This autumn 2011 issue of Clinical Nutrition News reports innovative ideas in the areas of nutrition advocacy, professional education, basic and clinical nutrition science, and clinical practice from the 33rd ESPEN Congress on Clinical Nutrition & Metabolism, a meeting held 3-6 September, 2011 in Gothenburg, Sweden. Hot topics included: new ways to screen for malnutrition in older people; recommendations for recognizing and treating cachexia in people with chronic illnesses such as cancer and kidney disease; and discovery of barriers to change in nutritional practices. This ESPEN Congress hosted more than 3,000 participants from 81 countries.

At ESPEN 2011, Abbott supported nutrition education in multiple ways. The Abbott Nutrition Night symposium featured Profs Arthur Van Zanten (The Netherlands) and Jens Kondrup (Denmark), who discussed nutritional care for critically ill patients along the recovery pathway from intensive care unit (ICU) to hospital ward to home discharge. Abbott Nutrition organized 7 meet-the-nutrition-expert discussions on the health and financial benefits of nutritional care in community and in hospital. The Abbott Nutrition Health Institute sponsored a symposium on identification and management of sarcopenia, which featured Profs Maurits Vandewoude (Belgium) and Stéphane Schneider (France).
Nutrition Awareness and Advocacy

What’s new from the nutritionDay program?

The worldwide nutritionDay survey is designed to take a one-day snapshot of malnutrition in hospitals around the world. Project Director Michael Hiesmayr (Austria) provided a 2011 update. Since its start in 2006, nutritionDay has registered 105,095 patients in 30 countries; the survey has recently been extended beyond hospitals to include intensive care units (ICUs) and nursing homes.

Recent nutritionDay results reflect how nursing home residents with swallowing difficulties (dysphagia) and cognitive impairment face serious nutritional and health consequences. Surveys of 8,000 nursing home residents in 9 European countries revealed that malnutrition was 3- to 5-times more prevalent in residents with dysphagia and cognitive impairment than in those without. For outcomes, six-month mortality was nearly twice as high among those with dysphagia than those without.

Key barriers to changing nutritional practices

In a session titled Implementation of Nutritional Routines, nurse educators Kate Gerrish and Sarah Laker from the Sheffield Teaching Hospital and the National Health Service Foundation Trust (UK) identified challenges to implementing new practices in hospitals. They reported two barriers that are particularly problematic. First, nurses are already busy, and while nutritional care is critical, there are many other ward issues. Second, changing established ways of thinking and working among clinicians is difficult.

So how did this UK team overcome these barriers? Here are some valuable lessons learned about implementing programs for improved nutritional care:

- To drive nutritional practice changes, all caregivers must be aware of the problem and prepared to address it.
- Nutritional care involves many people across the hospital system, so a multi-disciplinary team is needed—e.g., nurses, dietitians, catering staff, and physicians.
- Nutrition teams need strong leadership from within, “champions” who will model and advance new practices.
- Key educational messages must be repeated and reinforced. Resource materials and tools help implement changes in nutritional practice, but new practices must be reinforced with audits and follow-up training.
- Monitor results of practice changes in order to measure successes and shortfalls. Share findings with the entire team—early and often.

Given these challenges, what is the evidence that improvements in nutritional practice are possible? Nutrition nurse specialist Mette Holst (Denmark) and colleagues at a university hospital had remarkable success in implementing good nutritional practice over the course of just one year. The team was able to improve nutritional screening from 56% to 77% ($P < 0.001$); increase the proportion of patients who received nutritional plans from 21% to 56% ($P < 0.0001$); and increase monitoring of food intake from 29% to 58% ($P < 0.0001$). As a result, more patients at nutritional risk improved their energy intake: one year after implementation of a nutritional practice plan, significantly more patients met their energy intake threshold (post- vs. pre-implementation 68% vs 52%, $P < 0.007$).
Sarcopenia versus cachexia: what is the difference?

The concepts of sarcopenia and cachexia were front and center at ESPEN 2011. In an educational program, Profs Tommy Cederholm (Sweden) and Maurizio Muscaritoli (Italy) compared these conditions. Although each is defined distinctly, sarcopenia and cachexia often overlap, especially in older people.3

Sarcopenia (Greek sarx or meat + penia or loss) represents loss of muscle mass and function, which is most frequently related to aging.4,5 Cachexia (Greek cac or bad + hexis or condition) is a complex metabolic syndrome characterized by loss of muscle mass (with or without loss of fat) due to underlying illness.6,7 Cachexia—a condition coupled with inflammation, anorexia, and breakdown of muscle proteins—is recognized as severe wasting in diseases such as cancer, renal disease, chronic heart disease, chronic obstructive pulmonary disease, and sepsis.5-9 Thus, many sarcopenic individuals have no cachexia, but most cachectic individuals have sarcopenia.

