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Dysphagia

The Interdependency of Protein-Energy Malnutrition, Aging, and Dysphagia

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Abstract. Advancing age is increasingly associated with confounding chronic and acute ailments, predisposing elderly individuals to conditions such as malnutrition and swallowing dysfunction. This enhanced susceptibility to malnutrition and dysphagia in this aging demographic lends itself to exacerbating, disabling conditions that may result in increased morbidity and mortality in the event of an aspiration episode. Early identification of substandard nutritional status and subsequent intervention in the elderly dysphagic population may circumvent the deleterious effects of malnutrition.

Key words: Aging — Dysphagia — Immune function — Malnutrition — Muscle functioin — Deglutition — Deglutition disorders.

The swallowing act, or deglutition, is the product of an extremely complicated series of events that require an intact nervous system and adequate deglutitive musculature to initiate, facilitate, and conclude a safe swallow. Any interference with the precise and synchronized movements of the involved muscles and their associated structures may lead to the inability to swallow liquid or solid foodstuffs safely because of underlying central neurologic or isolated mechanical dysfunction, commonly called dysphagia. The elderly individual faces age and nutritionally induced physiologic changes that predisposes them to a heightened risk of developing dysphagia and as a result, nutritional disorders such as malnutrition. Furthermore, untreated dysphagia may result in protein-energy malnutrition (PEM), leading to life-threatening conditions, a result of increased risk of accompanying infections, secondary to compromise of the immune system due to the nutritional deficit [1]. It has also been noted that there are changes in T-cell-dependent immunologic responses that occur during senescence and that those more sensitive immune components are also most susceptible to nutritional insults [2]. Further, it is well known that skeletal musculature is a pivotal component in the adaptation to malnutrition or response of energy and amino acid metabolism to stressful stimuli such as infection or physical trauma [3]. However, advancing age has been linked with confounding factors altering muscle function, structure, and metabolism. It has recently been postulated that states of nutritional deficiency may also influence the deglutitive and respiratory muscles significantly to further diminish the impaired swallowing mechanism in the dysphagic individual. Therefore, one can deduce from the numerous complications the elderly dysphagic faces (predisposition to nutritional disorders and their ramifications on fatigability of muscle and immunocompetance, swallowing dysfunction and age-related physiologic deline in both musculature and immune function) that the associated morbidity and mortality would be increased. Recognizing the difficulty associated with distinguishing PEM from chronic illness plagues geriatric physicians. This review illustrates the conditions associated with PEM and dysphagia in the elderly individual to understand the interrelationships involved in modulating this demographic's health status and quality of life.

Incidence of Dysphagia in the Elderly

With the increase in life expectancy and incidence of acute and chronic ailments, a greater number of elderly

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are experiencing swallowing disorders [4]. For example, 75% of strokes occur in people older than age 65 years, and the incidence of stroke rises with age [5]. In addition, many patients having suffered major strokes often manifest dysphagia as part of their neurologic deficit [6]. Therefore, the elderly are particularly vulnerable to strokes and the resulting dysphagia [5]. Studies have shown that swallowing disorders may affect 10% of acutely hospitalized elderly [4] and 30–60% of nursing home patients [6], and general population surveys have suggested that up to 10% of individuals older than age 50 years experience troublesome dysphagia [7].

Etiology of Dysphagia in the Elderly

Age-related alterations in swallowing have been studied extensively [8–12]. This research has identified several age-associated changes, including decreased salivary flow [10], increased motor response time needed for chewing [11], impaired pharyngeal peristalsis, and upper esophageal sphincter opening [12]. However, complete physiologic understanding of age-induced swallowing impairment includes not only an analysis of motor function but also, an analysis of sensory function. Sensory discrimination decreases with increasing age in the arms, hands, feet, and face [13–15]. Recent research has sought to demonstrate that sensory discrimination thresholds in the oral cavity also increase with aging [13,16,17]. Aviv et al. [18] demonstrated a progressive diminution of sensory capacity in the laryngopharynx with increasing age, which, when accompanied with motor dysfunction commonly seen in aging, may contribute to the development of dysphagia and subsequent aspiration in the elderly. However, it has proven difficult to separate age-related motor and sensory phenomena from the effects of superimposed disease in this demographic [7]. Although previous studies have suggested that esophageal motor function deteriorates with age [8,19,20], more recent studies, which used more sophisticated recording techniques and excluded patients with confounding disease states, have demonstrated minimal or no age-related changes in esophageal motility [21–23].

