ADI

Associazione Italiana di Dietetica e Nutrizione Clinica ONLUS
Federata FESIN - Sezione Trentino Alto Adige

Nuove frontiere nella Nutrizione Clinica

13 - 14 aprile 2012
Palalevico - Levico Terme (TN)

CHIRURGIA PANCREATICA:

ASPETTI NUTRIZIONALI

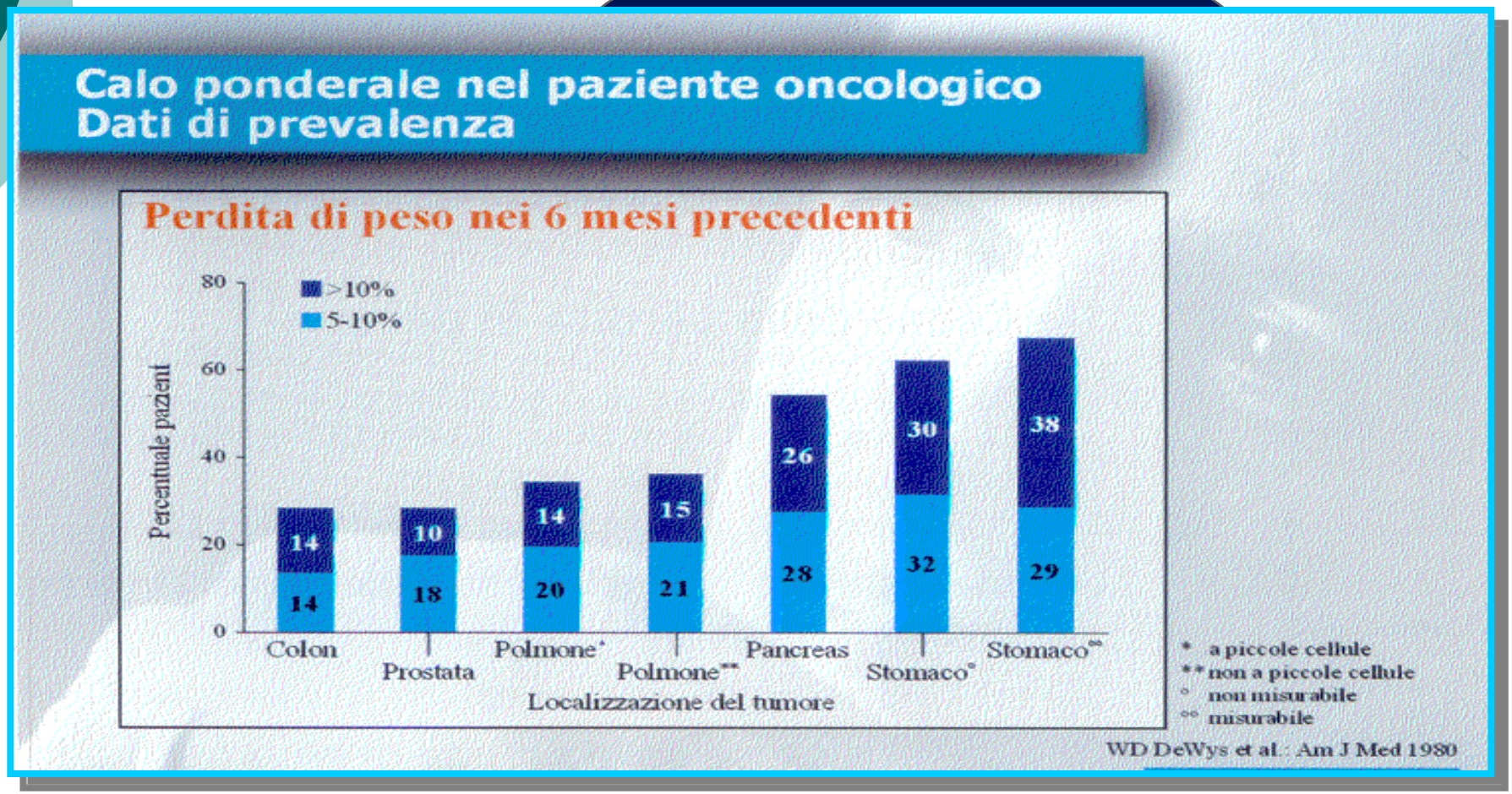
F. D'ANDREA

SCDO Dietetica e Nutrizione Clinica

A.O.U. Maggiore della Carità - Novara

Malnutrizione/cachessia neoplastica: incidenza

Sede del tumore:
++ GI superiore e capo-collo

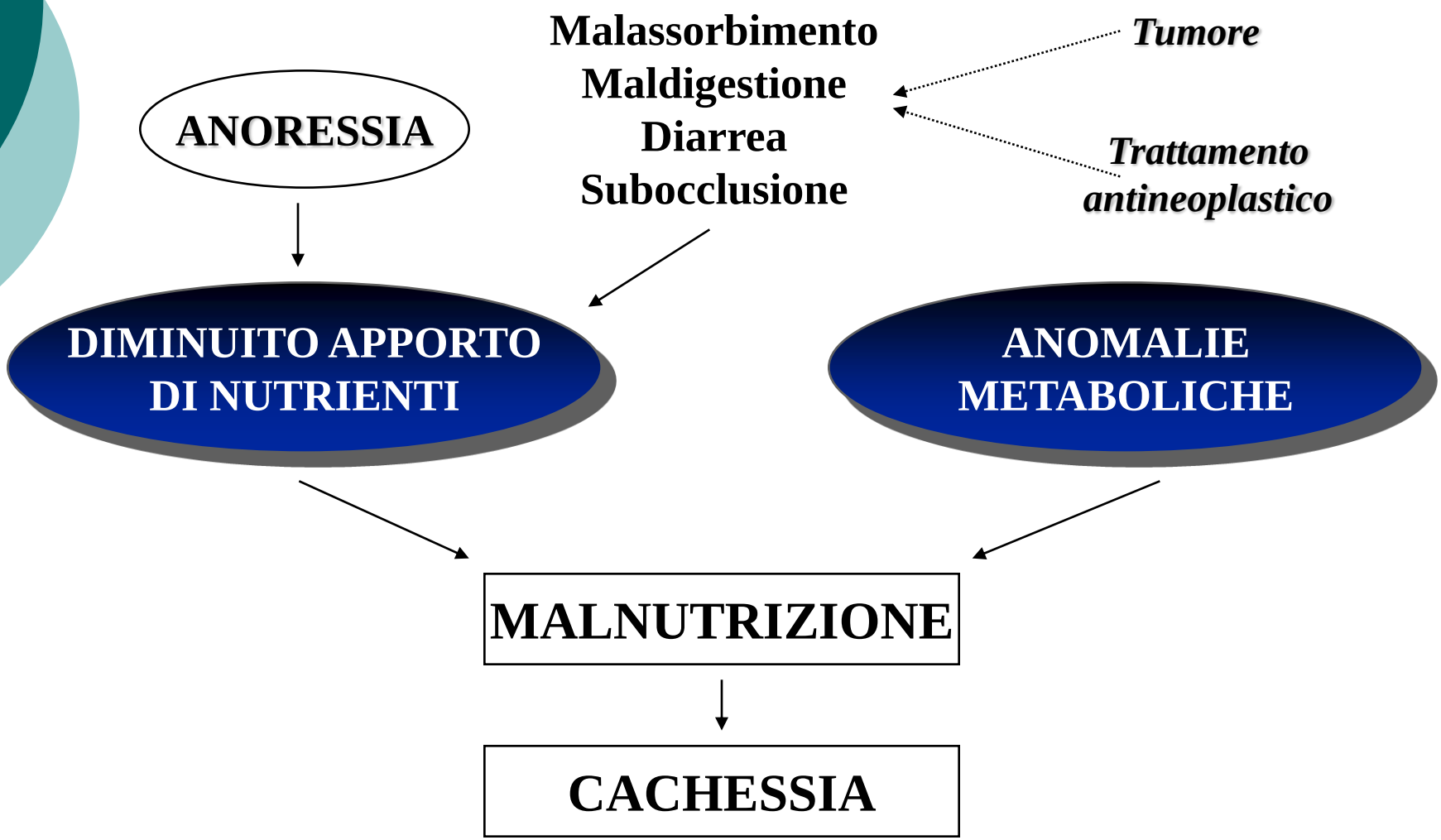


Calo ponderale nel paziente oncologico: quali le cause?

Meccaniche	Ostruzioni gastrointestinali pregiudicanti l'ingestione di cibo (es. tumori di testa e collo)
Da trattamento antineoplastico	CHEMIOTERAPIA Anoressia, nausea, vomito, diarrea, alterazioni del gusto e dell'olfatto, mucositi, stomatiti RADIOTERAPIA Anoressia, alterazioni del gusto, disfagia, mucositi, stomatiti, enteriti, costipazione, fistole, stenosi CHIRURGIA DEMOLITIVA Difficoltà digestive, malassorbimento, peggioramento dell'assunzione TRAPIANTO DI MIDOLLO OSSEO Alterazioni del gusto, della salivazione, danni intestinali
Correlate al tumore	Anoressia, precoce senso di sazietà, alterato metabolismo energetico, debolezza, perdita di massa muscolare

DE Rivadeneira et al. Cancer J Clin 1998

Malnutrizione/cachessia neoplastica: fisiopatologia



Anoressia neoplastica

PREVALENZA:

- 15 - 40% all'esordio
- fino 80% negli stadi avanzati

CAUSE:

- Modificazioni del gusto e dell'olfatto
- Disfagia, odinofagia
- Dispepsia
- Subocclusione
- Dolore
- Depressione
- Alterazioni metaboliche:

  acido lattico, triptofano, citochine, catecolamine, serotonina
- Chemio-radioterapia
- Terapia di supporto (FANS, oppiacei, ecc.)