In clinical practice, sarcopenia is diagnosed as low muscle mass in concert with low function (measured as low strength or low physical performance).5 Bioimpedance analysis (BIA) is a useful tool to detect low lean body mass. Gait speed is now a consensus choice for measurement of function; the cutoff of 0.8 m/sec appears reasonable because it predicts mortality.10 Low handgrip strength is a convenient alternative way to identify low muscle function.5

A principal difference between disease-related cachexia and age-related sarcopenia is that inflammatory cytokines are present in cachexia; serum C-reactive protein values higher than 5 mg/L suggest an inflammatory state.6 Newly published clinical criteria for diagnosis of cachexia in cancer patients use weight loss and the presence of sarcopenia:8

- Weight loss > 5% over 6 months (but not simple starvation); or
- Body mass index (BMI) < 20 kg/m² and any degree of weight loss > 2%; or
- Appendicular skeletal muscle index (SMI) consistent with sarcopenia (males < 7.26 kg/m², females < 5.45 kg/m²) with any degree of weight loss > 2%.

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**Definitions**

Sarcopenia (Greek sarx or meat + penia or loss): loss of muscle mass and function, most frequently related to aging.

Cachexia (Greek cac or bad + hexis or condition): a complex metabolic syndrome characterized by loss of muscle mass (with or without loss of fat) due to underlying illness.
Treating anorexia and wasting in chronic kidney disease

“One of every 2 to 3 dialysis patients experiences anorexia,” noted Prof Juan Jesus Carrero (Sweden) as he began a ESPEN 2011 session on chronic kidney disease (CKD) and nutrition. In patients with CKD, distressing consequences of anorexia with protein-wasting include decreased quality of life, increased hospitalization rates, and higher risk of death.

Anorexia and protein-energy wasting, evidenced by loss of body protein mass and decreased fuel reserves, have many underlying causes in CKD—including high levels of uremic toxins, perturbations of appetite-controlling hormones, and elevated levels of inflammatory cytokines. Depression, taste abnormalities, and poor dental health also contribute indirectly.

Lacking formal guidelines for treatment of people with anorexia and CKD wasting, Prof Carrero offered these treatment strategies: (1) identify and treat underlying causes such as depression and dental problems, (2) support food intake, (3) promote physical activity, and (4) consider appetite stimulants.

Nutritional status plays a huge role in determining health outcomes for a person with CKD. In dialysis populations, mortality is predicted by markers of protein-energy wasting (hypoalbuminemia, low serum cholesterol levels, low body mass index, and reduced dietary protein intake). While the association of wasting and mortality is commonly reported in maintenance dialysis patients, recent studies extend this finding to include patients in pre-dialysis stages of CKD as well. Nutritional intake can be improved by use of oral nutritional supplements (formulations specific for patients with CKD are available) and by use of intradialytic parenteral feeding.

Physical exercise is surprisingly good therapy for CKD patients on dialysis. Based on results from a large multinational study, hemodialysis patients who exercised regularly had increased appetite, less pain, were less likely to experience depression, and reported higher health-related quality of life.

Since the prevalence of wasting is higher in people with CKD than in older people or those with AIDS, there is a great need to look for novel treatment strategies. Dr T Alp Ikizler (USA) reviewed new and upcoming agents that may help overcome anorexia and protein-energy wasting in CKD patients. By category and examples, these include appetite stimulants (megestrol acetate, ghrelin), specific and non-specific anti-inflammatory agents (ani-TNF, fish oil), metabolic modulators (insulin sensitizer such as pioglitazone), anabolic hormones (human growth hormone, testosterone, nandrolone), and a myostatin inhibitor. Continued research and clinical testing are necessary to determine which of these new approaches are safe and effective.