Researchers currently speculate that age-related functional and sensory changes in the elderly result in a decreased functional reserve. Therefore, problems may develop more frequently when disease or generalized weakness, as a result of systemic illness or malnutrition, intervenes [7]. Superimposed pathologies such as parkinsonism may cause increasingly advanced expression of the normal aging changes [24]. This hypothesis would account for the increased incidence of swallowing dysfunction in this demographic. Thus, dysphagia in some elderly patients may be viewed at the extreme of a continuum of oral-motor changes characterizing normal aging.

Dysphagia can typically be divided into two categories: (a) abnormalities affecting the neuromuscular mechanism controlling movement of the tongue, pharynx, and upper esophageal sphincter (oropharyngeal dysphagia) and (b) disorders affecting the esophagus itself (esophageal dysphagia). Common causes and characteristics of oropharangeal and esophageal dysphagia are presented in Table 1. It is important to note that swallowing disorders in the elderly may result not only from the diseases and disorders listed in Table 1, but also from disturbances not typically associated with the younger dysphagic [7]. These problems may include cognitive dysfunction, deterioration of the muscles of mastication, osteoporosis affecting the mandible [6], atrophy of the alveolar bone if dentition is lost, and an increase in fatty and connective tissue in the tongue [10].

The Dysphagia and Malnutrition Correlation

Dysphagia is frequently viewed as a secondary or even minor disorder in the elderly compared with the primary diagnosis causing the dysphagia (stroke, surgery, acute disease/trauma). However, regardless of etiology, eating and swallowing disturbances in the elderly have been associated with a poor prognosis, with severe malnutrition as a common sequela [7]. Dysphagia should be viewed as a systemic disease associated with severe malnutrition, with a 13% risk of mortality [25]. PEM, also called protein-calorie malnutrition (PCM), may be defined as unintentional loss of greater than 10% of usual weight in less than 6 months and/or a serum albumin level of less than 3.5 g/dl [26]. There have been numerous studies detailing the association of PEM and dysphagia that delineate both the increased incidence of morbidity and mortality [27–31]. Acquired immune dysfunction, respiratory and cardiac insufficiency, decubitus ulcer formation, and impaired gastrointestinal function have all been described as consequences [27].

Aside from the difficulties in diagnosis and management frequently associated with dysphagia in the elderly, an important repercussion of the failure to swallow is starvation. Thus, dysphagic patients frequently manifest different degrees of nutritional compromise. Therefore, proper management must address not only the cause of the dysphagia, but also the consequent malnutrition [32]. Sitzmann [25] reported that, over a 1-year period, 100% of consecutive hospital admissions with a primary diagnosis of dysphagia ($n = 90$) were malnourished. In addition, more than 70% of those patients exhibited visceral protein depletion (transferrin < 200 mg/dl, albumin

Table 1. Causes and characteristics of oro- and esophageal dysphagia [6,7]

Condition	Symptoms	Causes
Oropharyngeal dysphagia	Difficulty initiating the act of swallowing, preventing food transfer from mouth to upper esophagus	Central nervous system: stroke, Parkinson's disease, multiple sclerosis, neoplasms
		Neuromuscular disorders: myathenis gravis, bulbar poliomyelitis, peripheral neuropathy (secondary to diabetes mellitus)
		Local structural lesions: webs, Zenker's diverticulum, abscess
Esophageal dysphagia	Difficulty with the transfer of ingested material down the esophagus	Motility disorders: achalasia, scleroderma, esophageal spasms
		Mechanical obstructions: neoplasms, peptic stricture, vascular lesions, medication-induced esophageal injury

< 3.5 g/dl, or total lymphocyte count < 1,500), suggestive of acute nutritional depletion, and more than 80% exhibited somatic protein wasting (manifested by significant weight loss and markedly abnormal anthropometric examination), reflecting the chronic character of dysphagia induced starvation [25]. Other researchers have presented associations between dysphagia severity and the degree of nutritional depletion. Moghissi and Teasdale [28] found that the amount of weight loss present in patients with esophageal cancer is highly predictive of their dysphagia severity score. Sheppard et al. [30] achieved similar predictability by using an index of body composition known as the body mass index [BMI = weight (kg)/height (m^2)] when studying 108 mentally retarded adults.

