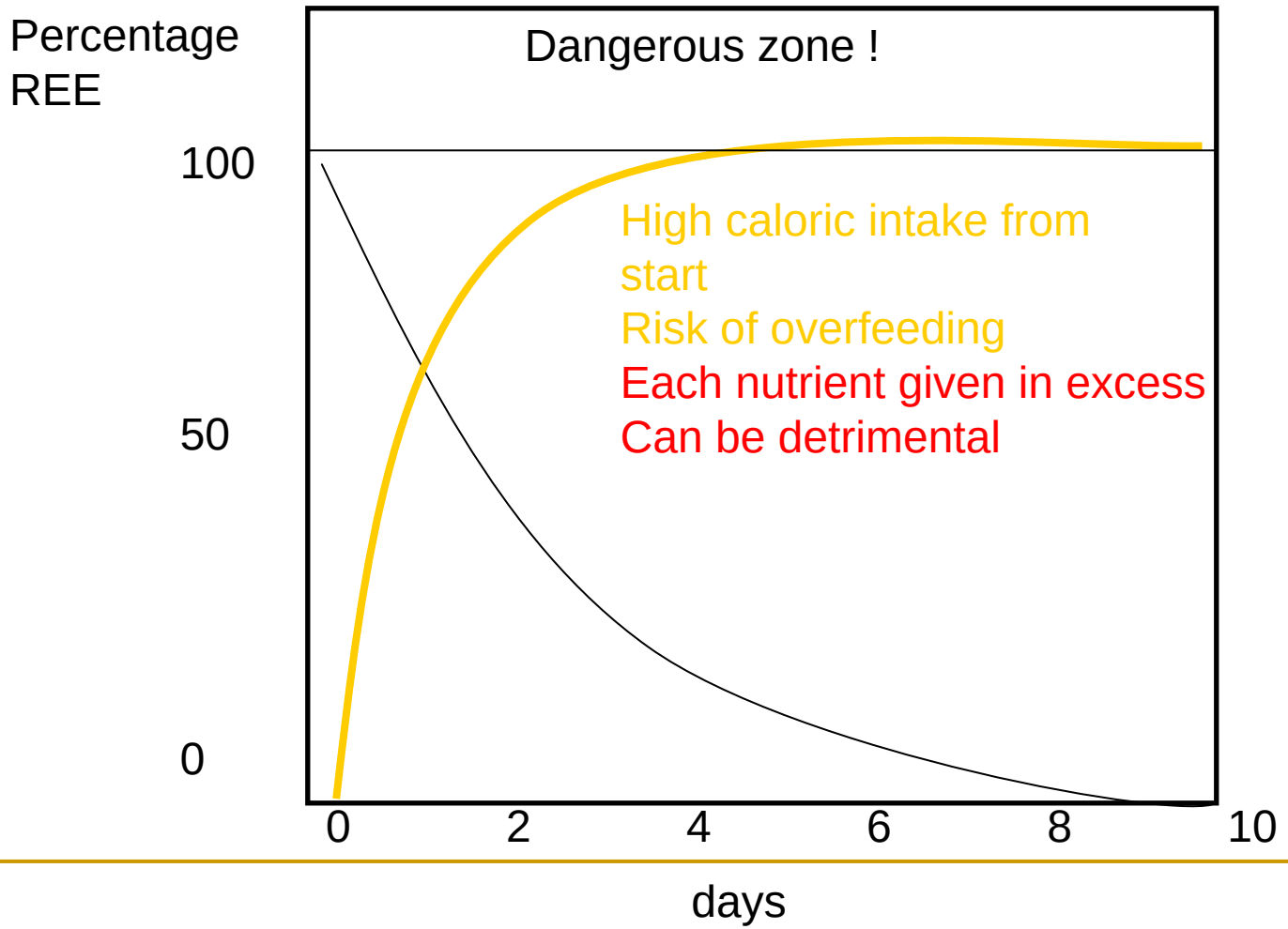
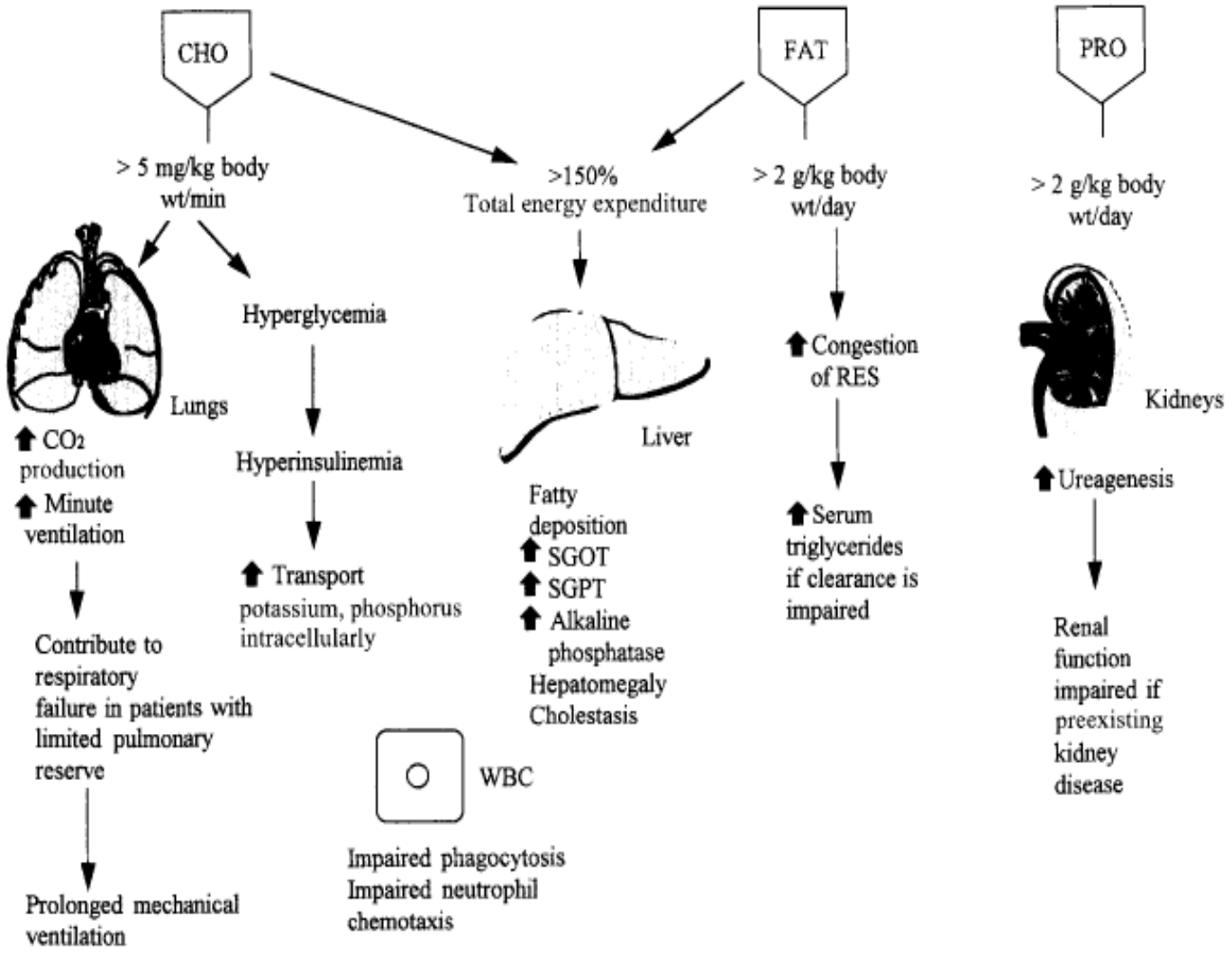