Exercise therapy and CKD

Physical exercise is surprisingly good therapy for CKD patients on dialysis. Hemodialysis patients who exercised regularly had increased appetite, less pain, were less likely to experience depression, and reported higher health-related quality of life.
Focus on patient-centered outcomes in cancer

As presenter of the honorary Arvid Wretlind lecture at ESPEN 2011, Prof Kenneth Fearon acknowledged recent remarkable advances in understanding cancer at the molecular level, yet he underscored the continuing importance of maintaining a focus on patient-centered outcomes.

Impact on daily activities
“In terms of ability to perform daily activities, a person with cancer is often more severely impaired than an individual with osteoarthritis or one recovering from surgery.”
Prof Kenneth Fearon (UK)

Fearon noted, “In terms of ability to perform daily activities, a person with cancer is often more severely impaired than an individual with osteoarthritis or a patient recovering from surgery.” Such impairment lowers quality of life.

In patients with cancer, weight loss is usually the first symptom noticed; the underlying cause of weight loss is cancer cachexia. With cachexia, inflammation related to a tumor itself or to the body’s response to a tumor leads to diminished appetite, metabolic changes, loss of muscle and fat tissues, and impaired capacity for physical activities. Together these conditions are called cancer cachexia, which has been formally defined and described by an international consensus group. Cachexia syndrome can develop progressively through various stages—from precachexia to cachexia to refractory cachexia and ultimately to death. The severity of cancer cachexia is classified according to degree of depletion of energy stores and body protein combined with degree of ongoing weight loss.

Treatment of cancer is multi-modal, and nutrition plays an essential role. In particular, interventions that help increase muscle (lean body mass) and support physical activity represent a new treatment strategy. Fearon advised that the goals of treatment should be adjusted according to the patient’s stage of cancer cachexia. For example, nutritional and other interventions in the precachexia stage can be used to preserve quality of life as long as possible, while few or no interventions are appropriate in the refractory cachexia stage.

Impact on daily activities

“In terms of ability to perform daily activities, a person with cancer is often more severely impaired than an individual with osteoarthritis or one recovering from surgery.”
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Treatment goals
“The goals of treatment should be adjusted according to the patient’s stage of cancer cachexia. Nutritional and other interventions in the precachexia stage can be used to preserve quality of life as long as possible, while few or no interventions are appropriate in the refractory cachexia stage.”
Prof Kenneth Fearon (UK)
New nutritional screening tools

In The Netherlands, nutrition is a serious matter for policy makers, hospital administrators, clinicians, and patients alike; nutritional screening is mandatory in all Dutch hospitals, residential care, and home care settings. In fact, screening rates are published on the internet to help patients decide where to seek care.

With the impetus of mandatory screening, experts in the Netherlands have developed and validated a series of easy-to-use screening tools called Short Nutritional Assessment Questionnaires: SNAQ for use in the hospital,¹⁹ SNAQRC for residential care,²⁰ and SNAQ65+ for home-living elderly.²¹,²² SNAQ65+ is the first-ever tool developed specifically for use in the community. This community screening tool relies on 4 patient determinations: amount of weight loss over the prior 6 months, mid-upper arm circumference, appetite in the last week, and ability or inability to climb 15 stairs. Each of the screening tools includes only simple questions and measures, so it can be completed in just a few minutes.

For Dutch hospitals, scores from SNAQ (www.fightmalnutrition.eu) or from the alternative Malnutrition Universal Screening Tool (MUST; www.bapen.org.uk/must_tool.html) are used for reimbursements. Of the 100 Dutch hospitals, 80% use SNAQ and 20% use MUST.

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**SNAQ**

<table>
<thead>
<tr>
<th>Short Nutritional Assessment Questionnaire</th>
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<tbody>
<tr>
<td>Did you lose weight unintentionally?</td>
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<tr>
<td>More than 6 kg in the last 6 months</td>
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<tr>
<td>More than 3 kg in the last month</td>
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<tr>
<td>Did you experience a decreased appetite over the last month?</td>
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<tr>
<td>Did you use supplemental drinks or tube feeding over the last month?</td>
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<tr>
<td></td>
</tr>
<tr>
<td>no intervention</td>
</tr>
<tr>
<td>moderately malnourished; nutritional intervention</td>
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<tr>
<td>severely malnourished; nutritional intervention and dietitian</td>
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</tbody>
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Payment for nutrition treatment

“SNAQ or MUST scores are used for reimbursement of nutrition treatment in the Netherlands—a policy that is important to fighting malnutrition.”

_Hinke Kruizenga, PhD (Netherlands)_
Assessment of nutritional status: what are we measuring?