Therapy Insight: cancer anorexia–cachexia syndrome—when all you can eat is yourself

Alessandro Laviano*, Michael M Meguid, Akio Inui, Maurizio Muscaritoli and Filippo Rossi-Fanelli

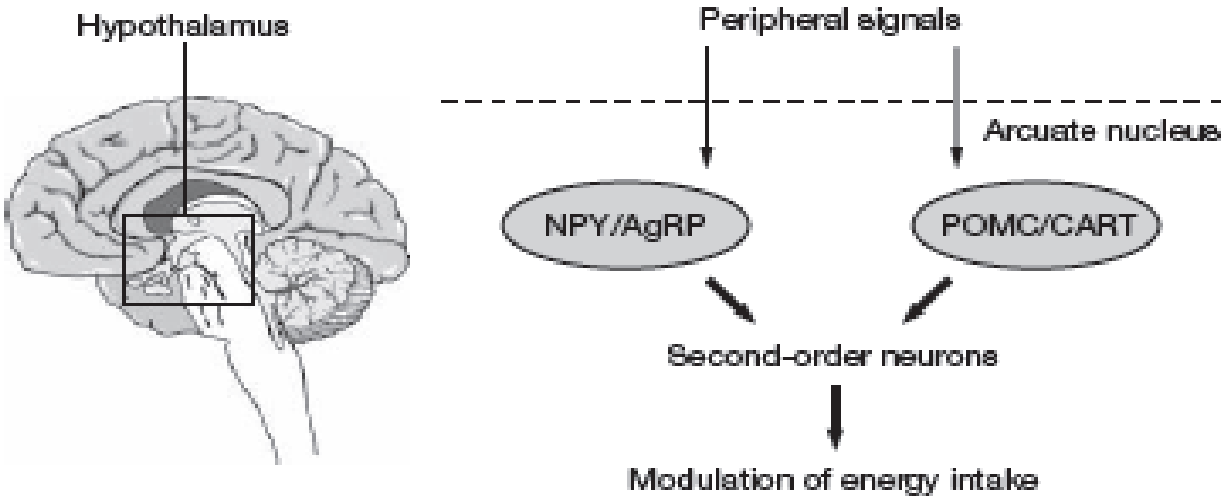


Figure 1 Diagram showing the modulation of food intake via the hypothalamus. In the hypothalamus, the arcuate nucleus receives information from the periphery and integrates these inputs to modulate food intake via second-order neurons. According to the information conveyed to the brain, peripheral signals may differentially activate/inhibit POMC/CART and NPY/AgRP neurons. When an energy deficit is signaled, anorexigenic POMC/CART neurons are inhibited and prophagic NPY/AgRP neurons are activated, resulting in increased energy intake. When an energy surplus is signaled, NPY/AgRP neurons are inhibited and POMC/CART neurons are activated.[16] AgRP, agouti-related protein; CART, cocaine-amphetamine-regulated transcript; NPY, neuropeptide Y; POMC, pro-opiomelanocortin.

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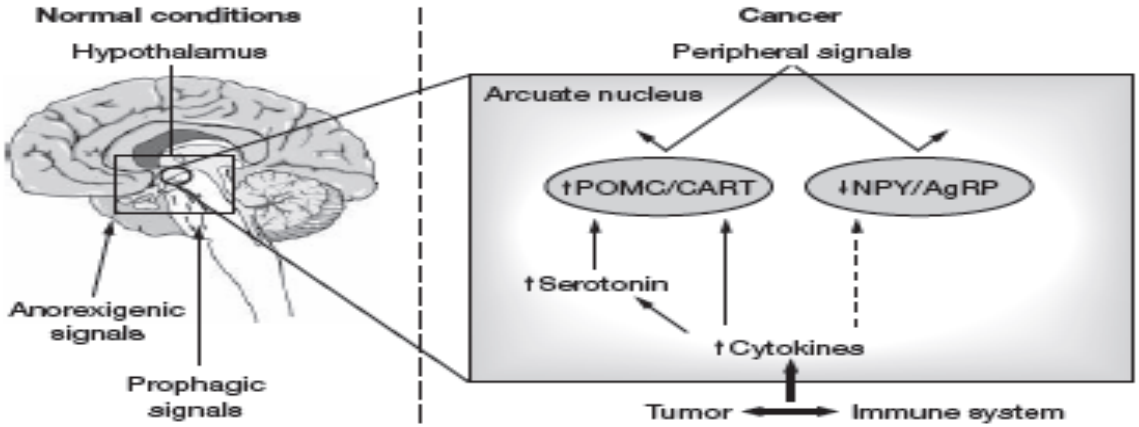


Figure 2 The balance between prophagic and anorexigenic signaling in the arcuate nucleus of the brain. Under normal conditions, energy intake is determined by the hypothalamic integration of peripheral signals conveying inputs on adiposity status, digestive processes, and metabolic profile. Some of these signals, including adipocyte-derived leptin, duodenum-derived cholecystokinin, and gut-derived peptide YY, inhibit energy intake. Other signals stimulate energy intake, including pancreas-derived insulin and stomach-derived ghrelin.[16] During cancer, tumor-host immune interaction leads to neuroimmune activation. Increased brain cytokine expression disrupts hypothalamic neurochemistry, particularly in the arcuate nucleus, where cytokines activate POMC/CART neurons, which mediate satiety and reduced food intake. This effect is at least in part mediated via increased serotonin synthesis and release. In addition, cytokines probably inhibit NPY/AgRP neurons, which mediate appetite and energy intake. These changes in hypothalamic neurochemistry lead to 'resistance' to peripheral signals informing the brain of ongoing energy deficits in the periphery. Consistent evidence indicates that cytokines may have a pivotal role in the long-term inhibition of feeding by mimicking the hypothalamic effect of excessive negative feedback signaling.[58] Tumor-induced changes in energy metabolism of hypothalamic neurons are not presented in the figure, but they are probably involved in the pathogenesis of cancer anorexia.[17] AgRP, agouti-related protein; CART, cocaine-amphetamine-regulated transcript; NPY, neuropeptide Y; POMC, pro-opiomelanocortin.

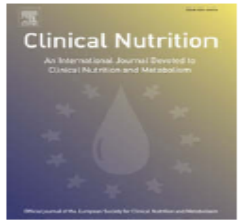
DEFINIZIONE di CACHESSIA

Clinical Nutrition (2008) 27, 793–799

available at www.sciencedirect.com

ScienceDirect

http://intl.elsevierhealth.com/journals/clnu



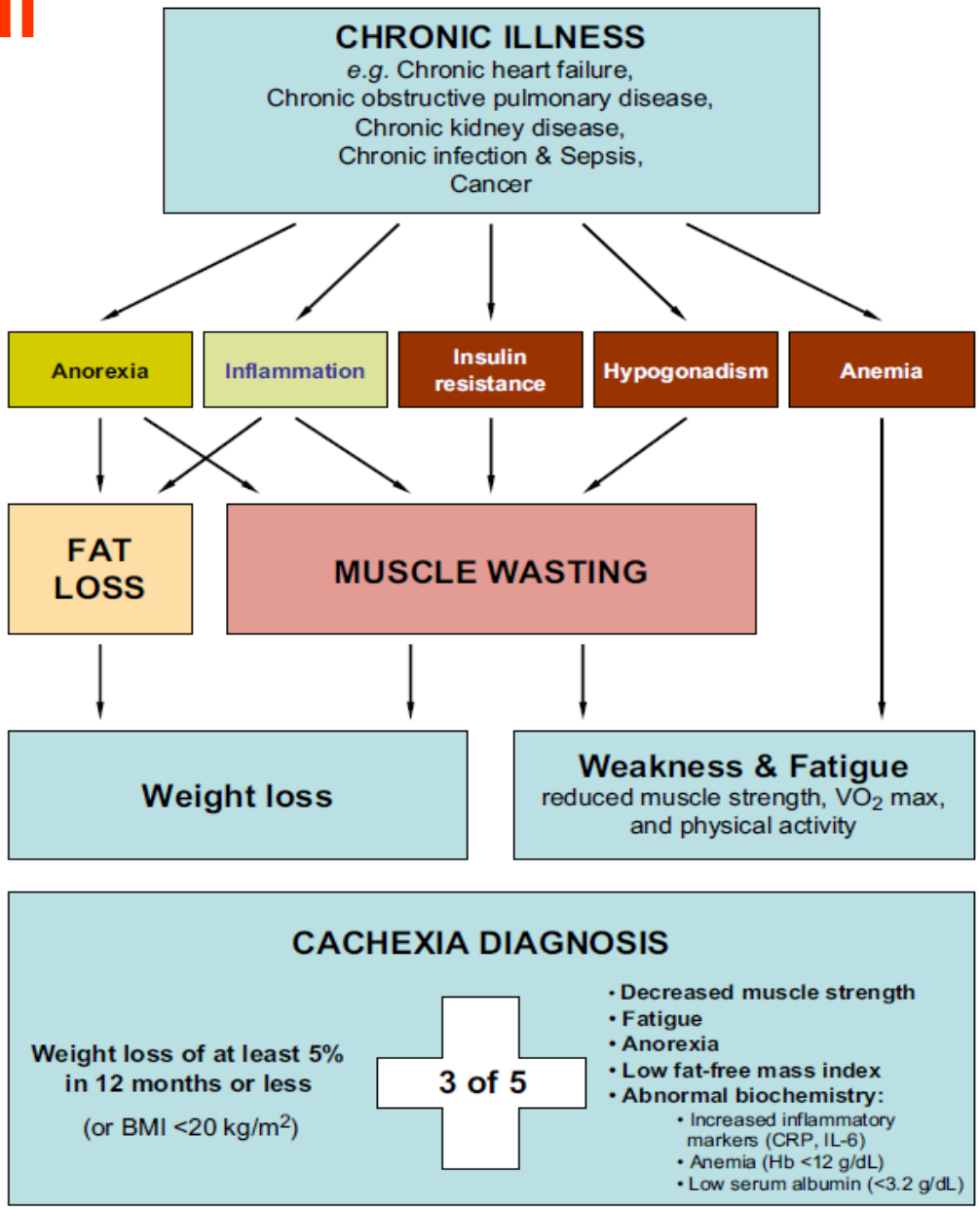
OPINION PAPER

Cachexia: A new definition

William J. Evans*, John E. Morley [a], Josep Argilés [a], Connie Bales [a], Vickie Baracos [a], Denis Guttridge [a], Aminah Jatoi [a], Kamyar Kalantar-Zadeh [a], Herbert Lochs [a], Giovanni Mantovani [a], Daniel Marks [a], William E. Mitch [a], Maurizio Muscaritoli [a], Armine Najand [a], Piotr Ponikowski [a], Filippo Rossi Fanelli [a], Morrie Schambelan [a], Annemie Schols [a], Michael Schuster [a], David Thomas [a], Robert Wolfe [a], Stefan D. Anker [a]