Adverse effect of overfeeding

Klein et al JADA 1998;98:795



Positive energy balance is associated with accelerated muscle atrophy and increased erythrocyte glutathione turnover during 5 wk of bed rest[1–3]

Gianni Biolo, Francesco Agostini, Bostjan Simunic, Mariella Sturma, Lucio Torelli, Jean Charles Preiser, Ginette Deby-Dupont, Paolo Magni, Felice Strollo, Pietro di Prampero, Gianfranco Guarnieri, Igor B Mekjavic, Rado Pišot, and Marco V Narici

Am J Clin Nutr 2008;88:950–8.

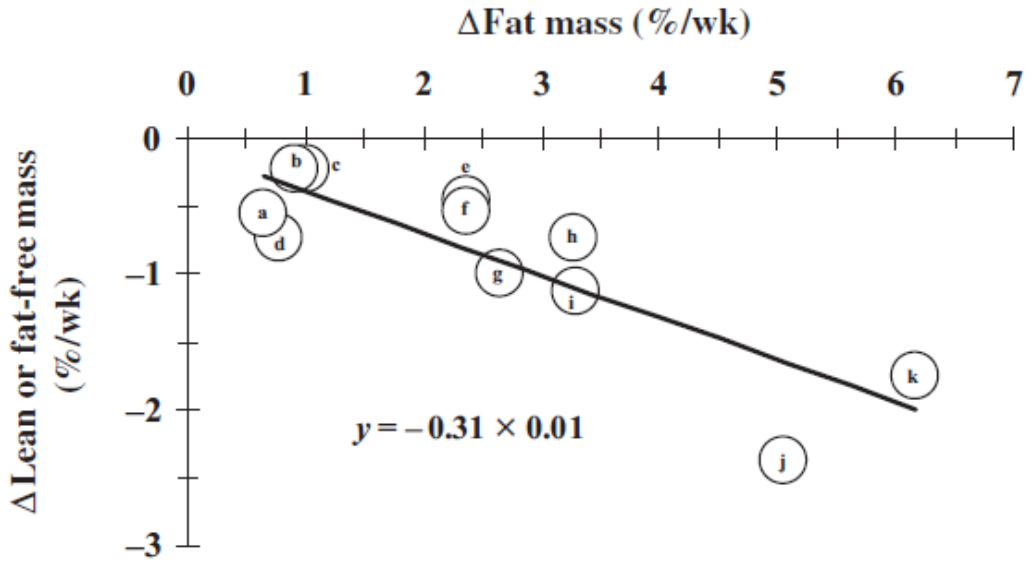
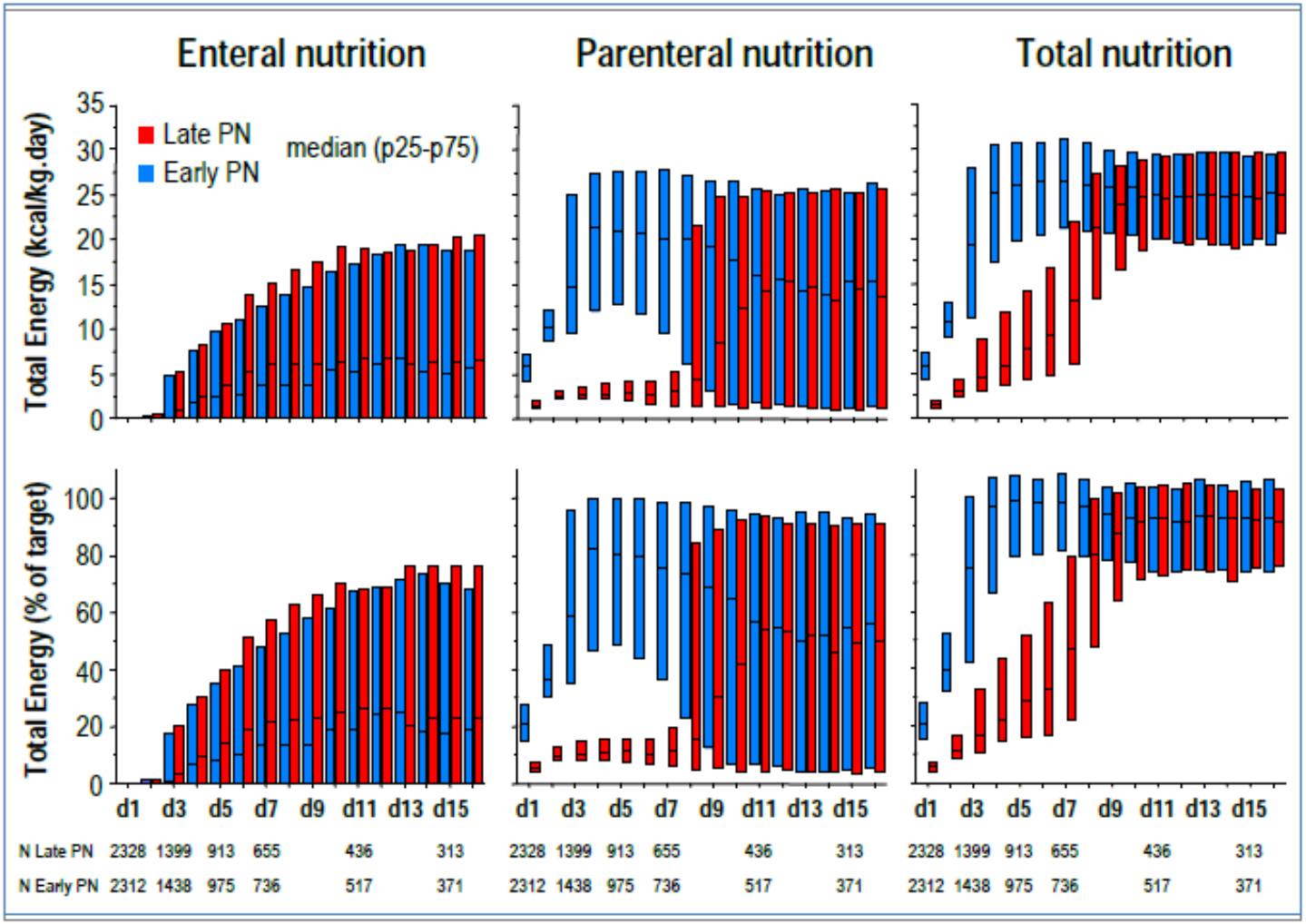


FIGURE 2. Relation between average values of absolute changes in fat mass and lean mass (by dual-energy X-ray absorptiometry) or fat-free mass (by bioelectrical impedance analysis) in previous studies and in the present study. Letters inside (or just outside of) circles represent values from these studies: a, Barbe et al (10); b, Scheld et al (13); c, Stein et al (12); d, present study, lower-energy-balance group; e, Blanc et al (6); f, Krebs et al (7); g, Lovejoy et al (11); h, Gretebeck et al (8); i, present study, higher-energy-balance group; j, Ferrando et al (9); and k, Olsen et al (5). $r = -0.85$, $P = 0.001$; $n = 11$.

