Pharmacological Nutrition After Burn Injury\textsuperscript{1,2}

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ABSTRACT Burn patients develop pathophysiological alterations, which include extensive nitrogen loss, malnutrition, markedly increased metabolic rate and immunologic deficiency. This predisposes burn patients to frequent infections, poor wound healing, increased length of hospitalization and increased mortality. The nutritional support requires high protein and high energy diets preferably administered enterally soon after injury. The effects of increased dietary components such as glutamine, arginine and (n-3) fatty acids and related compounds have been evaluated in burn victims. These components, when supplied in quantities two to seven times those in normal diets of healthy persons, appear to have beneficial pharmacological effects on the pathophysiological alterations associated with burns. However, the efficacy of immune-enhancing diets remains to be convincingly shown.


KEY WORDS: \textbullet{} burns \textbullet{} pharmacological nutrition \textbullet{} glutamine \textbullet{} arginine \textbullet{} (n-3) fatty acids

PATHOPHYSIOLOGICAL CHANGES IN BURN PATIENTS

Burn injury remains a major problem throughout the world. Extensive and deep burns cause severe metabolic, hormonal, immunologic and circulatory disturbances (Wolfe 1996).

Thermal injury induces hypermetabolism of varying intensity and duration depending on the extent and depth of the body surface affected, on the presence of infection and on the efficacy of early treatment. The increase in the basal metabolic rate varies from patient to patient and decreases progressively during the recovery period. During the first days after the injury ("flow" phase), energy requirements approach physiological limits exceeding by as much as 100\% the basal energy required by healthy persons (Cunningham et al. 1989). This increased energy expenditure contributes to malnutrition with severe body weight loss and negative nitrogen balance. In fed severely burned patients, the losses of body proteins may exceed 40 g N/d (Goodwin 1993), which is 10-fold higher than by a protein-starved healthy person (Crim and Munro 1994). These nitrogen losses occur mainly by protein exudation through the burned skin. Because of catabolic stress, body proteins are used for the production of 15–20\% of the total energy required (Goodwin 1993). Consequently patients with burns on >20\% of their body surface area should receive nutritional support specifically tailored for each individual (Deitch 1995). This applies also to patients with less severe burns suffering from malnutrition.

Burn patients have increased circulating catecholamine, cortisol and glucagon levels and maintain normal or slightly elevated insulin levels (Wilmore 1976). These hormonal changes promote increased proteolysis and lipolysis with the release of large amounts of amino acids, especially alanine and glutamine (Cynober 1989), glycerol and free fatty acids into the systemic circulation. Because burn injury causes a relatively larger increase in circulating glucagon than of insulin, amino acids and glycerol are used for gluconeogenesis. Ketogenesis declines and there is increased recycling of fatty acids in the "triacylglycerol-fatty acid cycle" (Wolfe 1996). Apparently glucose is the preferred energy substrate of cells like macrophages, leukocytes and fibroblasts in the burned area (Wilmore et al. 1977). Free fatty acids are used by normal skin and muscle cells as an alternative energy source or are metabolized via cyclooxygenase or lipoxygenase for the production of eicosanoid compounds.

A broad systemic response starts immediately after burn injury with activation of macrophages and neutrophils, the arachidonic acid and complement pathways, production of cytokines and proteases and stimulation of the metabolic response. All may adversely affect immune function (Alexander 1990). The immunologic response also is impaired with development of protein-energy malnutrition and specific micronutrient deficiency (Chandra 1991). In view of the extensive immunologic impairment, burns were classified as the major cause of acquired immunodeficiency before the high prevalence of acquired immunodeficiency syndrome (AIDS) (Sheares and Anderson 1989). The immunologic changes are transitory and may vary with age, extent of the burn, presence of infection and time after injury. Among other alterations, burn patients present abnormalities in neutrophil function, decreased opsonizing activity, reduced levels of IgG (Alexander et al. 1978), higher sensitivity of lymphocytes to inhibition by prostaglandin (PG)\textsuperscript{4} E2 (Grbic et al. 1991) and a reduced delayed skin hypersensitivity response (Munster et al. 1973).

\textsuperscript{1} Supported by a Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) postdoctoral fellowship to D.A.-S., who is on leave from Departamento de Clínica Médica, Universidade Federal de Uberlândia, 38400-902 Uberlândia, MG.