DEFINIZIONE di CACHESSIA

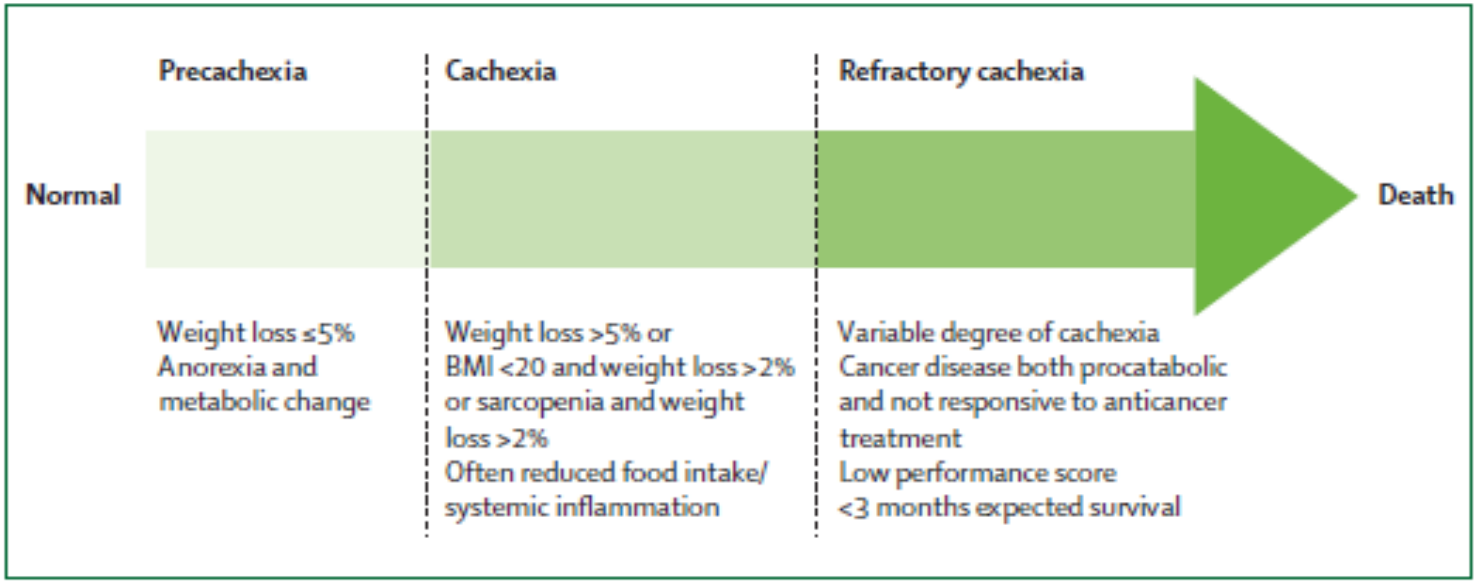
Clinical Nutrition (2008) **27**, 793–799



DEFINIZIONE di CACHESSIA

Definition and classification of cancer cachexia: an international consensus

Kenneth Fearon, Florian Strasser*, Stefan D Anker, Ingvar Bosaeus, Eduardo Bruera, Robin L Fainsinger, Aminah Jatoi, Charles Loprinzi, Neil MacDonald, Giovanni Mantovani, Mellar Davis, Maurizio Muscaritoli, Faith Ottery, Lukas Radbruch, Paula Ravasco, Declan Walsh, Andrew Wilcock, Stein Kaasa, Vickie E Baracos*



Lancet Oncol 2011; 12: 489–95

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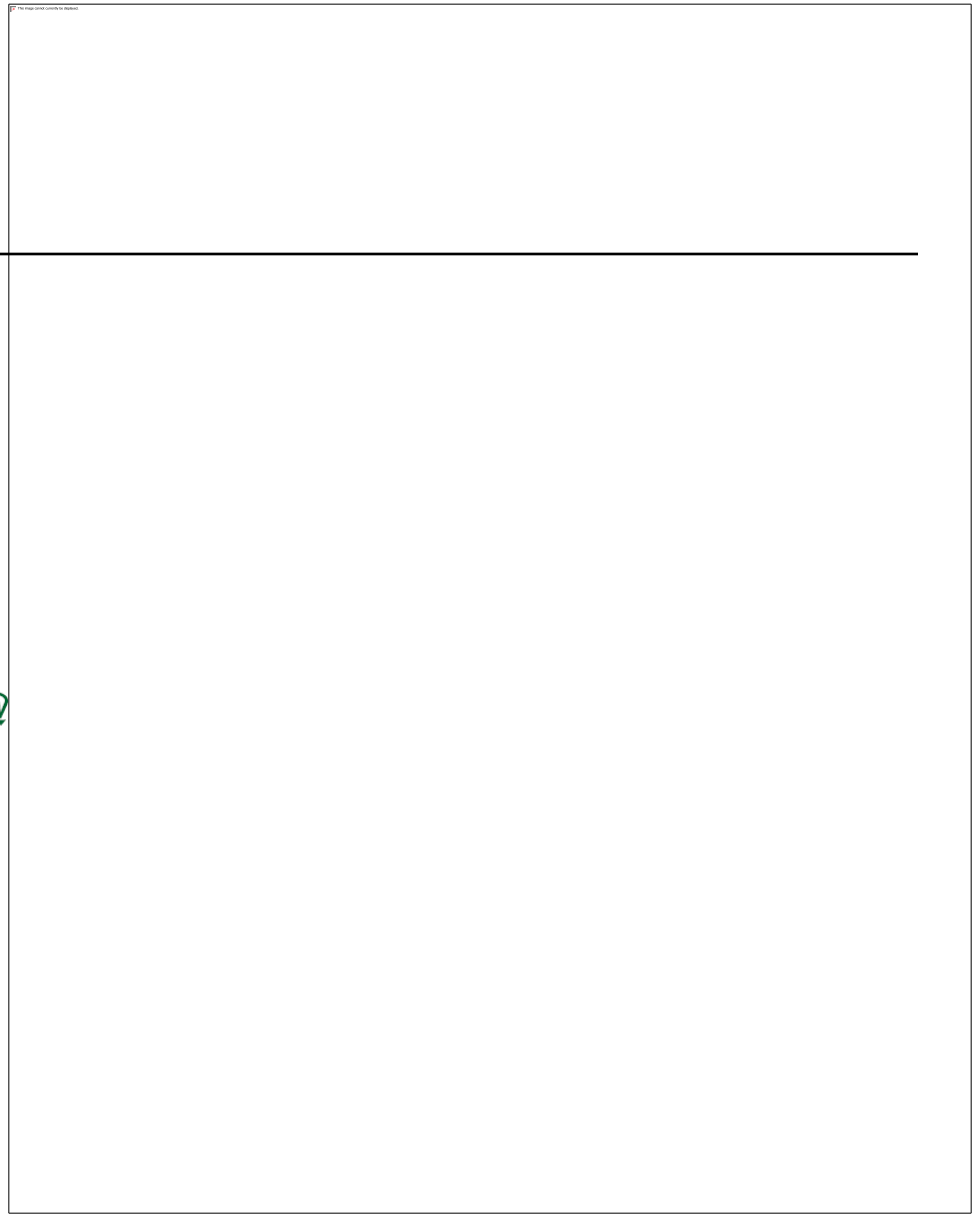
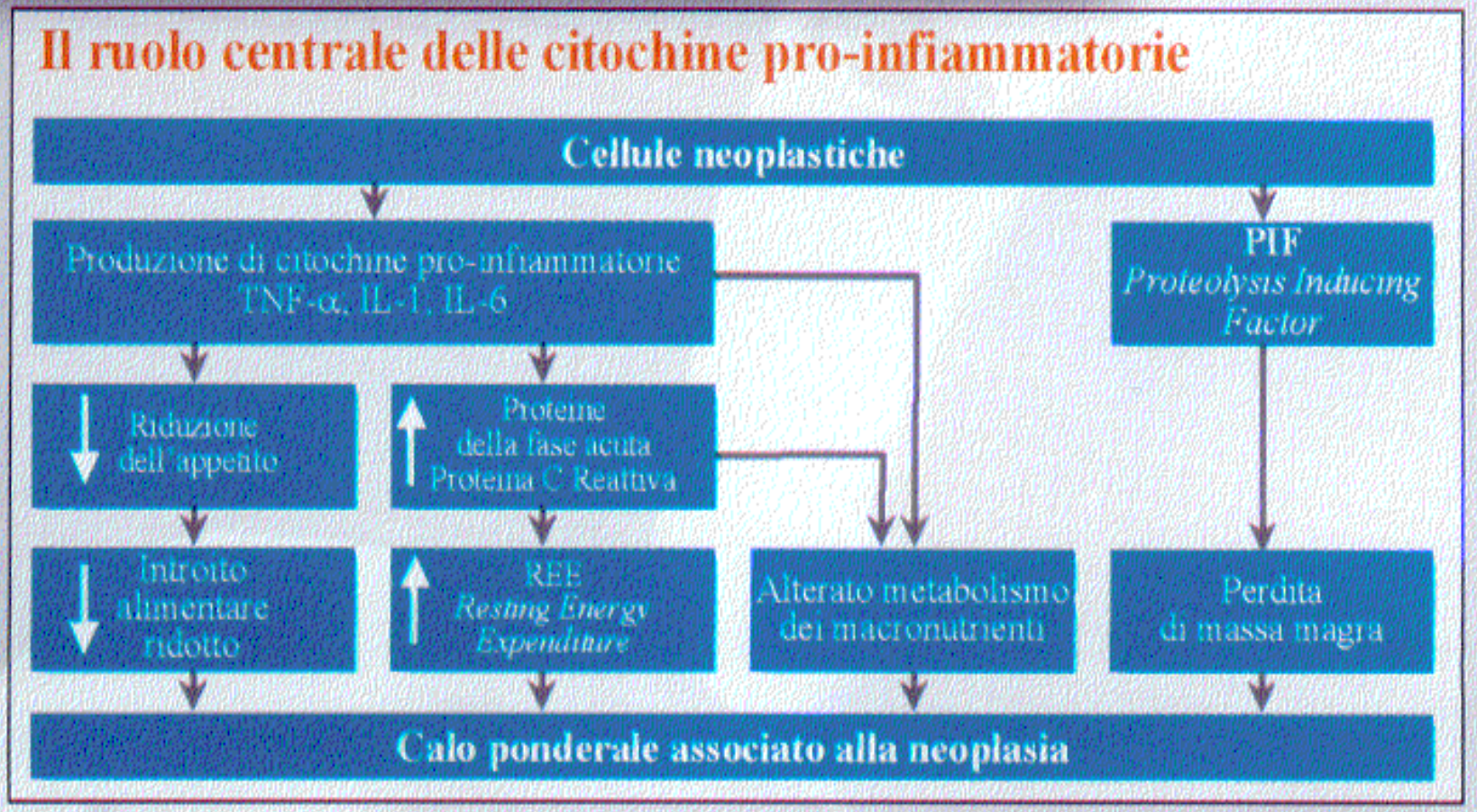
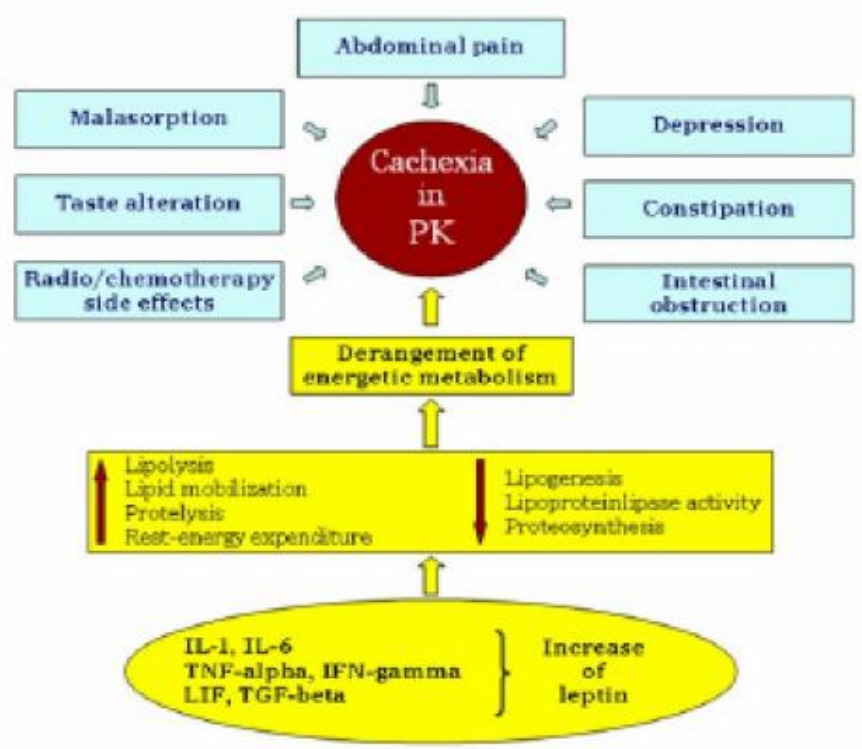