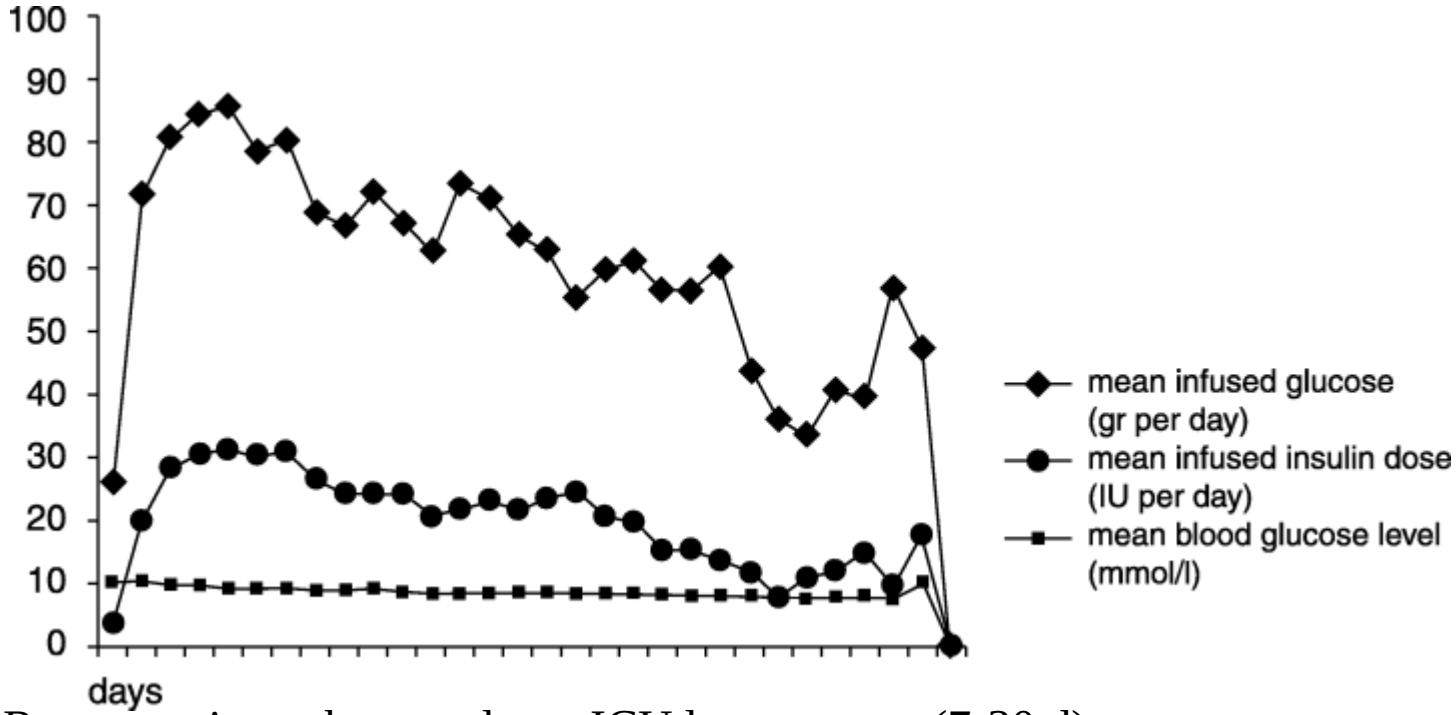
Caloric intake in the e-PaNIC trial

Figure 2: Nutrition



Intravenous glucose and hospital mortality

Van der voort Clin Endocrinol 2006;64:141



Retrospective cohort study on ICU long-stayers (7-30 d)

N = 273 (/ 2042)

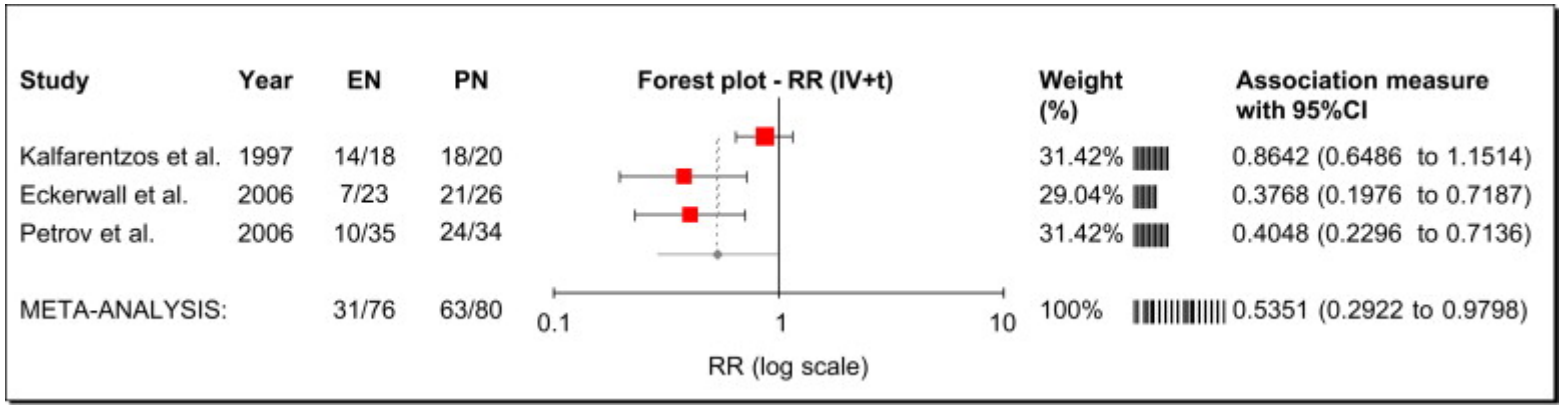
Hospital mortality lower when mean BG < 8 mmol/L

Logistic multivariate regression analysis : APACHE II and mean daily amount of IV Glucose associated with lower survival (OR 0.94 (0.9-0.98) and 0.65 (0.47-0.89))