Effects of PEM on the Dysphagic

In patients with suspected malnutrition, a host of nutritonal indices are used to define the deleterious effects of the condition and the need for nutritional support. Lopes et al. [33] attempted to define a malnourished state in a group of obese patients. During a period of fasting, the patients developed abnormalities in muscle function that were restored with refeeding at a time when measurements of nutritional status (body anthropometry, serum albumin and transferrin, creatinine height index, and total body nitrogen and potassium) did not change significantly [33]. Additional studies have reinforced the finding that an alteration in skeletal muscle function is a consequence of malnutrition [34–37]. As stated earlier, the elderly have an increased susceptibility to malnutrition and thus an enhanced likelihood of altered muscle function and induced skeletal muscle wasting. In individuals with PEM, changes in muscle contractility [34], relaxation rate [33], and endurance [38] have all been reported.

Although nutrient deprivation has been shown to cause alterations in muscle function, the musculature or deglutition has received only sparse attention. To date, there has been no experimental evidence to suggest that muscles of deglutition would be spared from this degeneration during periods of deficient nutrient intake. Deglutitive muscles are mainly striated, suggesting that their metabolic fate during malnutrition may more closely resemble that of skeletal muscles than internal organs. Research has demonstrated that peripheral muscle losses occur at a rate proportional to the rate of weight loss in both hypermetabolic disease states and isolated PEM [39]. A hypermetabolic disease is characterized by an elevation in basal metabolic rate (BMR) or resting energy expenditure (REE) due to the complications of an associated disease or acute trauma. In addition, Veldee and Peth [40] postulated that the multiplicity of muscles involved in deglutition have a moderate to high percentage of type II fibers present because successful swallowing is characterized by synchronized, rapid contractions requiring speed and short bursts of activity. Type II (fast twitch) muscle fibers are affected by PEM to a much greater extent than are type I fibers (slow twitch) [34,38,39,42]. Therefore, deglutitive muscles may be among the first to atrophy because of reduced food intake, compromising the integrity of the swallow, further decreasing intakes and increasing the risk of aspiration [40].

Aspiration is typically characterized by a wet girgly cough during or immediately after a swallow and is often a cardinal symptom identifying a swallowing disorder. Usually defined as the misdirection of foods or saliva into the airway, aspiration may result in chronic and severe problems for the dysphagic because food or saliva in the lungs can lead to edema and the growth of pathogens, in particular those that cause pneumonia. PEM can negatively affect dysphagia morbidity and mortality by diminishing swallowing function, thereby further decreasing nutrient intake, increasing the incidence of aspiration and thus the risk for pneumonia or

interfering with an individual's ability to recover from aspiration pneumonia [40].

There are several ways by which malnutrition may serve to increase the frequency, nature, or magnitude of aspiration episodes. Weakness is one of the cardinal symptoms of PEM [43], and weakness of the respiratory muscles may have a great impact on aspiration risk in the PEM affected dysphagic [40]. Arora and Rochester [44] demonstrated a loss of respiratory strength directly proportional to the degree of weight loss that was distributed evenly between inspiratory and expiratory muscles in individuals with PEM. Furthermore, Lewis and Belman [35] documented a significant reduction in the cross-sectional area of both fast twitch (type II) and slow twitch fibers (type I) fibers of the diaphragm muscle, although fast twitch were affected to a far greater extent (47% vs. 23%). The significant atrophy of the diaphragm muscle fibers and, in particular, the extent of atrophy of fast twitch muscle fibers exert a considerable impact on both the contractile and fatigue properties of the diaphragm [35,44–46]. Weakness of the respiratory muscles may therefore have a great impact on the systemic consequences of aspiration in the PEM-affected patient by limiting the ability to clear aspirated material, which may lead to an increased incidence of aspiration pneumonia [35]. Because of these physiologic alterations, PEM places the dysphagic individual at a higher risk for aspiration and subsequent pneumonia than either the well-nourished dysphagic or the malnurished nondysphagic [40].