Lancet Oncol 2011; 12: 489–95

Alterazioni metaboliche nella cachessia neoplastica



D Heber et al.: Nutritional Oncology 1999; MJ Tisdale: J Natl Cancer Inst 1997
JS Falconer et al.: Ann Surg 1994; MH Torosian: Cancer J 1995


Abdominal pain
Malasorption
Depression
Cachexia in PK
Taste alteration
Constipation
Radio/chemotherapy side effects
Intestinal obstruction
Derangement of energetic metabolism
Lipolysis
Lipid mobilization
Protelysis
Rest-energy expenditure
Lipogenesis
Lipoproteinlipase activity
Proteosynthesis
IL-1, IL-6
TNF-alpha, IFN-gamma
LIF, TGF-beta
Increase of leptin

J Cachexia Sarcopenia Muscle (2010) 1:1–5
DOI 10.1007/s13539-010-0002-6

EDITORIAL

Cachexia as a major underestimated and unmet medical need: facts and numbers

Stephan von Haehling · Stefan D. Anker

Table 1 Prevalence of cachexia and definitions used in studies of diseases frequently associated with body wasting

Disease	Classification	Reference	Definitions used	Number of patients	Prevalence of cachexia (%)
Cancer	Advanced head and neck cancer	Lees [17]	Incidence of any weight loss (mean weight loss 6.5 kg~10% of body weight)	n=100	57
	Non-small cell lung cancer	DeWys et al. [18]	Weight loss >5% of body weight at diagnosis	n=3,047	36
	Pancreatic cancer, perioperative	Bachmann et al. [19]	Cachexia: weight loss >10% of the pre-illness stable body weight	n=227	40.5
	Pancreatic cancer	DeWys et al. [18]	Weight loss >5% of body weight at diagnosis	n=3,047	54
	Colorectal cancer	DeWys et al. [18]	Weight loss >5% of body weight at diagnosis	n=3,047	28
Chronic heart failure	Ambulatory stable disease	Anker et al. [20]	Cachexia: weight loss >7.5% over at least 6 months	n=171	16
	Outpatients participating in the SOLVD trials	Anker et al. [20]	Cachexia: weight loss >5% over at least 6 months	n=1,929	42
Chronic kidney disease	Advanced CKD with or without haemodialysis	Mak & Cheung [21]	Malnutrition-inflammation-cachexia syndrome		30–60
Chronic obstructive pulmonary disease (COPD)	Outpatients with moderate to severe COPD	Koehler et al. [22]	Cachexia: weight loss >7.5%	n=103	33
		Vermeeren et al. [23]	Nutritional depletion: BMI≤21 kg/m^2 and/or fat-free mass index≤15 kg/m^2 (women) or≤16 kg/m^2 (men)	n=389	27
		Wilson et al. [24]	Malnutrition: less than 90% of ideal body weight	n=779	35
	Patients admitted for pulmonary rehabilitation	Schols et al. [25]	Malnutrition: less than 90% of ideal body weight	n=255	35
Rheumatoid arthritis		Elkan et al. [26]	Rheumatoid cachexia: fat-free mass index below the 25th percentile and fat mass index above the 50th percentile	n=80	m: 26 f: 18
		Roubenoff et al. [27]	Measurement of body cell mass	n=24	67

Anomalie metaboliche

	DIGIUNO		CACHESSIA	
Spesa energetica		↓		↑ = ↓
Metabolismo proteico				
Turnover proteico totale	↓		↑	
Catabolismo proteico muscolare	↓		↑	
Sintesi proteica muscolare		↓		↓
Sintesi proteica epatica	↓		↑ (pr. fase acuta)	
Metabolismo glucidico				
Turnover totale del glucosio		↓		↑
Gluconeogenesi epatica	↓		↓	
Attività del ciclo di Cori	=		↑	
Sensibilità all'insulina	↓		↓	
Metabolismo lipidico				
Lipolisi	↑		↑	
Lipogenesi	=		↑ = ↓	
Attività lipasi lipoproteica				
Livelli sierici acidi grassi liberi	=		↑	

Cachessia: segni e sintomi

- Anoressia
- Ipofagia
- Astenia
- Sazietà precoce
- Marcato calo ponderale
- Anemia
- Edema
- Cute pallida e atrofica
- Facies emaciata
- Muscolatura scheletrica gravemente depauperata
- Forte diminuzione dei depositi adiposi sottocutanei

Malnutrizione/cachessia: conseguenze

PRIMARIE

- Ritardata cicatrizzazione delle ferite e aumento delle deiescenze
- Immunodepressione (aumentato rischio di infezioni)
- Squilibri idroelettrolitici
- Decubiti
- Alterazioni intestinali strutturali/morfologiche e funzionali
- Riduzione della funzionalità muscolare
- Disfunzioni respiratorie

SECONDARIE

- Aumentata morbilità
- Peggior rapporto costo/beneficio e rischio/beneficio del trattamento antineoplastico
- Maggiore durata della degenza
- Minore qualità di vita
- Maggiore mortalità

AUMENTO DEI COSTI DI GESTIONE SANITARIA !

Sindrome Anoressia – Cachessia

NEOPLASIE DEL PANCREAS

- Sopravvivenza a 5 anni:
  - 25% dopo chirurgia + terapia adiuvante
  - 4% in pazienti non operabili

Kleff J et al – Pancreas, 2006

- Fino a 80% dei pazienti presentano cachessia al momento della diagnosi

Factors contributing to altered nutritional status and metabolism in patients with pancreatic disease and undergoing PD

Cancer/chronic pancreatitis:

- Anorexia (reduced food intake, abdominal pain, vomiting)
- Muscle protein wasting (Cachexia)
- Inflammation
- Diarrhea/steatorrhea
- Exocrine insufficiency (gland sclerosis or substitution with neoplastic cells, wirsung obstruction, enzyme inactivation)
- Endocrine disturbance (hyper- or hypoglycemia)
- Altered bile composition
- Chemoradiation
- Alcohol abuse

Surgery:

- Altered gastric emptying and pH
- Septic complications
- Poor mixing of food with bile and pancreatic enzymes
- Exocrine insufficiency
- Endocrine disturbance
- Delayed or increased intestinal transit time

RAZIONALE

PERCHÉ NUTRIRE IL PAZIENTE CHIRURGICO ?

Dalla Letteratura …….