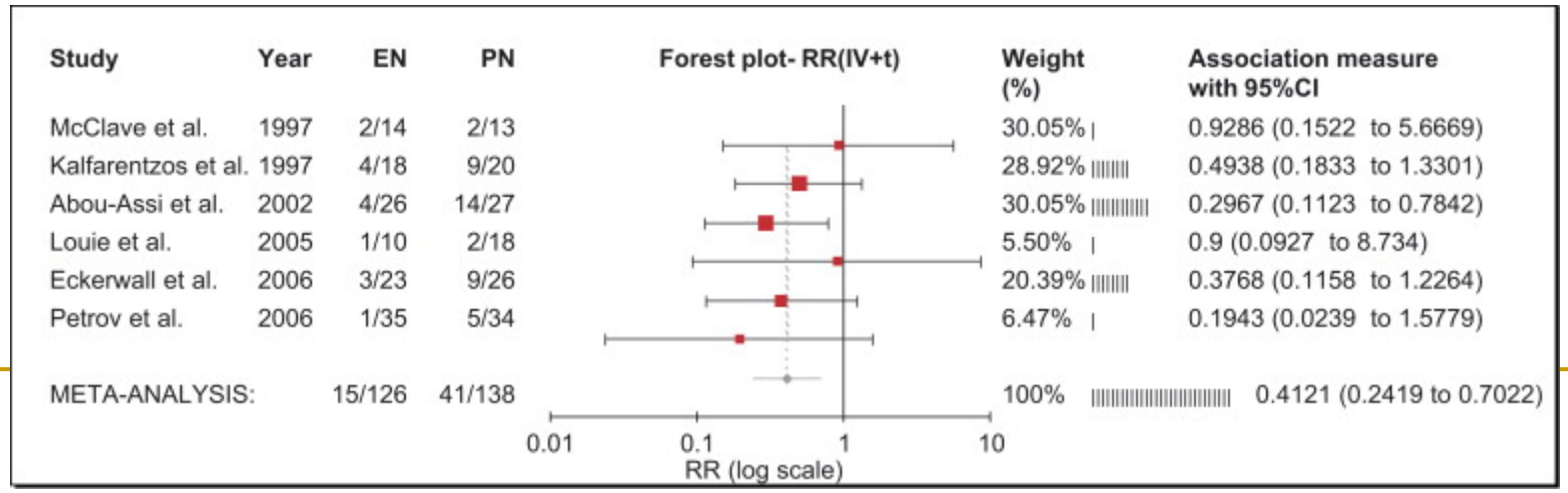
Influence of enteral vs parenteral nutrition on glucose control

Petrov Clin Nutr 2007;26:514

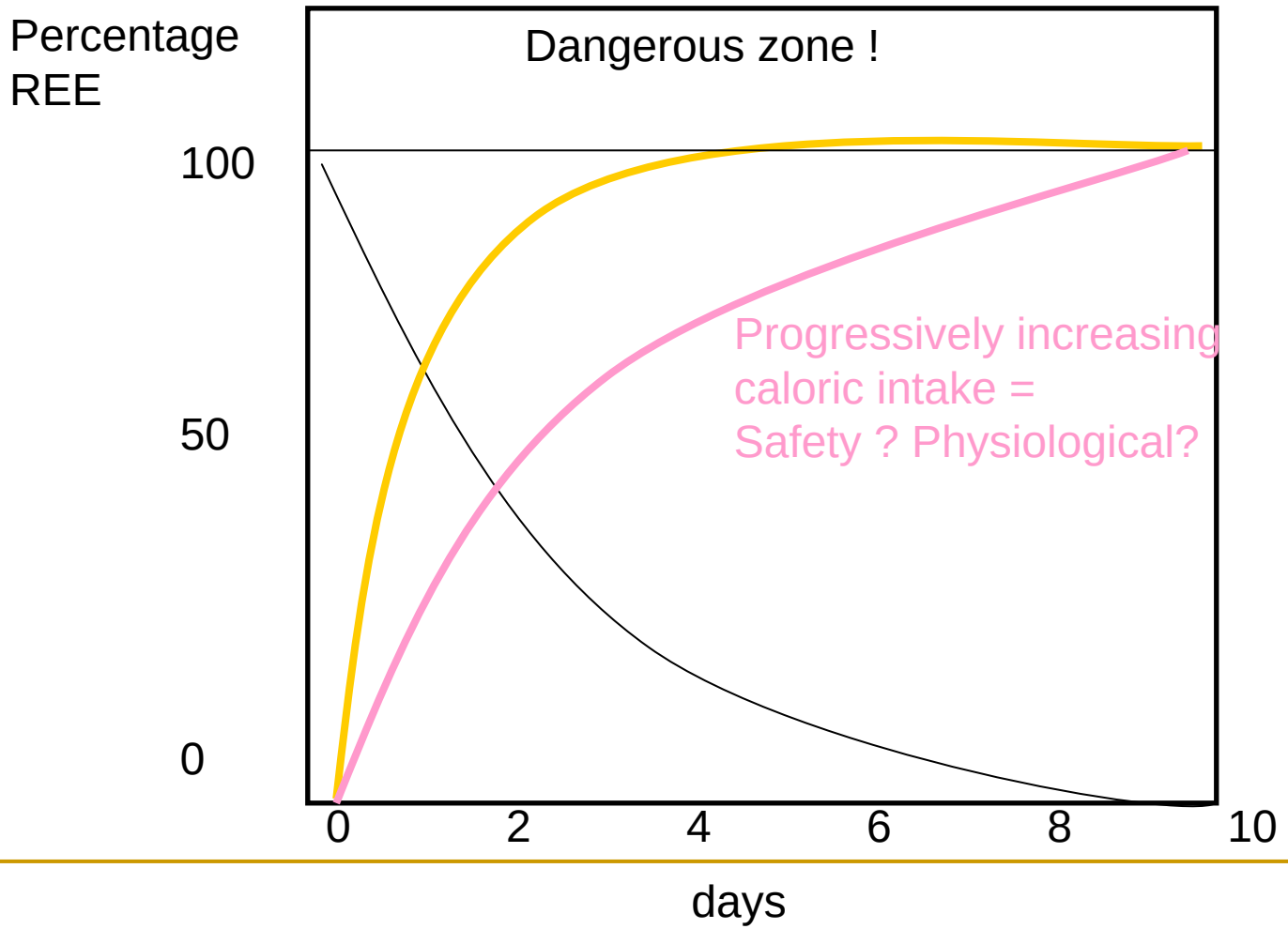
Random-effect model of relative risk of hyperglycemia associated with enteral nutrition compared with parenteral nutrition.