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These changes in host immunocompetence impair the immune system and predispose to infection and septic complications. Another pathophysiologic alteration that may predispose burn victims to infection is the impairment of the intestinal barrier. Intestinal permeability may be increased (Deitch 1990), probably related to ischemia/reperfusion injury (Swank and Deitch 1996) and reduced efficacy of the immunologic barrier due to secondary immunodeficiency. Impaired intestinal motility and mucosal edema associated with hypoalbuminemia foster overgrowth of intestinal pathogens. Modifications of the intestinal flora may be caused by broad-spectrum antibiotics, antacids and histamine H2 antagonists (Swank and Deitch 1996). Reduction of the mechanical and immunologic barriers of the gastrointestinal tract, with modification of the intestinal flora can facilitate translocation of bacteria and endotoxins and sepsis. This was shown in burned animals and correlated with translocation of intestinal bacteria, bacteremia and mortality (Alexander 1990). However, direct demonstration of bacterial translocation in humans has not been reported (Lippman 1995). Convincing data establishing a causal link among intestinal dysfunction, translocation of intestinal bacteria, bacteremia and organ failure in humans also are lacking (Redl et al. 1996). More convincing clinical studies on burn patients are needed to settle this question.

**NUTRITIONAL SUPPORT OF BURN PATIENTS**

The enteral rather than the parenteral route is preferred for most burn patients because it maintains trophism of the gastrointestinal tract by promoting the release of intestinal hormones and growth factors (Ziegler 1996). It also permits administration of more balanced and individualized nutritional support, eliminates catheter contamination problems and is less costly. Critically ill patients who receive parenteral nutritional support experience more infectious complications (Kudsk et al. 1992, Moore et al. 1992).

The time when nutritional support is initiated is also decisive in the prognosis of burn patients. Enteral feeding started within 6 h of injury is safe and effective and reverses several of the most important metabolic and hormonal effects of burns more rapidly (Chiarelli et al. 1990). Early initiation of enteral feeding of burned lower animals decreased their high catabolic response, translocation and survival of translocated bacteria, and prevented malnutrition and hypertrophic changes of the intestine (Gianotti et al. 1994).

Burn patients require individualized amounts of energy and protein to accelerate muscle and visceral protein synthesis and to reduce proteolysis (Wolfe 1996). Saffle et al. (1985) compared energy requirements measured by indirect calorimetry with standardized equations and found that resting energy expenditure averaged 76% of that predicted by the Curreri et al. formula (1974) and 1.47 times the basal energy expenditure calculated using the Harris-Benedict equation. Because the energy requirements of burn patients are specific and vary during recovery (Cunningham et al. 1989), the resting energy expenditure should be determined weekly by indirect calorimetry. The validity of indirect calorimetry for measurement of energy requirements of burn patients also was confirmed in studies of total energy expenditure. Goran et al. (1990) showed that data obtained with doubly labeled water correlated well with resting energy but not with basal energy expenditure. The administration of fixed amounts of energy to critically ill burn patients based on standardized equations or on the preestablished range of 167–188 kJ·kg⁻¹·d⁻¹ (De Biais and Wilmore 1994) is acceptable only when indirect calorimetry measurements cannot be made.

Alexander et al. (1980) found that administering high-protein diets to burned children improved their nutritional status, the immunologic response and the clinical course. It was recommended that burn patients receive 1.5 to 3.0 g·kg⁻¹·d⁻¹ protein with a nonprotein energy to g N ratio of ~100:1 (Gottschlich et al. 1990). Recent studies have questioned the administration of such high amounts of protein (Wolfe 1996).

In addition to excessive urea production (Patterson et al. 1997), protein depletion may be related to altered muscle amino acid transport (Wolfe 1996) and/or activation of the ubiquitin-proteasome pathway (Mitch and Goldberg 1996). Stable isotope studies have shown that muscle protein synthesis is independent of dietary protein intake in burned children receiving 1.15, 1.84 or 2.92 g protein·kg⁻¹·d⁻¹ (Patterson et al. 1997). On the basis of these considerations, 1.5 g protein·kg⁻¹·d⁻¹ appears to be adequate after burn injury (Wolfe 1996).