Infection is one of the most frequent and life-threatening complications of malnutrition. Many studies have shown that nearly every aspect of the body's defense can be damaged by inadequate nutrition. Immunoglobulin levels, antibody production, phagocytic function, inflammatory responses, complement function, secretory and mucosal immunity, and other defense mechanisms all may be impaired by the deficiency of biologically essential nutrients [47]. The most profound systemic effects of malnutrition, however, are on cell-mediated immunity because humans and animals with malnutrition have suppressed delayed cutaneous hypersensitivity and impaired T-lymphocyte transformation in response to mitogens [48–51]. How PEM affects the immunity of the lung is integral to the aspirating dysphagic individual for reducing the risk of pneumonia. It has been shown that PEM impairs lung defenses by impairing macrophage recruitment to the lung in response to organisms whose clearance requires normal cell-mediated immunity [52]. This research conclusion is supported by clinical observations that infections with agents such as measles and tuberculosis are more severe in humans with PEM [53–56]. Hence, dysphagic individuals with PEM may possess inadequate defenses to aspirated antigens, leading to an increased incidence of pneumonia.

The Aging and Malnutrition Correlation

Protein metabolism continues as a dynamic process throughout adult life. Although physical growth has ceased, proteins must be supplied to oversee cell maintenance and organ function in their roles as enzymes, hormones, and mediators of immune responses. In addition, sufficient protein must be consumed to replace body nitrogen losses including desquamated cells from the skin and gastrointestinal tract, body excretions (perspiration or digestive enzymes lost through the gastrointestinal tract), and end products of metabolism excreted in the urine. Advancing age, however, is characterized by an accumulation of chronic conditions and diseases affecting protein balance. The loss of appetite and frequent bouts of fever and other metabolic disturbances that affect this population lead to loss of tissue protein that must be replaced by dietary protein. The result is an added demand on nutrient intakes, which, if inadequate (to maintain calorie and protein balance), can cause a catabolic state, resulting in diminished reserves, weight loss, and malnutrition. Many studies have demonstrated an association between the aging process, the degree of nutritional deficiency, and subsequent mortality and morbidity in elderly populations [57–60]. If the malnutrition is not detected, a host of compounding, debilitating conditions may confound the health status of the elderly individual. The compromised individual may then exhibit a high vulnerability to infection and conditions of stress, which adversely affects tissue growth, wound healing, and cell-mediated immune responsiveness [7].

Primary aging is associated with protein metabolism alterations, thereby obscuring the "normal range" of laboratory parameters and body measurements. This may indicate that nutritional indices used in younger populations may not be applicable to the elderly, when early PEM detection is essential. For example, many patients lose weight while maintaining their serum albumin levels near normal until close to death [61]. Normal serum hepatic protein levels, in spite of visceral protein compromise, is commonly due to the chronic nature of PEM in the elderly [62] and other populations with the marasmic form of PEM. In a nursing home study by Rudman et al. [63], there was an apparent threshold value for albumin, cholesterol, hematocrit, and hemoglobin at which the risk of death increased. These threshold values were within the conventional normal range for adults; however, the annual death rate was 43.4% in elderly patients with serum albumin levels of 3.5–3.99 g/dl (normal ac-

ceptance range in adults is 3.5–5.0 g/dl) [63]. These results indicate that there may be a need to reevaluate the "normal" parameters used as indices for nutritional disorders in the elderly.

PEM is usually associated with children living in overpopulated city slums of underdeveloped nations, where sanitation is poor, infectious diseases may be widespread, and the foods that contain essential nutrients are typically in meager supply. Although PEM affects more than 500 million children worldwide, it is also a condition of epidemic proportions among our elderly population, with a documented incidence from 22% [64] to 59% [65]. Studies have shown that, although PEM in the elderly is common and potentially serious, it frequently goes undiagnosed until the loss of protein is significant [66].