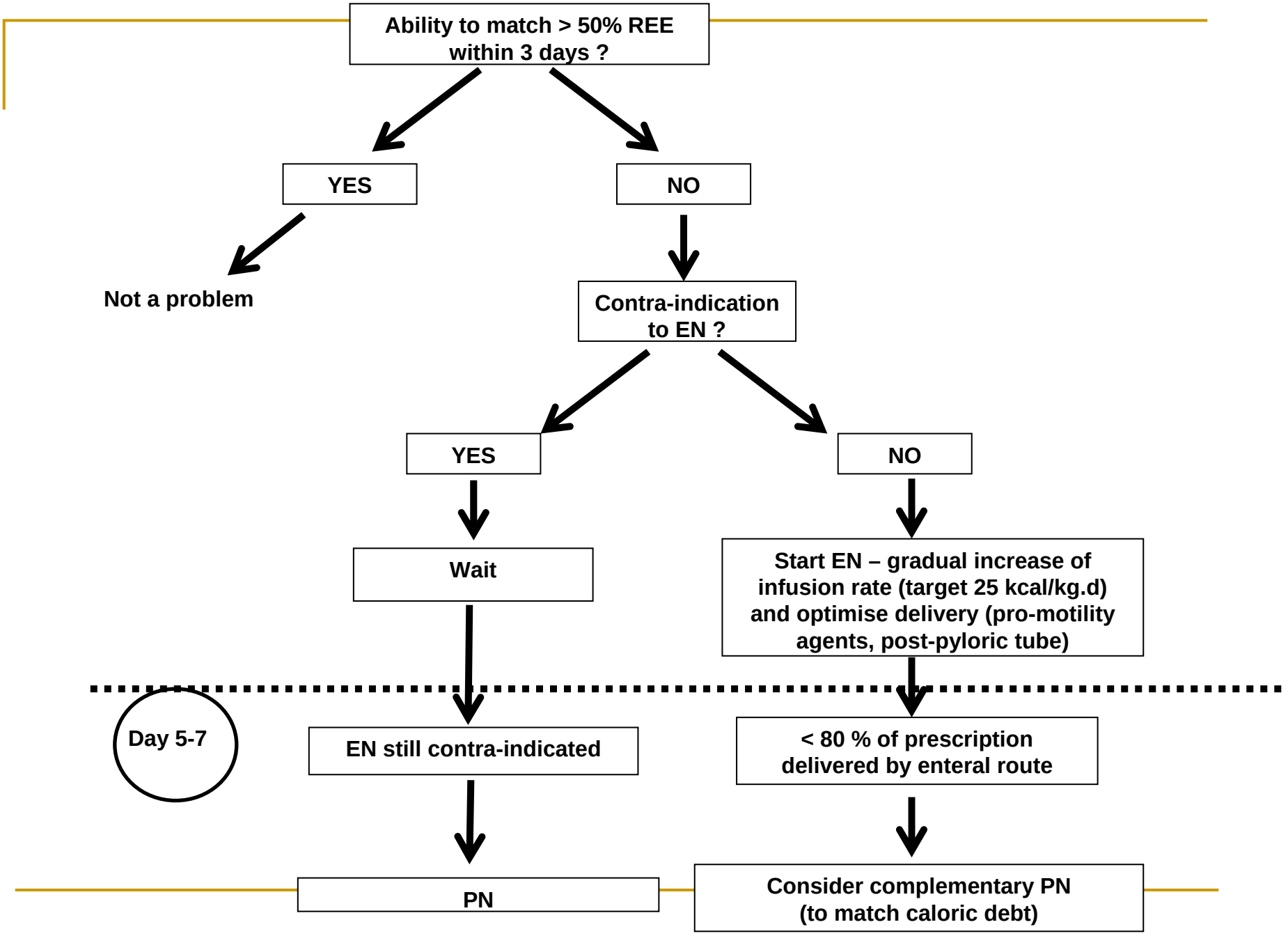


Random-effect model of relative risk of insulin requirement associated with enteral nutrition compared with parenteral nutrition.



How many calories should be prescribed?




Ability to match > 50% REE within 3 days ?
YES
NO
Not a problem
Contra-indication to EN ?
YES
NO
Wait
Start EN – gradual increase of infusion rate (target 25 kcal/kg.d) and optimise delivery (pro-motility agents, post-pyloric tube)
Day 5-7
EN still contra-indicated
< 80 % of prescription delivered by enteral route
PN
Consider complementary PN (to match caloric debt)

Current recommendations

- **Total energy 20-25 (catabolic phase) to 25-30 kcal/kg.d**
- **Carbohydrates (50-60 % of energy – 2-4 g/kg/d)**
  - IV glucose
  - Carbohydrates from enteral formulas
- **Lipids (25-35 % of energy – 1-1.5 g/kg/d)**
  - Triglycerides from enteral formulas / parenteral solutions
- **Nitrogen (15-20 % of energy – 1.2-1.8 g/kg/d)**
  - Intact proteins from enteral formulas
  - Aminoacids from parenteral solutions

Weight loss (%)	Protein loss * (%)
5	11.2 - 16.8
10	15.2 - 20.8
15	19.2 - 24.8
20	23.0 - 29.0
25	26.8 - 33.2

* in vivo neutron analysis. Hill G.L. J Parent Enteral Nutr 16, 197-218, 1992

La sarcopénie, wasting syndrome et cachexie/ICU AW : à ne pas confondre…

La sarcopénie : perte involontaire et physiologique de la masse, de la qualité et de la force des muscles squelettiques se produisant au cours du vieillissement

Le « wasting syndrome » : perte de poids (à la fois masses maigres et grasses) induite par des apports alimentaires inadéquates et/ou un syndrome de malabsorption. Ex : Sida

La cachexie / ICUAW : perte de masse maigre induite par un phénomène inflammatoire. Ex : Infection généralisée