**PHARMACOLOGICAL NUTRITION**

Despite the effectiveness of conventional nutritional support after burn injury, the measures currently used are limited and lead to deficiencies in certain cells/tissues/organ than pharmaceutical nutrition recommends for increasing the benefits of clinical nutrition for specific diseases (Fürst 1996a). It is based on the premise that nutritional support can be “tailor made” for the specific disease and/or organ involved. Pharmacological nutrition involves administration of two to seven times (not 100 or 1000 times) the usual amounts of selected normal dietary constituents with reduction of the remaining components to avoid overfeeding.

Dietary supplementation with pharmacological levels of specific amino acids and fatty acids, alone or in combination, may alter the clinical course of critically ill patients or of injured animals. It was reported to improve immunologic function (Calder 1994), reduce the intensity and the number of infections (Grimble 1996, Grimminger et al. 1993), stimulate the proliferation of ileal and colon mucosa (Schepbach et al. 1994), improve the barrier function of the intestine (Fox et al. 1988) and maintain muscle anabolism and nitrogen balance (Jepson et al. 1988). According to Häusinger’s hypothesis, pharmacological nutrition regulates cell hydration (Finn et al. 1996, Häusinger 1995). Among the nutritional supplements most frequently used in pharmacological nutrition of burn patients are glutamine, arginine and (n-3) fatty acids.

**GLUTAMINE**

Glutamine is of major importance for muscle metabolism and is a preferred energy source for cells of the intestinal mucosa (Windmueller and Spaeth 1980) and of the immune system, particularly macrophages and lymphocytes (Calder 1994). In catabolic states large amounts of amino acids are released from tissues providing essential substrates for visceral organs for acute phase protein synthesis, urea synthesis and energy production (Gamrin et al. 1996). Despite the accelerated release of amino acids from skeletal muscles, blood glutamine may not be increased after burns (Gore and Jahoor 1994). While intracellular glutamine depletion is typical during malnutrition or varying degrees of stress, there are numerous reports that plasma glutamine concentrations remain normal. Decreased plasma glutamine levels have been reported only after severe burns, multiple trauma or multiple organ failure (Calder 1995, Stinnett et al. 1982). Although increased protelysis is important for defense against disease, it markedly reduces body stores of proteins and free amino acids (Gamrin et
al. 1996), causes serious organ dysfunction and impairs host defense. It has been suggested that depletion of the intracellular pool of free glutamine in burn patients may result from the marked decrease in muscle synthesis of glutamine (Gore and Jahoor 1994). Consequently, glutamine may be “conditionally essential” following burn injury.

A number of studies have shown beneficial effects of supplying glutamine, its precursors (ornithine α-ketoglutarate and α-ketoglutarate) (Cynober 1991) or glutamine containing diets like alanine-glutamine or glycine-glutamine (Fürst et al. 1990), in quantities corresponding to 25–35% of the dietary protein (Wilmore 1994). Glutamine may improve the clinical response of human patients or lower animals in metabolic stress by increasing nitrogen retention and muscle mass (Stehle et al. 1989), preserving integrity of the intestinal mucosa (Scheppach et al. 1994) and intestinal permeability (van der Hulst et al. 1993), maintaining immunologic function (Calder 1994) and reducing infections (van der Hulst et al. 1996, Ziegler et al. 1992). Some investigators have obtained evidence that glutamine also preserves glutathione levels in liver and other tissues (Fürst 1996b).

Interpretation of the effects of glutamine on bacterial intestinal translocation requires consideration of the types of injury and nutritional support and the method used to detect translocation (Bjarnason et al. 1995; for a review see Lippman 1995). Animal studies have shown that glutamine supplementation decreases bacterial translocation and survival of translocated bacteria and increases animal survival (Gianotti et al. 1995). However, no studies have demonstrated these responses of glutamine supplementation in human patients after burn injury. Lower animals treated with methotrexate, total parenteral nutrition or an elemental diet have shown both positive and negative responses to glutamine supplementation (for a review see Lippman 1995). Although glutamine has been reported to preserve the histological structure of the intestinal mucosa (Scheppach et al. 1994) and intestinal permeability (van der Hulst et al. 1993), its effects on the translocation of intestinal bacteria in patients have not been convincingly shown.

Parry-Billings et al. (1990) reported that plasma glutamine levels of burn patients decreased by 58% and remained depressed for ≥21 d after injury. Their culture experiments showed that the depressed glutamine concentrations were associated with significantly reduced proliferation of lymphocytes from healthy volunteers and phagocytosis by peritoneal macrophages from normal mice. They suggested that the reduction of plasma glutamine may reduce immunologic competence after burn injury. Ogle et al. (1994) also reported that glutamine improved the bactericidal ability of abnormal neutrophils from pediatric patients after burns.