<u>un dato sicuro e confermato</u>:

- il paziente chirurgico è frequentemente a rischio di alterazione dello stato nutrizionale
- la malnutrizione si associa a un maggior rischio di mortalità (Studley HO. JAMA 1936 !!!) e di complicanze postoperatorie (Giner M. Nutrition 1996) con conseguente aumento dei tempi di degenza e dei costi

MALNUTRIZIONE

CAUSE

- **Ridotto introito di nutrienti**

  a. nel preoperatorio

  - malattie ostruttive
  - sintomi indotti dall'alimentazione
  - fattori psicologici

  b**. nel postoperatorio**

  - ileo
  - complicanze
  - resezioni gastrointestinali
  - reintervento

MALNUTRIZIONE

CAUSE

- **Aumentate perdite di nutrienti**
  - vomito, diarrea, aspirazione nasogastrica
  - maldigestione: gastrectomia, insufficienza pancreatica, ecc
  - malassorbimento: malattie infiammatorie intestinali, intestino corto, enterite attinica, ecc
  - emorragie, fistole, ferite, decubiti, drenaggi

- **Aumentato fabbisogno di nutrienti**
  - riparazione tissutale
  - **risposta metabolica** allo "stress"

RISPOSTA METABOLICA

FASI: ***(Cuthberson 1942)***

- **"Ebb phase"**
  - dura alcune ore
  - riduzione del consumo di ossigeno, del consumo energetico e della temperatura corporea
  - scarso impatto sullo stato nutrizionale

- **"Flow phase"**
  - dura da pochi giorni a settimane
  - **ipermetabolismo** (aumento del consumo di ossigeno, del consumo energetico e della temperatura corporea) e **ipercatabolismo**
  - rapido depauperamento delle riserve glucidiche nel fegato, lipidiche nel tessuto adiposo e proteiche nel muscolo scheletrico

RISPOSTA METABOLICA

COMPRENDE:

- **Risposta neuroendocrina**
  - ipersecrezione ormoni catabolici ad azione anti-insulinica
  - insulino-resistenza (nonostante ipersecrezione insulinica)
  - riduzione livelli circolanti di ormoni tiroidei

- **Risposta infiammatoria sistemica**
  - sintesi di prostaglandine, leucotrieni, trombossani
  - sintesi di citochine

RISPOSTA INFIAMMATORIA SISTEMICA

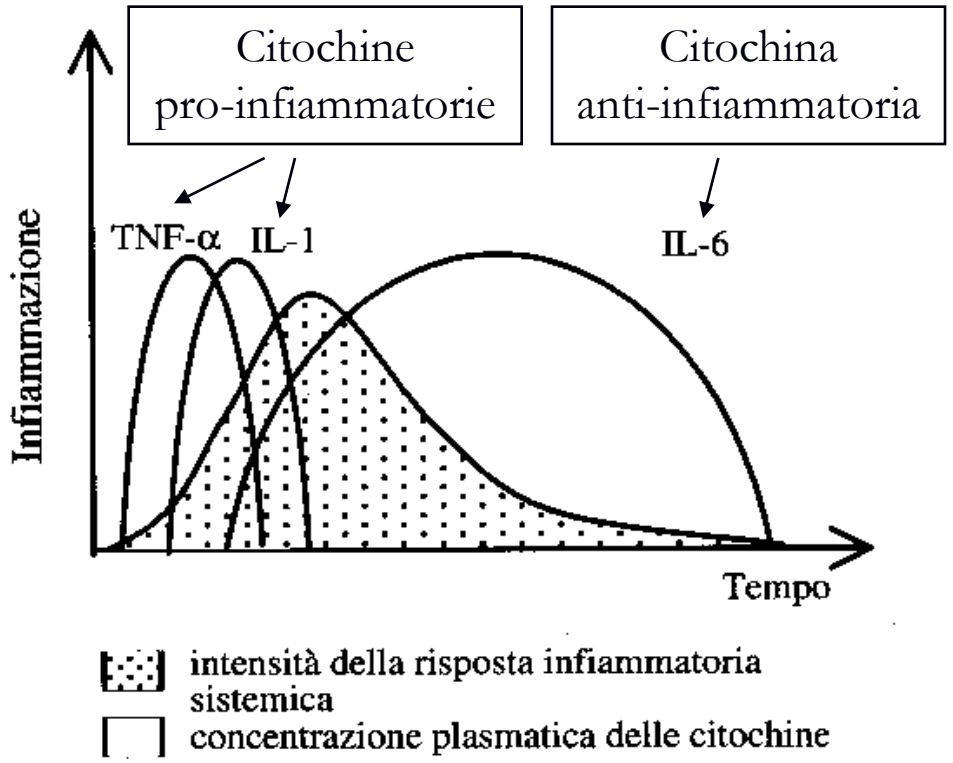
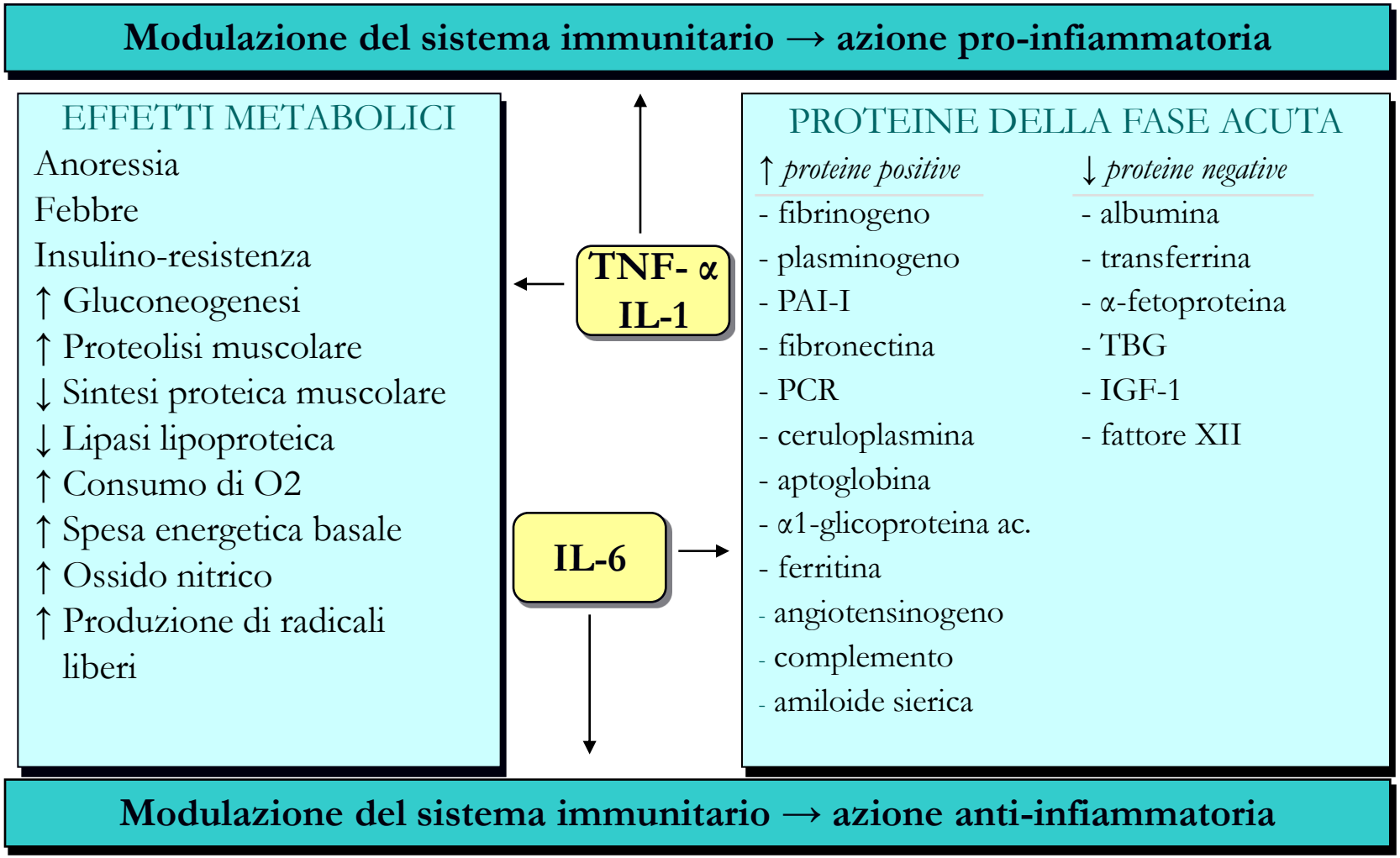


FIG. 4.9 – Tipica attivazione a cascata del sistema delle citochine nello "stress" metabolico acuto[286,307].

Negli stati clinici più gravi come la sepsi ed il politrauma la risposta infiammatoria sistemica può essere molto intensa nella prima fase, tanto da configurare una sindrome clinica ben definita (SIRS) che può sfociare nello shock o nella MOF

RISPOSTA INFIAMMATORIA SISTEMICA



RISPOSTA METABOLICA

ALTERAZIONI METABOLICHE:

- **Metabolismo lipidico**

  - aumento della lipolisi e mobilizzazione di acidi grassi liberi (utilizzati a scopo energetico)

- **Metabolismo proteico**

  - aumento della proteolisi muscolare e utilizzo degli aminoacidi a scopo energetico, per la gluconeogenesi epatica e per la sintesi delle proteine della fase acuta

- **Metabolismo glucidico**

  - aumento dei livelli ematici di glucosio da ipersecrezione di cortisolo e catecolamine ed insulino-resistenza

  - aumentata utilizzazione di glucosio nel sistema immunitario, nervoso centrale, emopoietico e nel tessuto di riparazione

RISPOSTA METABOLICA

" **Elective operations not associated with infection or other complications produce transient, modest increases in nutrient requirements**, wich are of no clinical consequence. Resting Energy Expenditure may be increased as little as 10 % in such patients for as little as 2 days. "