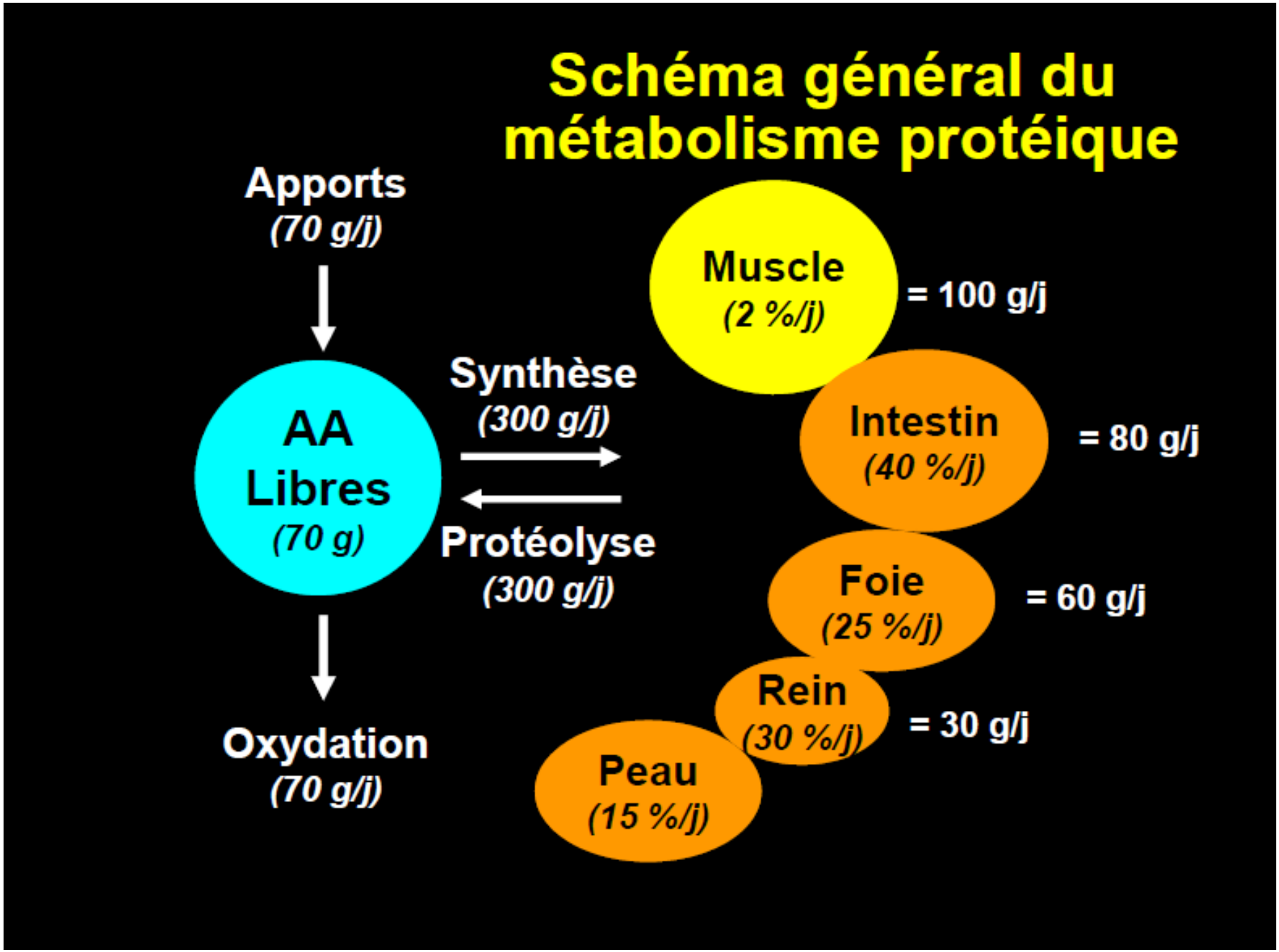


Schéma général du métabolisme protéique
Apports
(70 g/j)
AA
Libres
(70 g)
Oxydation
(70 g/j)
Synthèse
(300 g/j)
Protéolyse
(300 g/j)
Muscle
(2 %/j)
= 100 g/j
Intestin
(40 %/j)
= 80 g/j
Foie
(25 %/j)
= 60 g/j
Rein
(30 %/j)
= 30 g/j
Peau
(15 %/j)

Changes in Skeletal Muscle With Age

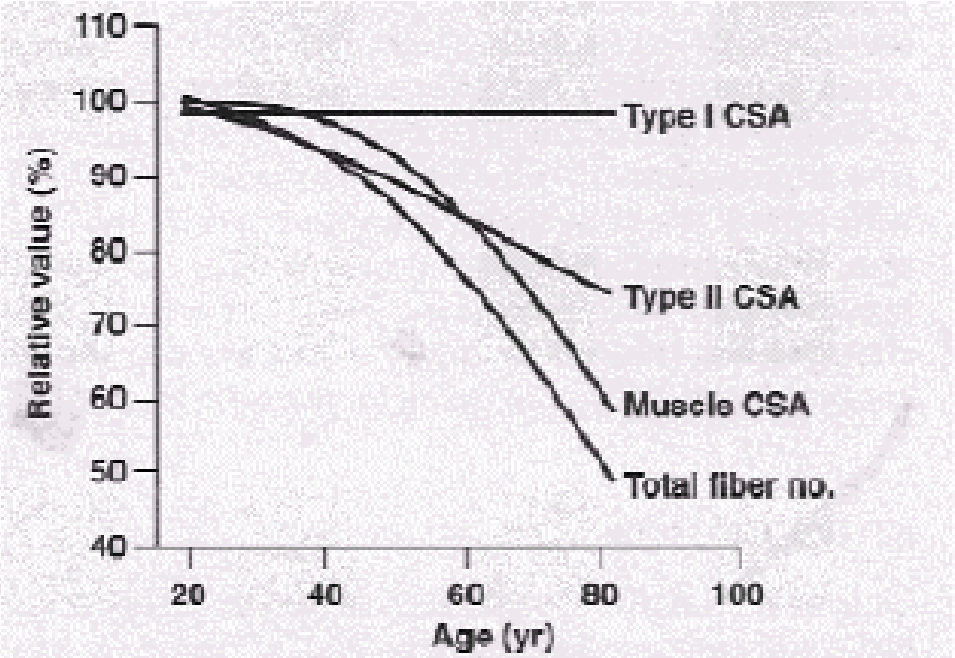


Fig. 2 - Relative changes in muscle size parameters in humans. Data are summarized from whole vastus lateralis reported by Lexell et al. (18). The decline in total muscle cross-sectional area (CSA) appears to be due to both a reduction in total fiber number and atrophy of type II fibers. The proportion of fiber types was unchanged, but due to the reduced size of type II fibers, the proportion of the total area occupied by type II fibers also declined with aging.