Although Parry-Billings et al. (1990) and Ogle et al. (1994) suggested the efficacy of glutamine supplementation, they did not supply it to their patients. We have also not found evidence of its use after burn injury. However, when 10–30 g/d ornithine α-ketoglutarate were administered, nitrogen balance improved, plasma phenylalanine and urinary 3-methyl-histidine decreased, serum proteins rose and clinical outcomes improved (Cynober 1991). Because ornithine α-ketoglutarate increases muscle, hepatic and plasma glutamine in burn victims (Le Bricon et al. 1997), some of the positive response may be the result of glutamine repletion. These effects of glutamine and the severe depletion of intracellular glutamine after injury, deserve further investigation.

ARGinine

Arginine is a nonessential dietary component for healthy adults. It is the specific precursor for nitric oxide, and a secretagogue for insulin, glucagon, prolactin, catecholamines, corticosterone, somatostatin and growth hormone. L-arginine may affect tissue and cell growth by modulating the excretion of ornitoc acid (a precursor of pyrimidines and RNA) (Vishek 1985) and increasing collagen synthesis by the arginine-ornithine-proline pathway. Recently agmatine, (decarboxylated arginine), was shown to interact with α2-adrenergic receptors and was evaluated for the treatment of essential arterial hypertension (Reyes et al. 1994).

Intravenous infusion of arginine at doses of 0.5 g/kg (maximum of 30 g) is used as a routine test stimulus of growth hormone and insulin secretion. This quantity of arginine provokes a dose-dependent increase in urinary lysine excretion by competitive inhibition of tubular resorption (Vinnars et al. 1970) and transient hypotension to 30–40% of normal (Lorentz et al. 1993). Subjects with hepatic and/or renal insufficiency may show severe irreversible hyperkalemia (Bushinsky and Gennari 1978). Burned children and dogs suffer severe reductions in plasma arginine levels that may decline to 30–40% to normal within 4 wk after severe thermal injury. The percentage decreases in plasma and muscle arginine of burned dogs were similar (Stinnett et al. 1982). Yu et al. (1995a, 1995b) also observed that arginine flux measured in the whole body via the plasma pool was higher in patients with burns. However, the plasma citrulline flux and conversion of citrulline to arginine were within the range shown by healthy persons (Yu et al. 1995a). Due to increased degradation of arginine and to the small contribution of arginine from the urea-arginine cycle for protein synthesis, these investigators proposed arginine as conditionally essential for severely burned patients and an exogenous supply to maintain arginine balance (Yu et al. 1995a, 1995b). Vishek and Shoemaker (1986) earlier proposed that arginine is a conditional dietary essential amino acid for other disease states such as hepatotoxicity (Beauvoir et al. 1996).

Arginine supplementation for critically ill patients has been provided via the gastrointestinal tract and pharmacological doses of arginine administered have been two- to fourfold higher than the amounts in the normal American diet (Vishek 1986). Studies have shown that supplementation with arginine enhances collagen deposition into wounds of healthy people (Barbul et al. 1990) and induces a moderate increase in nitrogen retention in cancer patients undergoing surgery (Daly et al. 1988). The most evident effect of arginine supplementation is improved immunologic function characterized by increased lymphocyte mitogenesis in response to stimuli like phytohemagglutinin and concanavalin A (Barbul et al. 1990, Daly et al. 1988) and induction of macrophage cytotoxicity (Hibbs et al. 1987). Evidence shows that arginine and nitric oxide modulate calcium channels (Rozanski and Witt 1994) and sodium transport (Compeau et al. 1994).