ASPEN 1998

RISPOSTA METABOLICA

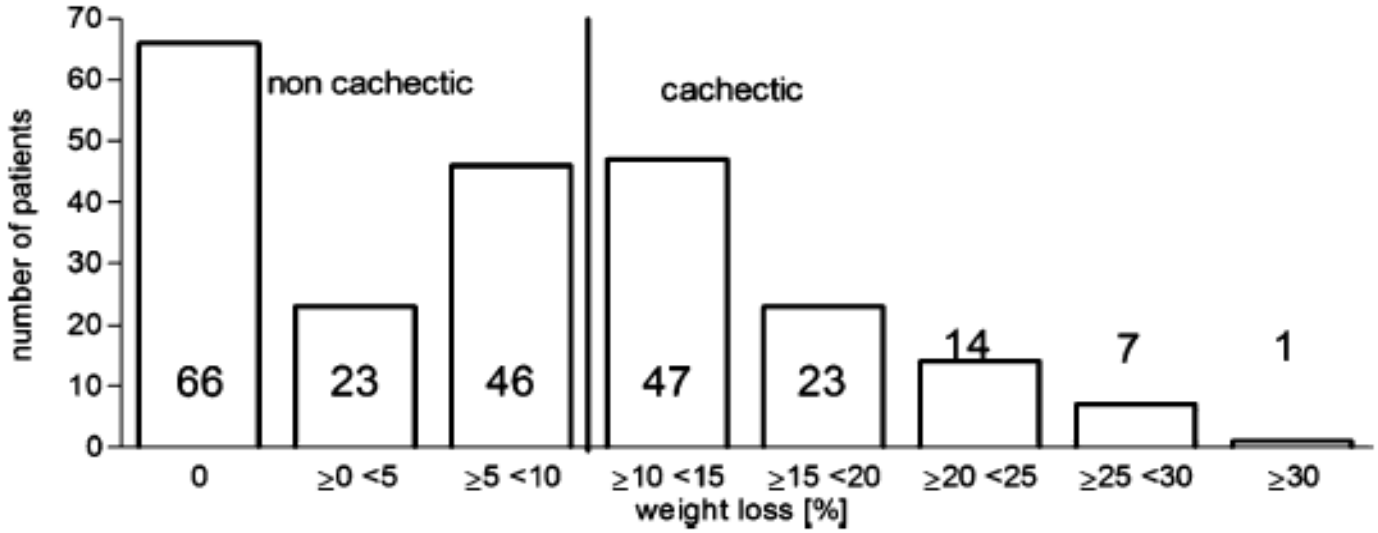
" Greater metabolic responses occur in proportion to the **severity of the stress**. Failure to recover promptly, leading to the need for reoperation, prolonged sepsis, or other major stress may produce critical illness. Preoperative hypermetabolism may be induced by long-term inflammatory or infectious conditions. Examples include abscesses associated with long-term inflammatory bowel disease or diverticulitis. "

ASPEN 1998

J Gastrointest Surg (2008) 12:1193–1201
DOI 10.1007/s11605-008-0505-z

Cachexia Worsens Prognosis in Patients with Resectable Pancreatic Cancer

Jeannine Bachmann · Mathias Heiligensetzer · Holger Krakowski-Roosen · Markus W. Büchler · Helmut Friess · Marc E. Martignoni



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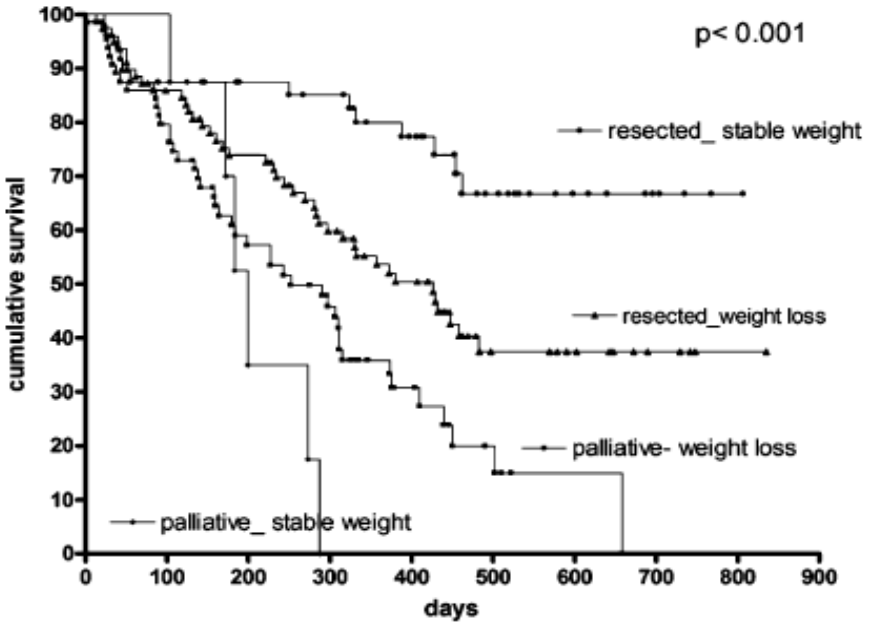
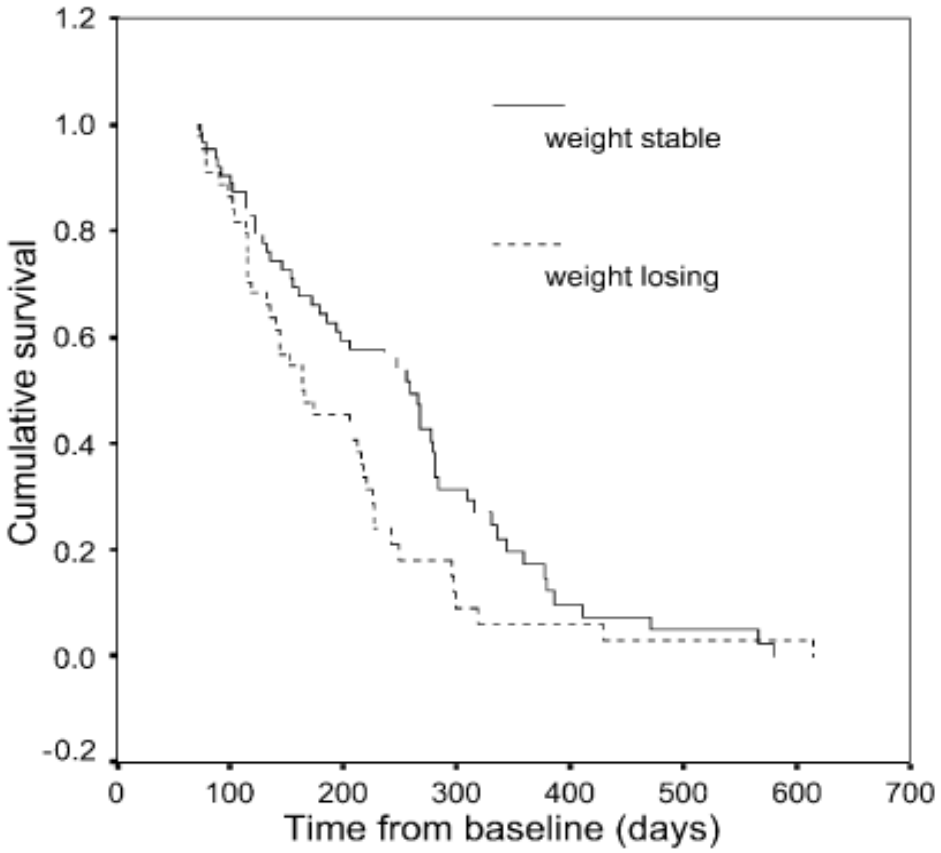


Weight stabilisation is associated with improved survival duration and quality of life in unresectable pancreatic cancer ☆

Wendy Davidson[a,b,*], Susan Ash[a], Sandra Capra[b], Judith Bauer[b,c], on behalf of the Cancer Cachexia Study Group

Clinical Nutrition (2004) 23, 239–247

Clinical Nutrition



Research article

Open Access

Pancreatic cancerrelated cachexia: influence on metabolism and correlation to weight loss and pulmonary function

Jeannine Bachmann[1], Knut Ketterer[1], Christiane Marsch[2], Kerstin Fechtner[3], Holger Krakowski-Roosen[4], Markus W Büchler[2], Helmut Friess[1] and Marc E Martignoni*[1]

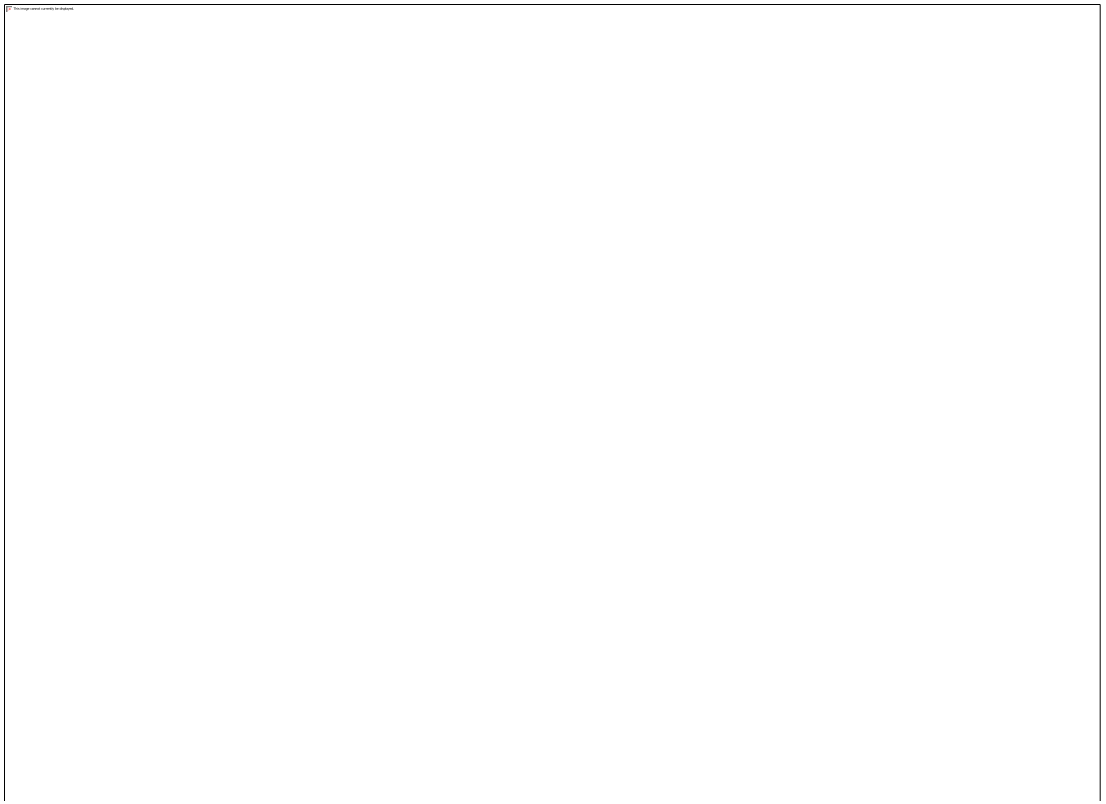


Figure 2
Kaplan-Meier survival curve in 198 patients with pancreatic cancer with and without cachexia ($p < 0.001$) showing a significant longer survival in patients without cachexia.