DEGREE OF SARCOPENIA

"sarx" – flesh

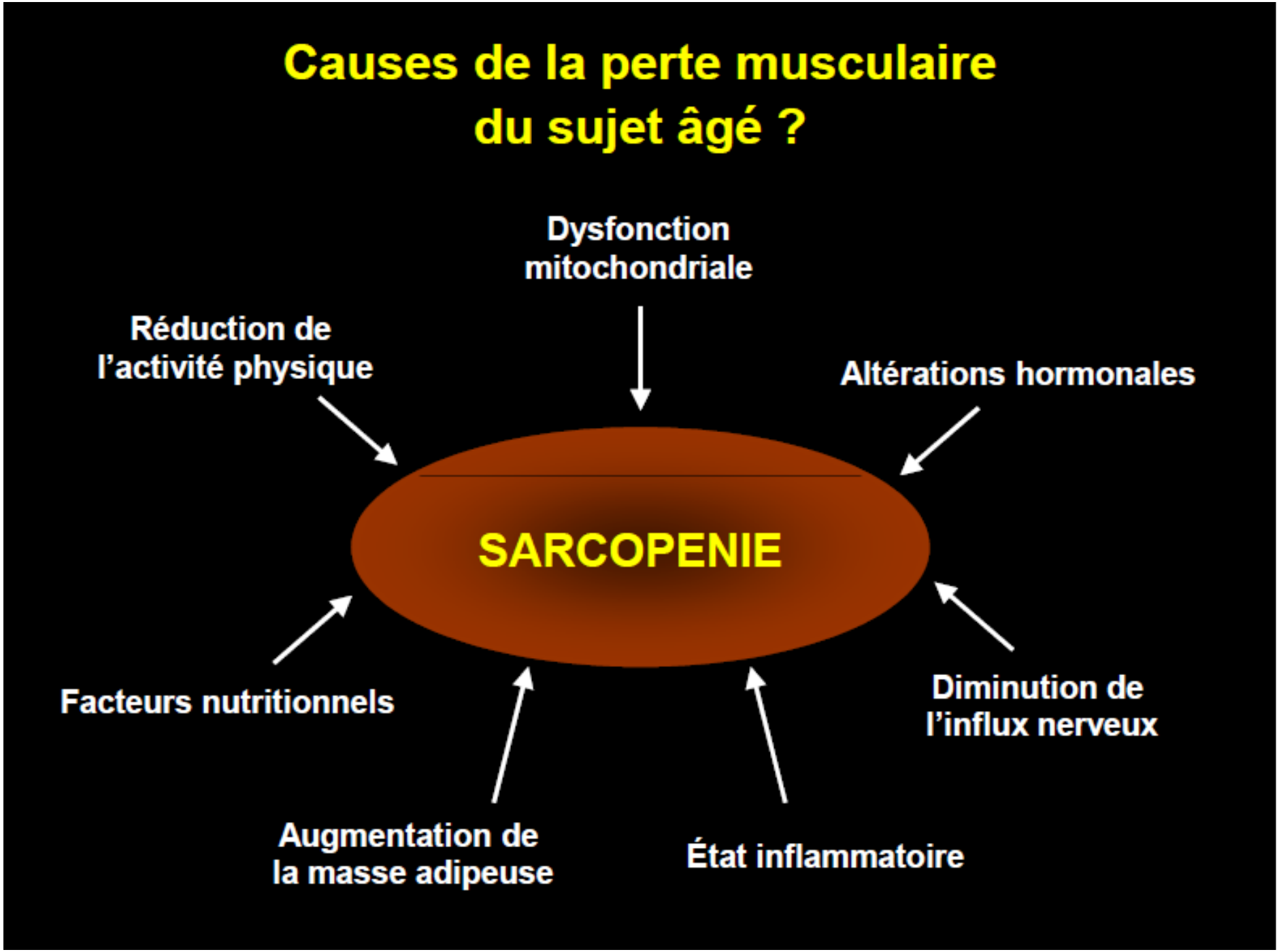
"penia" – loss or deficiency

Class I

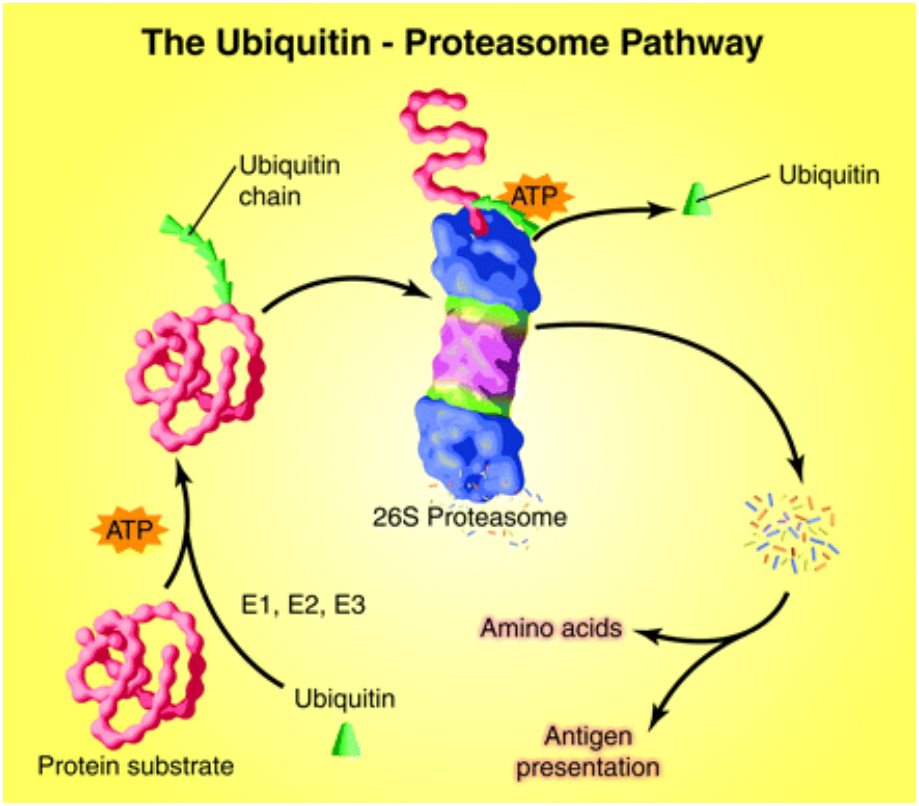
A value of lean body mass 1 to 2 standard deviations below the average value calculated in healthy, young adults.

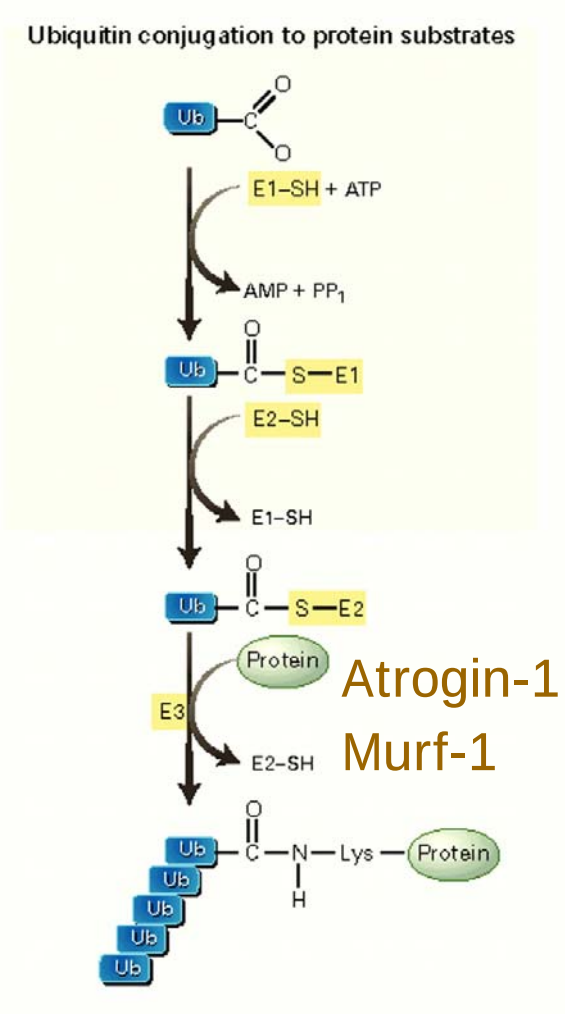
Class II

A value of lean body mass greater than 2 standard deviations below the average value calculated in healthy, young adults.