Clinicians usually assess nutritional status by reviewing a patient’s appetite and food intake, BMI, and recent weight loss. However, many physicians also select some objective laboratory measures to monitor nutritional status. At ESPEN 2011, Prof L Ellegård (Sweden) reviewed serum proteins as biochemical indices that are decreased in states of malnutrition.\(^{23}\)

<table>
<thead>
<tr>
<th>Biochemical indices used to assess nutritional status</th>
<th>Normal range</th>
<th>Half life</th>
<th>Relative cost</th>
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<tbody>
<tr>
<td>Serum indices</td>
<td></td>
<td></td>
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<tr>
<td>Albumin</td>
<td>35-50 g/L</td>
<td>20 days</td>
<td>1</td>
</tr>
<tr>
<td>Transferrin</td>
<td>2-4 g/L</td>
<td>8 days</td>
<td>1</td>
</tr>
<tr>
<td>Orosomucoid</td>
<td>0.55-1.05 g/L</td>
<td>5 days</td>
<td>2</td>
</tr>
<tr>
<td>Prealbumin</td>
<td>~0.3 g/L</td>
<td>1-2 days</td>
<td>3</td>
</tr>
<tr>
<td>Retinol binding protein</td>
<td>~60 mg/L</td>
<td>½ day</td>
<td>2</td>
</tr>
</tbody>
</table>

He reported that factors such as half-life and cost determine whether each index is predictive and practical. For example, serum albumin has a half-life of about 20 days, so it is a good long-term prognostic indicator but not a good measure of change in worsening disease or response to treatment; this test is relatively inexpensive. On the other hand, the half life of prealbumin is 1-2 days, so it is a better indicator of short-term changes, but this test is more expensive to perform.

Assessment predicts cancer prognosis

In a poster presentation at ESPEN 2011, Arends and colleagues (Germany) evaluated ways to predict prognosis in cancer patients using serum values for albumin and the inflammatory marker C-reactive protein (CRP; half life = 0.8 days) along with body composition by bioimpedance analysis (BIA).\(^{24}\) They found that the chances of surviving 100 days are very low—just 1 in 20—for patients with serum albumin levels < 30 g/L, CRP values > 50 mg/L, and BIA phase angle < 3.5°. With further association of various cutoff points and outcomes, the researchers anticipate using such information to help make complex decisions about artificial nutrition. Thus, in patients with advanced tumors, biochemical indices and other measures can help assess nutritional status, predict outcomes, and set goals of nutritional therapy.

Nutritional measures can predict cancer outcomes

For patients with serum albumin levels < 30 g/L, CRP values > 50 mg/L, and BIA phase angle < 3.5°, the chances of surviving 100 days are very low—just 1 in 20.

Prof Jann Arends (Germany)
Sarcopenia: New Paradigms in Identification and Management was the topic featured by the Abbott Nutrition Health Institute satellite symposium. Keynote speakers were European geriatricians Prof Maurits Vandewoude (Belgium) and Prof Stéphane Schneider (France).

Sarcopenia, or loss of muscle mass and function, is prevalent in older populations, and takes high personal and health tolls. Specifically, sarcopenia:

- increases mobility disorders and risk of falls and fractures
- impairs ability to perform activities of daily living
- causes disabilities, loss of independence
- increases risk of death

**Sarcopenia causes**

Many factors can contribute to development of sarcopenia, including age-related hormonal and physiological changes, low physical activity, disease-related metabolic alterations, and malnutrition. Eventually, loss of muscle function falls below a threshold needed for physical function, and a person can no longer move around to perform usual daily activities.

According to Prof Vandewoude, sarcopenia is considered primary when no causes other than aging can be found; sarcopenia is secondary when other factors are present. For example, sarcopenia of disuse can develop with bedrest due to injury or illness or to a sedentary lifestyle. Disease-related sarcopenia is often associated with inflammation, as in malignancy and conditions of advanced organ failure (heart, lung, liver, or kidney). Nutritional inadequacy also plays a part, and is a problem in people who have difficulty purchasing or preparing food, in those with malabsorption and other gastrointestinal disorders, or in those taking medications that cause anorexia. When any or all of these conditions occur simultaneously, sarcopenia is worsened toward or below the threshold of immobility.