Published: 28 July 2009

BMC Cancer 2009, 9:255 doi:10.1186/1471-2407-9-255

Valutazione nutrizionale

OBIETTIVI

1. Identificazione dei pazienti malnutriti o a rischio di malnutrizione *(screening nutrizionale)*
2. Conferma della presenza di malnutrizione ed inquadramento della gravità e delle cause *(diagnosi nutrizionale)*
3. Identificazione dei pazienti a rischio di sviluppare complicanze in corso di trattamento antineoplastico *(prognosi nutrizionale)*
4. Definizione delle modalità d'intervento *(terapia nutrizionale)*
5. Valutazione dell'efficacia della terapia nutrizionale in atto *(monitoraggio nutrizionale)*

La valutazione nutrizionale deve essere integrata da una completa valutazione dello stato di validità (performance status) e della qualità di vita

Screening nutrizionale

INDICE DI RISCHIO NUTRIZIONALE

Cognome ..... Nome. .....

Data di nascita ..... Età ..... Altezza (cm) ..... Peso (Kg) .....

Reparto ..... Letto ..... Data ingresso.....

Selezionare un solo punteggio per ogni sezione

1

ETÀ 0-17 ANNI	PUNTEGGIO
PESO ATTUALE	
Peso ideale (per l'altezza)	0
90-99% del peso ideale	2
80-99% del peso ideale	4
< 79% del peso ideale	6

ADULTI (≥ 18 anni)	PUNTEGGIO
CALO PONDERALE ULTIMI 3 MESI	
No	0
0-3 Kg	1
> 3-6 Kg	2
≥ 6Kg	3

2

Omettere la sezione 2
se età 0-17 anni

BMI (Body Mass Index)	
≥ 20	0
18-19	1
15-17	2
< 15	3

3

APPETITO

- Buono, consuma più di tre pasti al giorno 0
- Scarso, avanza più della metà dei pasti 2
- Assente, incapace di mangiare, nulla per bocca (per più di 4 pasti) 3

4

CAPACITÀ DI MANGIARE/TRATTENERE IL CIBO

- Nessuna difficoltà, in grado di alimentarsi autonomamente. Non diarrea o vomito 0
- Difficoltà nel manipolare alimenti (es: impiego di utensili speciali) 1
  Vomito, frequente rigurgito, lieve diarrea
- Difficoltà nella deglutizione, necessità di alimenti di consistenza modificata 2
  Problemi di dentatura e/o masticazione che compromettono l'assunzione di cibo.
  Alimentazione rallentata. Moderato vomito e/o diarrea (1-2 episodi/die nel bambino)
  Necessità di aiuto ad alimentarsi
- Incapacità ad assumere alimenti per bocca. Incapacità a deglutire (disfagia totale) 3
  Severo vomito e/o diarrea (>2 episodi/die nel bambino). Malassorbimento.

5

FATTORE DI STRESS

- **Nessuno** 0
- **Lieve** Chirurgia minore. Infezioni minori 1
- **Moderato** Malattie croniche. Chirurgia maggiore. Infezioni. Fratture. Piaghe da decubito. Malattie infiammatorie intestinali. Altre malattie gastrointestinali 2
- **Severo** Traumi/fratture/ustioni multiple. Ulcere/decubiti multipli. Sepsi severe. Neoplasie maligne 3

TOTALE

Reilly JJ

Arch Dis Child 1995

European Review for Medical and Pharmacological Sciences

Role of nutrition in gastrointestinal oncological patients*

R. DI LUZIO, S. MOSCATIELLO, G. MARCHESINI

Clinical Dietetics, "Alma Mater Studiorum" University, Bologna (Italy)

2010; 14: 277-284

Table II. Most commonly used tests for the assessment of the nutritional status in oncological patients.

Subjective Global Assessment (SGA)[16] – Based on:
- Medical history (weight change, dietary intake, gastrointestinal symptoms, functional impairment)
- Physical examination (muscle wasting, subcutaneous fat loss, edema)

Malnutrition Universal Screening Tool (MUST) for adults[17] – Based on:
- Body Mass Index
- Weight loss in 3-6 months
- Nutritional intake in the last few days

Nutritional Risk Screening (NRS 2002)[18] – Based on:
- Body Mass Index
- Weight loss in the last 3 months
- Dietary intake in the last week
- Severity of disease (e.g., intensive therapy)
- Age

Mini Nutritional Assessment (MNA)[20] – Based on:
- Food intake in the last 3 months
- Weight loss in the last 3 months
- Mobility
- Psychological stress or acute disease in the last 3 months
- Neuropsychological problems
- Body Mass Index or calf circumference

Nutritional Risk Index (NRI)[19] – Based on:
- Serum albumin
- Ratio of current weight to usual weight

Note that the majority of test were not specifically developed for oncological patients, and may be confidently used in any patient at risk of malnutrition.

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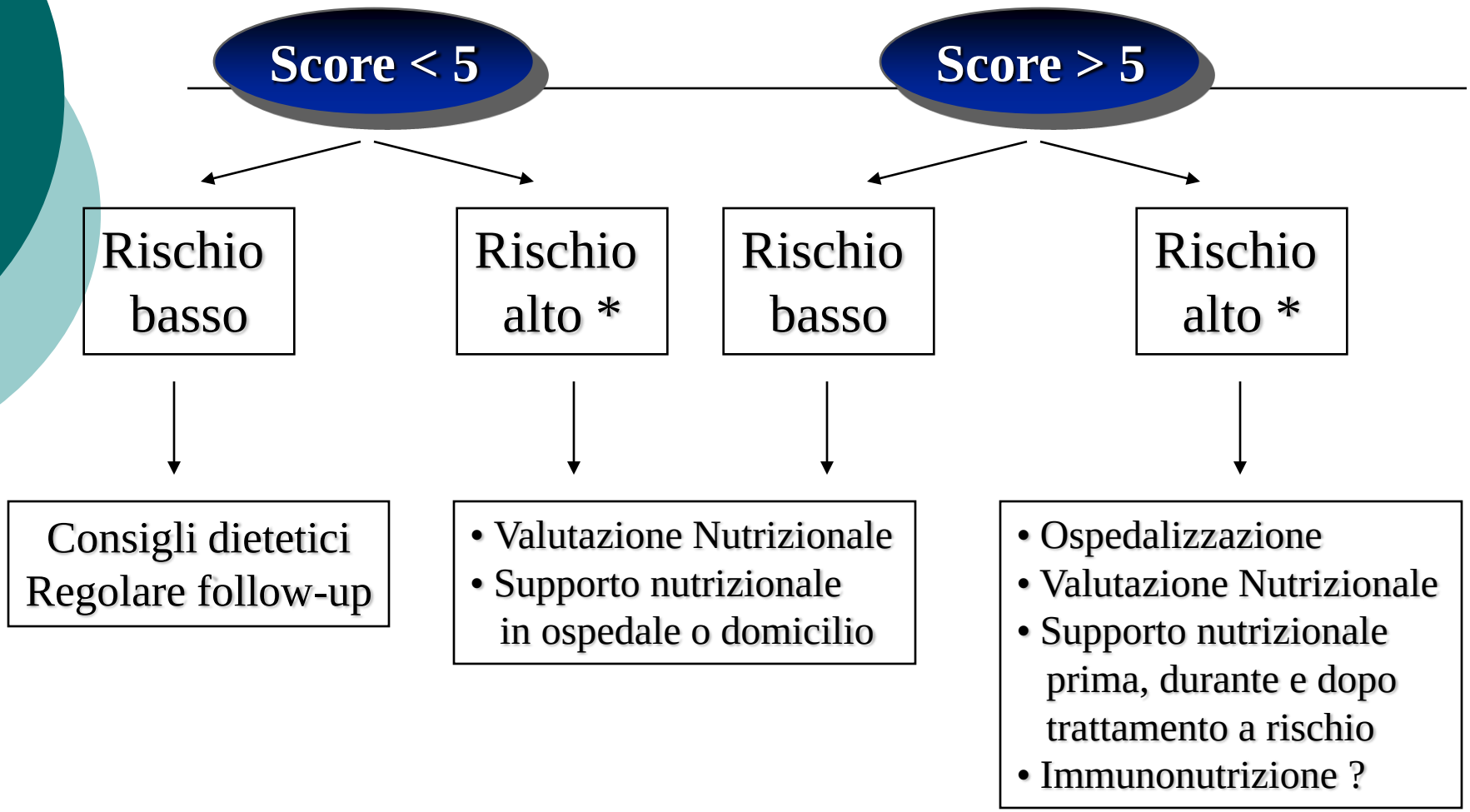
A.S.P.E.N. Clinical Guidelines: Nutrition Support Therapy During Adult Anticancer Treatment and in Hematopoietic Cell Transplantation

David Allen August, MD[1]; Maureen B. Huhmann, DCN, RD, CSO[2]; and the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) Board of Directors