Causes de la perte musculaire du sujet âgé ?
Dysfonction mitochondriale
Réduction de l'activité physique
Altérations hormonales
SARCOPENIE
Facteurs nutritionnels
Diminution de l'influx nerveux
Augmentation de la masse adipeuse
État inflammatoire

The ubiquitin-proteasome system





Atrogin-1

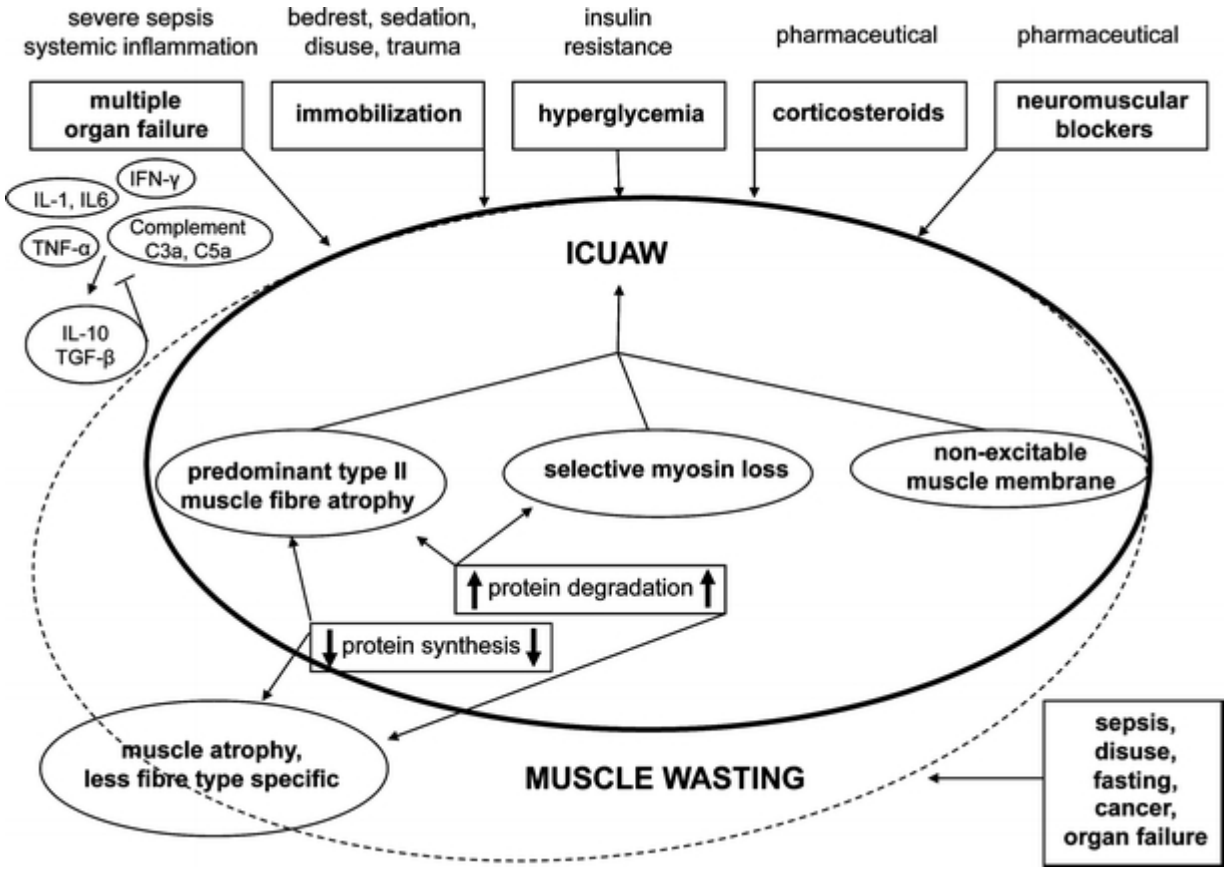
Murf-1

Lecker SH et al., 2006

Mitch M et al., 1996

Potential mechanisms of ICUAW

Schefold *J Cachex Sarcopenia Muscle.* 2010; 1: 147–157.



One-Year Outcomes in Survivors of the Acute Respiratory Distress Syndrome

Margaret S. Herridge, M.D., M.P.H., Angela M. Cheung, M.D., Ph.D., Catherine M. Tansey, M.Sc., Andrea Matte-Martyn, B.Sc., Natalia Diaz-Granados, B.Sc., Fatma Al-Saidi, M.D., Andrew B. Cooper, M.D., Cameron B. Guest, M.D., C. David Mazer, M.D., Sangeeta Mehta, M.D., Thomas E. Stewart, M.D., Aiala Barr, Ph.D., Deborah Cook, M.D., and Arthur S. Slutsky, M.D., for the Canadian Critical Care Trials Group

N Engl J Med 2003;348:683-93.

GLOBAL ASSESSMENT

At the time of discharge from the ICU, patients who survived the acute respiratory distress syndrome were severely wasted and had lost 18 percent of their base-line body weight (Fig. 2). Seventy-one percent of patients (59 of 83) returned to their base-line weight by one year. All patients reported poor function and attributed this to the loss of muscle bulk, proximal weakness, and fatigue. Most patients had

DISTANCE WALKED IN SIX MINUTES

The distance walked in six minutes improved over the 12 months after discharge from the ICU but still remained lower than the predicted value[38] (Table 3). The patients attributed exercise limitation to global muscle wasting and weakness, foot drop (as a result of nerve-entrapment syndromes that began in the ICU), immobility of large joints (heterotopic ossification[40,41]), and dyspnea. The proportion of

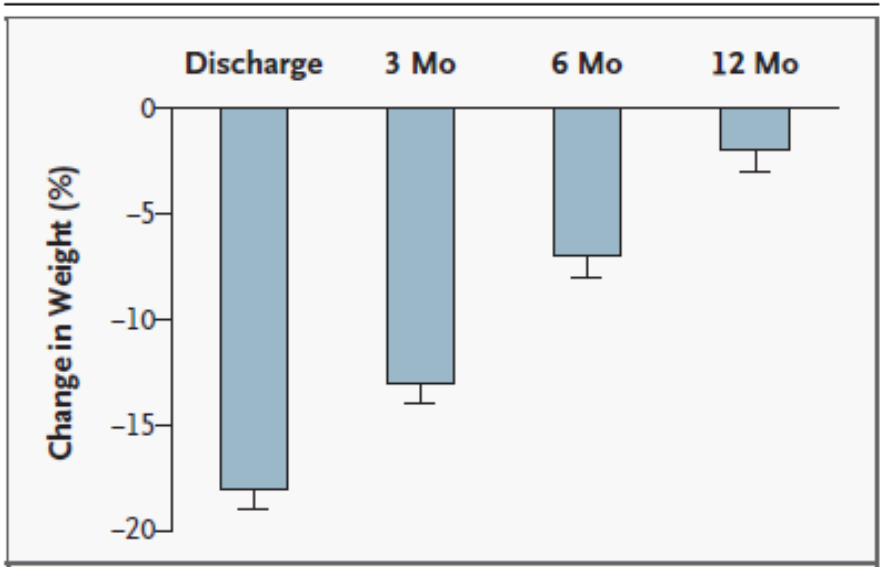


Figure 2. Mean (+SE) Change in Weight from Base Line among Patients with the Acute Respiratory Distress Syndrome at the Time of Discharge from the ICU and at 3, 6, and 12 Months.

RENDEZ-VOUS

- Update in Nutritional support – Rome 16-19 décembre - *www.intensive.org*

- Journées francophones de nutrition – Bruxelles 10-12 décembre 2014