**Sarcopenia diagnosis**

Prof Vandewoude reviewed how the diagnosis of sarcopenia is based on documentation of evidence for low muscle mass along with low muscle function (detected as either low muscle strength or low physical performance). Given the serious consequences of sarcopenia, he recommended screening all older patients using measurements that can be easily performed in the office and other clinical settings. He discussed bioimpedance analysis (BIA) for muscle mass, handgrip dynamometry for strength, and usual gait speed for performance, as well as many other tools.

Sarcopenia has been subdivided into three stages: presarcopenia, sarcopenia, and severe sarcopenia. Prof Vandewoude advised, “Recognizing these different stages may help select treatments and set appropriate recovery goals.”

<table>
<thead>
<tr>
<th>Stages of sarcopenia</th>
<th>Muscle mass</th>
<th>Muscle strength</th>
<th>Performance</th>
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<tbody>
<tr>
<td>Presarcopenia</td>
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<tr>
<td>Sarcopenia</td>
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<td>Or</td>
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<tr>
<td>Severe sarcopenia</td>
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**Stages of sarcopenia in clinical practice**

Recognizing the different stages of sarcopenia may help in the selection of treatments and setting appropriate recovery goals.

*Prof Vandewoude (Belgium)*

Sarcopenia is variously attributed to age, low physical activity, disease, or malnutrition—or a combination of these factors.
Sarcopenia treatment

For sarcopenia treatment, the best strategy is early identification of risk and early treatment to help prevent progression. However, there are times when sarcopenia is diagnosed at more advanced stages. Prof Stéphane Schneider reviewed recent studies to evaluate a wide range of treatments with potential to help reverse sarcopenia.

Treatment of sarcopenia is all about achieving optimal anabolic responses with adequate intake of dietary protein; Prof Schneider suggested that older adults may require levels higher than the standard Recommended Dietary Allowances (RDA) for all adults. Treatment can also be improved by intake of specific anabolic amino acids or related bioactive metabolites. Vitamin D is newly recognized for its role in building muscle as well as bone tissue. Along with proper nutrition, exercise—especially resistance training—plays a vital role in building and maintaining muscle strength.

The table below summarizes strategies proposed by Prof Schneider to increase lean body mass and physical function.

<table>
<thead>
<tr>
<th>Proposed strategy to prevent or reverse sarcopenia</th>
<th>Comments</th>
</tr>
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<tbody>
<tr>
<td>Increase protein intake</td>
<td>The current RDA is 0.8 g protein/kg body weight/day for all adults is probably inadequate to maintain muscle or lean body mass. An age-specific revision to 1.0 g/kg/day may be necessary for older adults.</td>
</tr>
<tr>
<td>Increase amino acid bioavailability</td>
<td>It is not yet clear whether anabolic benefits are greatest when protein intake is evenly distributed as 3-4 meals(^{30}) or taken mostly as a single pulse;(^{31}) further testing is needed.</td>
</tr>
<tr>
<td>Add specific substrates to diet</td>
<td>HMB, leucine, and citrulline all appear to increase protein synthesis through anabolic actions via the cellular mTOR pathway;(^{32}) head-to-head comparison or combination therapy has not been tried.</td>
</tr>
<tr>
<td>Vitamin D supplementation</td>
<td>Vitamin D intake clearly improves muscle strength and performance; optimal dose for older people is yet to be determined.</td>
</tr>
<tr>
<td>Resistance training</td>
<td>Muscle-building exercise is considered a key strategy for management of sarcopenia.(^{33})</td>
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</tbody>
</table>

Sarcopenia and protein

Treatment of sarcopenia is all about achieving optimal anabolic responses with adequate intake of dietary protein.
Abbott Nutrition Night: Nutrition for ICU Patients

At Abbott Nutrition Night (ANN) of ESPEN 2011, Dr Refaat Hegazi (USA, Abbott Medical Director of Adult Nutrition Research and Development) focused on critical illness as a catabolic disease. For example, a patient who has experienced a 15% weight loss may actually lose up to 35% of peripheral skeletal muscle mass; this lost muscle is not likely to be restored even one year after hospital discharge. Nutrition during and after an ICU episode is important to recovery. “ICU nutrition has evolved from its early role as nutritional support to its current role as nutritional therapy,” advised Dr Hegazi.