Recent studies show marked deregulation of arteriolar tone in patients with endotoxemic septic shock (Gomez-Jimenez et al. 1995) and that increased permeability to bacteria in critically ill patients (Fink 1996) is induced by nitric oxide. Because burn patients show elevated tissue fluid nitric oxide and arginine is its only precursor (Preiser et al. 1996), caution in arginine supplementation is warranted. The use of nitric oxide inhibitors during septic shock refractory to other forms of treatment caused decreased cardiac output with increased vascular tonus, mean arterial pressure and right atrial and pulmonary artery occlusion pressure (Lorentz et al. 1993, Petros et al. 1994). Cobb et al. (1993) and Fink and Payen (1996) concluded that nitric oxide inhibitors at the doses commonly prescribed may be deleterious due to increased cardiac afterload and decreased cardiac index. These investigators suggested that
the role of endogenous nitric oxide in directing organ blood flow, maintaining the integrity of the microcirculation and blocking leukocyte-endothelial cell interactions underscores the potential importance of nitric oxide in pathogenesis of multiple organ failure. This interpretation is based upon the effect of nitric oxide produced only from endogenous arginine. The possibility that increased nitric oxide production from arginine supplementation may affect septic patients has not been addressed. Although arginine supplementation for non-septic burn patients in amounts sufficient to normalize serum and intracellular levels appears to be safe, the effects of arginine supplementation on nitric oxide production in septic burn patients should be carefully evaluated.

**(n-3) FATTY ACIDS**

Dietary fatty acids modulate the phospholipid composition of cell membranes and the quantities and types of eicosanoids synthesized. Unesterified fatty acids derived from tissue phospholipids are metabolized by cyclooxygenases with the formation of prostaglandins, prostacyclin and thromboxanes and by lipoxygenases with the formation of leukotrienes (LT) and hydroxyeicosatetraenoic acids (Grimble 1996, Hwang 1989).

(n-3) fatty acids have been administered to patients with inflammatory and immunological diseases and cancer. Oral supplementation of (n-3) fatty acids decreased the number of tender joints in active rheumatoid arthritis (Kremer et al. 1987), rate of renal function loss in IgA nephropathy (Donadio et al. 1994) and expression of the interleukin (IL)-2 receptor on mitogen-stimulated lymphocytes in inflammatory skin diseases (Soyland et al. 1994). Oral fish oil also produced a modest corticosteroid sparing effect in active ulcerative colitis (Hawthorne et al. 1992). In patients with sporadic colorectal adenoma, oral fish oil normalized the abnormal cellular proliferation pattern (Anti et al. 1994). Intravenous administration of (n-3) fatty acids did not induce significant changes in pulmonary function in cystic fibrosis (Katz et al. 1996) and had only a moderate effect on indices of disease activity in patients with Crohn’s disease (Ikebata et al. 1992). However, one patient with severe ulcerative colitis treated with high intravenous doses of (n-3) fatty acids (8.4–12.6 g/d) for 9 or 29 d showed virtual disappearance of symptoms related to the disease (Grimminger et al. 1993).

The effects of modifying the (n-6)/(n-3) ratio on inflammatory and immunological response were initially attributed to modifications in the types of PG, LT and thromboxanes synthesized because eicosapentaenoic acid [(n-3) fatty acid] competes with arachidonic acid [(n-6) fatty acid] apparently by being the substrate preferred by leukocytes (Hwang 1989). (n-3) fatty acids inhibit endothelium-leukocyte interaction and natural killer cell activity, and reduce synthesis of platelet aggregating factor (Grimminger et al. 1995). Later, reduction in production of proinflammatory cytokines IL-1α, IL-1β, IL-6, and tumor necrosis factor (TNF) were proposed as additional mechanisms of action of (n-3) fatty acids (Endres et al. 1989, Meydani et al. 1991). In healthy individuals, suppression by 18 g of concentrated fish oil/d given for 6 wks on the production of IL-1α, IL-1β and TNF persisted for 10 wk after administration (Endres et al. 1989). Pro-inflammatory cytokines participate in defense against injury but high concentrations of circulating cytokines correlate with poor outcomes in severe disease states (Blok et al. 1996, Grimble 1996). After thermal injury, TNF (De Bandt et al. 1994) and IL-6 levels (Schlutter et al. 1991) were greatly increased. IL-6 concentration continued to rise until death and reached higher levels in nonsurviving than in surviving burn patients (Schlutter et al. 1991). Patients with autoimmune and chronic inflammatory diseases benefit by the prolonged effect of (n-3) fatty acids. However, in critically ill patients the benefits of the suppression of pro-inflammatory cytokines may be limited to a specific stage of the disease and may prove undesirable at other times (Ayala and Chaudry 1995). The (n-3) fatty acids, by reducing the mitogenic lymphocyte proliferation, IL-2 production and the expression of IL-2 receptors (Meydani et al. 1991), may induce immunodepression and increase the incidence of infectious diseases. Thus (n-3) fatty acid supplementation may be beneficial as an anti-inflammatory regimen or for decreasing the severity of autoimmune diseases. However, its prolonged action and suppression of cell-mediated immunity raise doubts about its usefulness for critically ill patients.

