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ESPEN Guideline

ESPEN guidelines on nutrition in dementia

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SUMMARY

Background: Older people suffering from dementia are at increased risk of malnutrition due to various nutritional problems, and the question arises which interventions are effective in maintaining adequate nutritional intake and nutritional status in the course of the disease. It is of further interest whether supplementation of energy and/or specific nutrients is able to prevent further cognitive decline or even correct cognitive impairment, and in which situations artificial nutritional support is justified.
Objective: It is the purpose of these guidelines to cover these issues with evidence-based recommendations.
Methods: The guidelines were developed by an international multidisciplinary working group in accordance with officially accepted standards. The GRADE system was used for assigning strength of evidence. Recommendations were discussed, submitted to Delphi rounds and accepted in an online survey among ESPEN members.
Results: 26 recommendations for nutritional care of older persons with dementia are given. In every person with dementia, screening for malnutrition and close monitoring of body weight are recommended. In all stages of the disease, oral nutrition may be supported by provision of adequate, attractive food in a pleasant environment, by adequate nursing support and elimination of potential causes of malnutrition. Supplementation of single nutrients is not recommended unless there is a sign of deficiency. Oral nutritional supplements are recommended to improve nutritional status but not to correct cognitive impairment or prevent cognitive decline. Artificial nutrition is suggested in patients with mild or moderate dementia for a limited period of time to overcome a crisis situation with markedly insufficient oral intake, if low nutritional intake is predominantly caused by a potentially reversible condition, but not in patients with severe dementia or in the terminal phase of life.
Conclusion: Nutritional care and support should be an integral part of dementia management. In all stages of the disease, the decision for or against nutritional interventions should be made on an individual basis after carefully balancing expected benefit and potential burden, taking the (assumed) patient will and general prognosis into account.

Abbreviations: AD, Alzheimer's disease; APOE, apolipoprotein E-e4 allele; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; MCI, mild cognitive impairment; MNA, mini nutritional assessment; MNA-SF, mini nutritional assessment short form; MMSE, mini mental state examination; RCT, randomized controlled trial.

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1. Development of guidelines on nutrition in dementia

The European Society for Clinical Nutrition and Metabolism (ESPEN) launched a process of developing guidelines on nutrition care for patients with dementia. The group included physicians, nutritionists and dietitians with a background in geriatrics, nutrition and/or ethics, all experienced in treatment and nutritional therapy of persons with dementia, as well as the guidelines coordinator (SMS); all are authors of this guideline document.

The experts followed the GRADE method, which was based on determinations of *grade of evidence* and *strength of recommendation*; the methodology is described elsewhere [1]. A two-day live meeting was organized in Biedenkopf, Germany, in April of 2014, and three phone conferences were held.

Descriptive findings that did not lead to specific recommendations are grouped in a first part (general considerations), whereas all questions that led to comparisons of interventions and to recommendations are grouped in a second part (recommendations).

A systematic literature search was conducted in PubMed and the Cochrane Library. The *grade of evidence* (GOE) was determined by a number of factors, starting with the number and type of research studies [2]. Grading from *High* to *Very Low* was used to rate the quality of the underlying evidence and the level of certainty for effect (Table 1) [3]. Highest quality evidence resulted from consistent results or meta-analysis of multiple randomized controlled trials, with the next highest level defined by at least one well-designed randomized controlled trial. Moderate and low-level evidence came from controlled trials that were not randomized, from cohort- or case-controlled studies, or from multiple time series trials. Very low-level evidence was from expert clinical experience or from descriptive studies. The grade was then decreased if there were limitations to study quality, inconsistencies in findings, imprecise or sparse data, or high likelihood of reporting bias. The grade was increased if there was high consistency of findings or strong evidence of association (Table 1).

The *strength of recommendation* was based on a consensus discussion, which included expression and deliberation of expert opinions, risk-benefit ratio of recommendation, costs, and a review of supportive evidence, followed by Delphi rounds and votes until agreement was reached (Table 2).

Last, a list of all statements was sent to all 2611 ESPEN members with an e-mail address on file to ask for approval/disapproval of every statement, and in the latter case to provide justification. 86 ESPEN members completed the survey, with approval ratings ranging from 70% to 93%. Comments based on the literature were taken into account in the final version of the manuscript.

Table 1
Grades of evidence [3].

Level	Definitions of evidence
High	Further research is unlikely to change our confidence in the estimate of effect.
Moderate	Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate.
Low	Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate.
Very low	Any estimate of effect is very uncertain.

Table 2
Strength of recommendation.

Strength of recommendation	
Strong	We recommend/do not recommend
Weak	We suggest/do not suggest

2. General considerations

2.1. Definition of dementia

Dementia is on the rise in our aging societies, not only in Europe and North America but worldwide. Dementia becomes more and more common, but it is not normal healthy aging and it is not benign. It is a malignant and devastating condition leading invariably to dependence and finally death [4]. As a clinical syndrome, it is characterized by global cognitive impairment with a decline in memory and at least in one other cognitive domain, such as language, visuospatial, or executive function. It represents a decline from the previous level of cognitive functioning, and is associated with impairment in functional abilities and, in many cases, behavioral and psychiatric disturbances [5,6]. Many diseases can cause a dementia syndrome; Alzheimer's disease and cerebrovascular dementia are the two most common causes, and many cases of dementia involve both these disorders. Lewy body disorders (Parkinson's disease and dementia with Lewy bodies) and frontotemporal dementia are less common but still make up 8% of people referred to a memory clinic [5]. Although some potentially reversible conditions, such as hypothyroidism or vitamin B_{12} deficiency, are often thought to cause dementia, no more than 1.5% of cases of mild to moderate dementia are fully reversible. Age is the best-studied and strongest risk factor for dementia, which explains the increasing burden of cognitive disorders in the years to come [7]. Other risk factors for Alzheimer's disease include genetic risk factors such as having a first-degree relative with a history of Alzheimer's disease, having the apolipoprotein $\varepsilon 4$ genotype or suffering from the Down syndrome. Cardiovascular risk factors such as hypertension are associated with an increased risk for both Alzheimer's disease and vascular dementia. Also lifestyle factors such as low educational level or head trauma may play an important role [7].

Dementia causes a high burden of suffering for patients, their families, and society [8]. For patients, it leads to increased anxiety, depression and dependency and complicates other comorbid conditions. For families, it also leads to anxiety, depression, and increased time spent caring for a loved one. The annual societal cost of dementia is huge, due to health care and related costs as well as lost wages for patients and family caregivers.

As for other chronic conditions and geriatric syndromes the processes underlying the development of a progressive cognitive disorder such as the dementia syndrome spans a far longer period than previously thought. The first changes occur in the brain long before the first memory complaint will be present. Autosomal dominant Alzheimer's disease was associated with a series of pathophysiological changes over decades in cerebral spinal fluid biochemical markers of Alzheimer's disease, brain amyloid deposition, and brain metabolism as well as progressive cognitive impairment [9]. Therefore, minor cognitive disorders, such as Mild Cognitive Impairment (MCI) and even more so major cognitive disorders such as dementia are only a later and terminal stage and clinical expression of the longstanding and progressive changes occurring in the brain.

The changes in cognition will have an impact on the functional status of the individual. The person will pass on from being independent, over becoming frail to finally being disabled and dependent [10]. Indeed, the cognitive changes will render the individual slowly more vulnerable and frail. These functional changes together with the assessment of difficulties in communication and social interaction will determine the severity of the condition. The transition from a normal asymptomatic state over mild cognitive impairment to early, mild to moderate and finally severe dementia