It is well known that the elderly population experiences a higher frequency of illnesses than do young adults. The presence of various infections, cancer, autoimmune and immune complex disorders, and degenerative disease in this age group indicate a decline in immunocompetence with advancing age [67]. Because nutrition is a critical determinate of immunity, it can be postulated that the deteriorating nutritional status of the elderly may contribute to declining immunity and increased morbidity in old age. Many studies have shown that nearly every aspect of bodily defense mechanisms can be damaged by inadequate nutrition. Coincidentally, those T-cell functions most sensitive to given nutritional perturbation are precisely those that have been established to decline with age in the individual [2]. Moreover, the progressive T-cell dysfunction with advancing age has been implicated in the etiology of many of the chronic degenerative diseases in the elderly, including arthritis, cancer, vascular injury, and a pronounced increased susceptibility to infectious disease [68]. Immunoglobulin levels [67], antibody production [66], phagocytic functioin [69], inflammatory responses [70], complement function [71], secretory and mucosal immunity [47], and other defense mechanisms may be impaired by the absence of essential nutrients. Complicating this diminished immune capacity in the malnourished elderly, point-in-time surveys have detailed a 15–30% prevalence of active (protein depleting) infection in some elderly populations, with new episodes of fever and infection occurring on the average of once every 3 months [67]. Each febrile episode culminates in acute or prolonged hypermetabolism that further depletes visceral protein stores and eventually results in a negative nitrogen balance (nitrogen output exceeds input) [7]. This cyclic pattern of repeated infection and ensuing hypermetabolism places extra stress on dietary requirements, leading to further deterioration in nutritional status if intake is not increased. Swallowing dysfunction tends to prevent the convalescent "catch-up hyperphagia," which enables younger unimpaired subjects to replete their body composition after wasting illnesses [63], as type and consistency of food choices are limited, thereby decreasing the overall intake.

Implications of Malnutrition in the Elderly

The skeletal musculature plays an important role in adaptation to malnutrition or response of energy and amino acid metabolism to stress stimuli such as infection or physical trauma [1]. Advancing age however, is linked with confounding factors altering muscle function, structure, and metabolism. The deterioration of skeletal muscle function and its neural inputs begins approximately between the ages of 50 and 60 years in humans [72]. For example, individuals between the ages of 50 and 70 years have a 15% loss in muscle strength per decade [73], and those between the ages of 70–80 years have a 30% loss [74]. The decline in muscle strength with aging can be attributed to an overall loss of muscle mass as a result of diminished muscle fiber size and decreased skeletal muscle fiber number [72]. Lexell et al. [75] showed that men lose approximately 40% of their total muscle muscle mass between the ages of 24 and 80 years, with the most significant loss (30%) between the ages of 50 and 80. An example of age-related decline in muscle mass is the atrophy of the vastus lateralis muscle (muscle anterior and inferior to greater trochanter of femur). There is an equivalent loss in the number of both type I (slow twitch) and II (fast twitch) muscle fibers and a preferential loss of type II fiber cross-section [75], which may account for the slowing of contraction times exhibited in the elderly [76].

Younger people having a greater muscle mass and protein reserve can stave off bodily stresses more efficiently. They can have a reduced nutrient intake for up to 10 days with no apparent change in nutritional status or disease outcome [77]. After the initial period, inadequate intakes of protein and energy may result in significant lowering of serum albumin levels and impaired immunologic, hematologic, and hepatic function, adversely affecting recovery. In contrast, older people with less muscle mass experience the negative effects of reduced protein and caloric intakes, resulting in increased morbidity and mortality within 2–3 days [77].

Aspiration and the Effects of PEM on the Elderly Dysphagic

In individuals with an underlying swallowing disorder, the additional compromise in swallowing function

caused by PEM may significantly raise the risk of aspiration episodes and pneumonia and hinder the ability to consume adequate nutrition [39], which may result in additional deleterious effects due to concomitant nutrient deficiencies. For example, it has been noted that elderly individuals without underlying high-risk conditions have a reported excess mortality of 9 per 100,000 from lower respiratory tract infection. This rate can rise to 217 per 100,000 with one high-risk condition such as malnutrition and to 979 per 100,000 with two or more associated factors (malnutrition and aspiration) [78]. When the elderly dysphagic patient exhibits PEM, the combined weakness of respiratory muscles and depressed immune system provide little resistance to bacterial infection.