Citation Design Level	Assessment	Subjects	Results
Read et al[28] (2005) Time series Level: III	MNA vs PGSGA; cancer patients	157	Both tools reliably detected malnutrition; MNA lacks specificity
Sungurtekin et al[30] (2004) Cross-sectional Level: III	SGA vs NRI; abdominal surgery patients	100	Both tools reliably detected malnutrition and predicted postoperative complications (length of stay)
Bauer et al[26] (2003) Cross-sectional Level: V	MUST vs SGA; cancer patients	65	MUST had low sensitivity (59%) and specificity (75%)
Bauer et al[27] (2002) Cross-sectional Level: V	PGSGA vs SGA; cancer patients	71	PGSGA had 98% sensitivity and 82% specificity in predicting SGA categories
Ferguson et al[39](1999) Cross-sectional Level: V	MST vs SGA; cancer patients undergoing XRT	106	MST had 100% sensitivity and 81% specificity in predicting SGA category
Isenring et al[40] (2006) Cross-sectional Level: V	MST vs PGSGA; cancer patients receiving chemotherapy	50	MST had 100% sensitivity and 92% specificity in predicting PGSGA category
van Bokhorst-De Van Der Schueren et al[32] (1997) Cross-sectional Level: III	Standardized nutrition assessment; advanced head and neck cancer patients	64	Weight loss of >10% in the previous 6 months associated with increased risk of major post-operative complications
Unsal et al[31] (2006) Cross-sectional Level: V	SGA pre- and post-XRT; cancer patients	207	Incidence of malnutrition increased following XRT but generally resolved by 6 months post-XRT

Intervento nutrizionale: albero decisionale



* situazioni a rischio: chemioterapia pesante, radioterapia addomino-pelvica o cervicale, chirurgia maggiore, trapianto di midollo

Nitemberg G. Crit Rev Oncol Hematol 2000

Importance of early nutritional screening in patients with gastric cancer

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British Journal of Nutrition (2011), **106**, 1773–1778

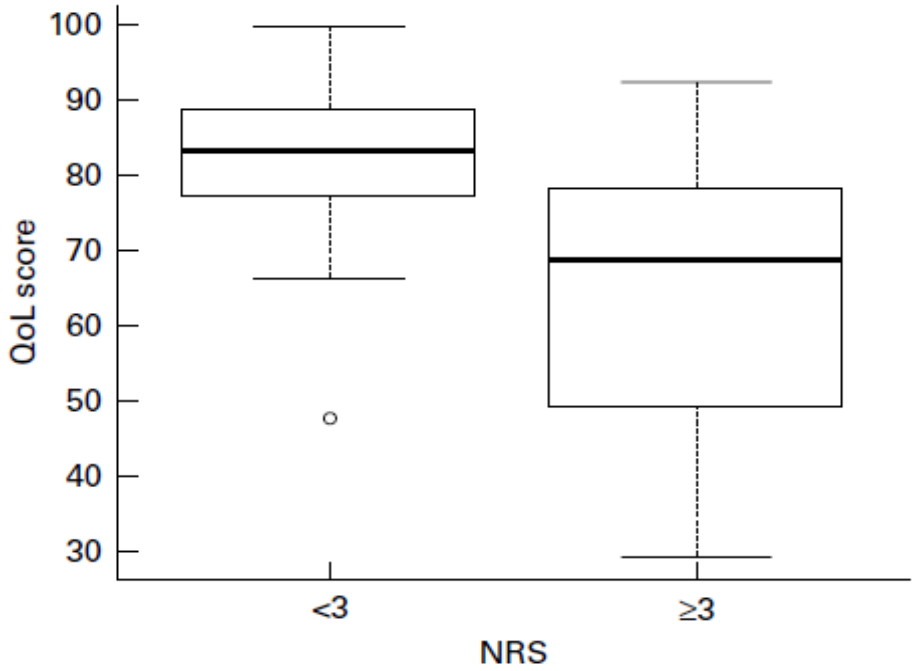

Fig. 1. Quality of life (QoL) score *v.* nutritional risk score (NRS). Each 'box-plot' shows some descriptive statistics of QoL score, i.e. (from bottom to top line): 1st quartile, median (bold line), 3rd quartile and maximum value. The circle represents one extreme value. Patients with NRS ≥ 3 (twenty-nine patients) presented lower QoL score values as compared with patients with NRS < 3 (fifty-eight patients).

The nutritional risk in oncology: a study of 1,453 cancer outpatients

Federico Bozzetti · Luigi Mariani · Salvatore Lo Vullo · The SCRINIO Working Group · Maria Luisa Amerio · Roberto Biffi · Riccardo Caccialanza · Giovanni Capuano · Isabel Correja · Luca Cozzaglio · Angelo Di Leo · Leonardo Di Cosmo · Concetta Finocchiaro · Cecilia Gavazzi · Antonello Giannoni · Patrizia Magnanini · Giovanni Mantovani · Manuela Pellegrini · Giuseppe M. Rovera · Lidia Rovera · Giancarlo Sandri · Marco Tinivella · Enrico Vigevani

Factor	No. of pts.	Mean	*P*	Score ≥3	
				%	*P*
Tumour site					
Oral cavity	116	1.5		28.5	
Oesophagus	80	3.1		62.5	
Stomach	206	2.3		43.7	
Pancreas	90	2.6	<0.0001[a]	54.3	<0.0001[a]
Small bowel	33	1.1	<0.0001[b]	6.1	0.0002[b]
Colon–rectum	518	1.5		24.3	
Lung	217	1.7		28.1	
Upper respiratory airways	84	1.4		28.6	
Other	107	1.4		25.2	
Tumour stage					
0	10	0.6		10.0	
I	51	1.4	<0.0001[a]	19.6	<0.0001[a]
II	150	1.5	0.0308[b]	23.3	0.1333[b]
III	298	1.8		33.2	
IV	578	1.7		29.8	
ECOG PS					
0	514	0.9		10.1	
1	426	2.0	<0.0001[a]	35.5	<0.0001[a]
2	185	3.3	<0.0001[b]	79.5	<0.0001[b]
3	47	4.0		91.5	
4	4	5.0		100.0	

Table 2 Mean NRS score and percentage of patients with nutritional risk (NRS score ≥3), according to main patients' characteristics

Support Care Cancer 2012

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Table III. Summary of recommendations of nutritional support in gastrointestinal oncological patients.

Item	Action	Enteral nutrition	Parenteral nutrition
Nutritional assessment	• Assess nutritional status at diagnosis (see Table II) and periodically during treatment		
Nutritional support	• Provide nutritional support if malnourished (body weight loss >10% or SGA grade C) or it is anticipated unable to eat in the 2 weeks before intervention. • Use pre-operative (5-7d) immuno-nutrition (arginine, w-3 fatty acids, nucleotides) in all subjects undergoing major abdominal surgery, independently of their nutritional status • Total energy intake is assumed to be similar to that in non-cancer patients (25-30 kcak/kg/d in ambulatory patients; 20-25 in bedridden)	• Prefer oral feeding • Consider dietary advice/ counseling during radio- and chemo-therapy • Use tube feeding if local conditions prevent oral feeding • In incurable patients minimum energy intake and water supply should be considered	• Only in patients with severe cachexia who do not tolerate enteral feeding • Only in patients with severe cachexia who do not tolerate enteral feeding • Whenever prolonged periods of inadequate oral or enteral nutrient supply are expected • High lipid supply (up to 50% of total energy) may be beneficial in subjects with severe cachexia • Consider ethical problems in incurable patients

Derived from[42-46], with simplifications.

ESPEN Guidelines 2006 – ESPEN Guidelines 2009

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Table IV. Specific recommendations nutritional advice in gastrointestinal oncological patients.

Symptom	Action
Dysphagia	• Eat soft food • Consume small frequent meals • Chew foods thoroughly • Sip liquid slowly with meals • Blenderize puree foods
Gastro-esophageal reflux	• Sit upright while eating • Avoid caffeine, alcohol, tobacco, chocolate and peppermint • Use PPI, H2-blockers and antacids
Early satiety	• Eat small, frequent meals • Avoid carbonated beverages • Limit intake of high fat foods
Dumping syndrome	• Gradually increase meal size and liberalize diet • Alternate solids and liquids • Limit simple carbohydrates
Gastric stasis	• Incorporated semi solid foods • Limit intake of high fat foods • Use medication that stimulate peristalsis and gastric emptying
Diarrhea	• Increase fluid intake • Increase foods containing soluble fiber • Use antidiarrheal medication
Nausea and vomiting	• Consume foods without odor • Avoid hot and cold foods • Limit physical activity before meals • Avoid lying down after meals • Increase fluid intake • Use antiemetic medications

ESPEN Guidelines 2006 – ESPEN Guidelines 2009

aspen
Clinical Guidelines

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Guideline Recommendations	Grade
A. Nutrition Support Therapy During Anticancer Treatment	
1. Patients with cancer are nutritionally-at-risk and should undergo nutrition screening to identify those who require formal nutrition assessment with development of a nutrition care plan.	D
2. Nutrition support therapy should not be used *routinely* in patients undergoing major cancer operations.	A
3. Perioperative nutrition support therapy may be beneficial in moderately or severely malnourished patients if administered for 7-14 days preoperatively, but the potential benefits of nutrition support must be weighed against the potential risks of the nutrition support therapy itself and of delaying the operation.	A
4. Nutrition support therapy should not be used *routinely* as an adjunct to chemotherapy.	B
5. Nutrition support therapy should not be used *routinely* in patients undergoing head and neck, abdominal, or pelvic irradiation.	B
6. Nutrition support therapy is appropriate in patients receiving active anticancer treatment who are malnourished and who are anticipated to be unable to ingest and/or absorb adequate nutrients for a prolonged period of time (see Guideline 6 Rationale for discussion of "prolonged period of time").	B
7. The palliative use of nutrition support therapy in terminally ill cancer patients is rarely indicated.	B
8. ω-3 Fatty acid supplementation may help stabilize weight in cancer patients on oral diets experiencing progressive, unintentional weight loss.	B
9. Patients should not use therapeutic diets to treat cancer.	E
10. Immune-enhancing enteral formulas containing mixtures of arginine, nucleic acids, and essential fatty acids may be beneficial in malnourished patients undergoing major cancer operations.	A