Prof Arthur Van Zanten (Netherlands) opened his presentation with the challenge, “We are doing a bad job of feeding ICU patients; we can do better.” For patients in the ICU, two main goals should be (1) to avert an energy deficit, and (2) to prevent or minimize loss of lean body mass. Prof Van Zanten showed that an ICU patient receiving about 1,000 kcal per day will build an energy deficit as high at 12,000 kcal over the course of a week; such shortfall is associated with increased risk for complications, mainly infections. According to Prof Van Zanten, for every 1,000 kcal per day fed to an ICU patient, the risk of mortality can be lowered by 25-50%, particularly in individuals who are severely under- or overweight.

Prof Van Zanten (Netherlands)

ICU feeding guidelines emphasize evidence-based strategies that yield improved outcomes. Prof Van Zanten summarized current recommendations:

- Since the catabolic clock is ticking, start feeding as early as possible—preferably within 24 to 48 hours of admission.
- Do not wait for bowel sounds.
- Feed all hemodynamically stable patients, including those on pressors.
- Also feed obese patients.
- Do not hold enteral nutrition because of gastric residual volumes (GRV) over 350 mL or even 500 mL if other signs of feeding intolerance are absent.
- Consider using a prokinetic drug, e.g. metoclopramide, to improve feeding tolerance.
- Use small bowel feeding if upper GI feeding is not tolerated.
- Consider using specialized enteral formulas containing certain nutrients for specific subgroups of patients. For example, formulas containing the omega-3 eicosapentaenoic acid, omega-6 gamma linolenic acid, and antioxidants moderate uncontrolled inflammation, while formulas with arginine and glutamine lessen immune suppression.

Prof Jens Kondrup (Denmark) next reviewed nutritional therapy used after discharge to home. The statistics on recovery from critical illness such as acute respiratory distress syndrome (ARDS) are unsettling. After one year, only half of ARDS survivors had returned to work, and more than one third had not yet achieved normal 6-minute walking distances. After 5 years, exercise limitation, psychological sequelae, decreased physical quality of life, and increased costs and use of health care services are long-term legacies of severe lung injury.

In order to better such outcomes, Prof Kondrup urged continued and regular monitoring of nutritional status after discharge. He advised intake of 30-35 kcal/kg body weight/day (including 1.2-1.5 g protein/kg/day) for gain of lean body mass. In most cases, such goals can be met by use of oral nutritional supplements. However, enteral tube-feeding is required in cases of severe disability, and parenteral feeding is needed in rare patients who have lost GI function. For people with poor nutritional status, Kondrup advised considering oral and enteral feeding products formulated to drive anabolism, e.g., energy-dense, high-protein formulas that contain other anabolic ingredients.

Together, the speakers at ANN for ESPEN 2011 delivered a clear take-home message: careful attention to nutrition is important over the entire course of recovery—from ICU to home discharge.
Is there a genetic predisposition to cancer cachexia?

Cancer cachexia develops along a spectrum of losing weight and lean body mass. Since muscle mass in health is 90% determined by genetics, Prof Kenneth Fearon asked the next logical question, “Is there a genetic predisposition to cancer cachexia?”

Two principal strategies are available to identify genes involved in disease—forward and reverse genetics. Forward genetics searches the genome for differences in the presence or absence of a condition. Reverse genetics examines changes in expression of genes that are logical candidates. Much of the work done on cancer cachexia to date was based on the reverse genetic approach of studying candidate genes and their component nucleotides.

So far, a relatively small number of studies have linked single-nucleotide changes in pro-inflammatory cytokine genes to the development of cachexia and accelerated demise in advanced cancer. In particular, an interleukin-6 (IL-6) change was associated with systemic inflammation and adverse outcome in esophageal and pancreatic cancers. Other candidate genes encode proteins with anti-inflammatory functions or regulate energy production and homeostasis, production and maintenance of adipose and muscle tissue, and appetite control. Altogether, a total of 184 polymorphisms with functional or clinical relevance to cancer cachexia have been identified in 92 candidate genes.

With a better understanding of genes that predispose to cachexia, it may someday be possible to identify predictive or early markers, which could be used to help guide individual treatment choices.

**What else is new in nutrition science?**

At ESPEN 2011, we heard news about cytokines, hormones, and food substances with potential roles in treatment of sarcopenia, diabetes, and cancer. Watch as

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<table>
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<tr>
<th><strong>New in nutrition science</strong></th>
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<tbody>
<tr>
<td>Defensins</td>
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<tr>
<td>HMB</td>
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<tr>
<td><em>Salacia oblonga</em></td>
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<td>Ghrelin</td>
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these new science concepts move from bench to bedside.

References