Bacteria commonly enter the lower respiratory tract and encounter an effective host defense system that prevents them from establishing an invasive infection. The fact that so few individuals who aspirate actually develop pneumonia, is itself a testimony to the effectiveness of the host defense system of the lower respiratory tract. In fact, in most individuals the tracheobronchial tree is sterile [79,80]. In contrast to the lower respiratory tract, the oropharynx is not sterile and has its own endogenous microflora, which ordinarily consists of nonpathogenic Gram-positive and anaerobic bacteria [81]. This flora shifts to one of predominately enteric Gram-negative bacilli in hospitalized and institutionalized patients [82,83]. In addition, it has been shown that the prevalence of Gram-negative bacterial colonization increases in elderly patients with increasing care needs such as eating dependency [24]. Oropharyngeal colonization occurs when microorganisms overcome the normal host defenses of the upper airway [81], and this Gram-negative colonizatioin has been shown to be directly related to the extent and severity of patient illness [82]. After microorganisms are aspirated from a colonized oropharynx, they must pass through the epiglottis and vocal cords to enter the tracheobronchial tree [81].

When periods of nutritional vulnerability overlap with suboptimal immune function caused by senescent changes, a synergistic interaction may occur. The exaggerated susceptibility of the malnourished elderly to disease, such as pneumonia, emphasizes the pivotal role that nutritional status may play in modulating this susceptibility. Risk modulation is especially critical in the elderly dysphagic because of their predisposition to malnutrition and coinciding immune dysfunction, increased aspiration risk due to weakened respiratory muscles as a result of PEM, and age-related decline of immunocompetence. Regardless of swallow function, however, the elderly individual faces changes in pulmonary function that occur with advancing age and may decrease cough effectiveness, pulmonary clearance, diffusion capacity, and oxygen saturation [4]. These age-associated alterations may enhance the risk of the elderly dysphagic to experience aspiration episodes and thus exacerbate morbidity and increase mortality in this population. In fact, Splaingard et al. [84] noted that as many as 60% of deaths related to pneumonia are secondary to aspiration from swallowing problems in the elderly. It is evident that proper nutritional management is essential in the elderly dysphagic to help stave off the confounding nutritional disorder, PEM.

Summary

Normal oral intake requires fine neuromuscular control of structures in the oral cavity, pharynx, larynx, and esophagus to reduce food to a consistency appropriate for swallowing and to propel the resulting bolus from the mouth to the stomach [10]. Elderly populations have an increased incidence of swallowing problems or dysphagia, which may be attributed to age-associated physiologic and nutritional decline, leaving them more vulnerable to confounding disease and illness. In addition, this demographic faces an increased risk of PEM, which may prevent the consumption of adequate nutrition due to the negative influence on the deglutitive musculature. PEM may also sizably increase the risk and severity of aspiration episodes due to reduced respiratory muscle strength and impaired immune function. Furthermore, an exaggerated susceptibility to disease with advancing age has been shown. When periods of nutritional vulnerability coincide with suboptimal immune function, as may be seen in elderly populations, the negative interaction between the compromised nutritional status and immunocompetence markedly increases the risk of complications and the development of concurrent disease states. One can infer, with the increased complications this population faces (malnutrition, swallowing dysfunction and age-related physiologic decline in both musculature and immune function), that the associated morbidity and

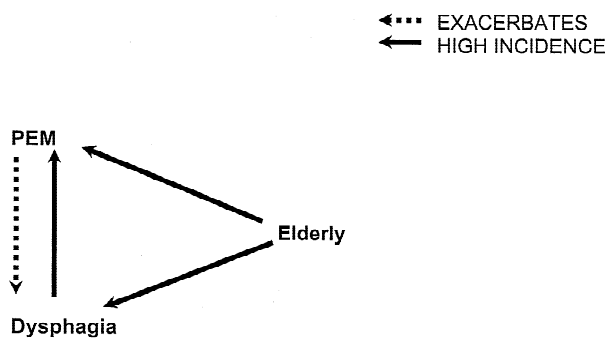

Fig. 1. The interdependency of protein-energy malnutrition (PEM), aging, and dysphagia.

mortality would be increased. Figure 1 illustrates the interrelationship of PEM and dysphagia in the elderly individual. Consequently, it would be expected that this population would be hospitalized more often, have greater lengths of stay for more serious chronic conditions, and consume a disproportionate number of health care dollars than younger persons [85]. Therefore, proper nutritional management is critical for the elderly dysphagic population to prevent the insidious development of PEM and the confounding immune and musculature weakening insults.

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