Cyclooxygenase and lipoxygenase derivatives of arachidonic acid participate in exacerbating inflammatory responses and reduce the immunologic responses after burns. Macrophages and monocytes of burn patients produce large amounts of PGE2 (Ninnemann and Stockland 1984). PGE2 reduces T-lymphocyte proliferation and migration and the response to lymphokines. It also inhibits natural T killer cells, activates a network of suppressor cells and downregulates the antigen-presenting function of peritoneal macrophages. Series 4 LT increase cell adhesion and are potent neutrophil chemotactic factors. Hydroxyeicosatetraenoic acid has immunosuppressive effects on T-lymphocyte proliferation while enhancing suppressor T-lymphocyte activity (Ayala and Chaudry 1995).

Several studies have evaluated the effect of reduced PGE2 levels caused by cyclooxygenase inhibitors (Grbic et al. 1991, Molloy et al. 1993) or by reduction of the (n-6)/(n-3) fatty acid ratio (Garrel et al. 1995, Gottschlich et al. 1990, Saffle et al. 1997) on the inflammatory and immunologic response after burns. The reduction of PGE2 levels by cyclooxygenase inhibition partially restores sensitivity to prostaglandin, downregulates the synthesis of TNF, restores anti-peptidoglycan polysaccharide IgM to normal levels, improves the mitogenic response to phytohemagglutinin in peripheral blood mononuclear cells and increases IL-2 production. However, prostaglandins also have multiple physiologic effects, including protection of mucosal surfaces (Fukushima et al. 1992). Although the inhibition of PGE2 synthesis improves the immunologic response, it may increase translocation of intestinal bacteria. Fukushima et al. (1992) demonstrated in burned mice that the PGE2 agonists misosprostol, enisosprost and 16,16-dimethyl PGE2 reduce bacterial translocation and improve survival.

Reduction of the (n-6)/(n-3) ratio by administration of fish oil (10% of total energy) to burned guinea pigs decreased weight loss, increased skeletal muscle mass, lowered resting metabolic energy expenditure and serum C3 levels and increased cell-mediated immune responses, opsonic indices and serum transferrin (Alexander et al. 1986). However, these effects of (n-3) fatty acids were not observed by Peck et al. (1990) using burned mice infected with Pseudomonas aerugi-

nosa. Animals fed 40% of their dietary total energy in fish oil showed higher mortality than those given an equal amount of safflower oil. It is possible that the difference was due to the (n-6)/(n-3) fatty acid ratio and to the larger amount of fat. Mochizuki et al. (1984) showed that dietary lipid levels supplying 5 and 15% of nonprotein energy are optimal for nutritional support after burn injury.

Garrel et al. (1995) demonstrated that low-fat nutritional support (15% energy total) reduced infectious morbidity and shortened hospitalization time in burn victims compared with the control group given 35% of total energy as fat. However, low-fat groups, with or without fish oil, showed no differences in time to healing/percent burn, incidence of pneumonia, ni-
Immune-enhancing diets

Different combinations of nutrients like glutamine, arginine and n-3 fatty acids have been added in pharmacological quantities (2–7 times the quantities of normal intake) to diets called immune-enhancing diets, which are used for nutritional support in critically ill patients. Reductions of hospitalization time and frequency of acquired infections were reported for critically ill patients receiving immune-enhancing diets (Bower et al. 1995, Cerra et al. 1991, Daly et al. 1992, Kudsk et al. 1996, Moore et al. 1994). However, most of these studies can be criticized for their experimental design and differences in intake of nitrogen (Bower et al. 1995, Daly et al. 1992, Moore et al. 1994), energy (Cerra et al. 1991) or lipid (Daly et al. 1995) (Barton 1997).

Many effects of immune-enhancing diets remain to be evaluated in burn patients. Burn patients receiving the Shriners’ diet (Gottschlich et al. 1990), which is high in protein, low in fat, linoleic acid-restricted and enriched with (n-3) fatty acids, arginine, cysteine, histidine, vitamin A, zinc and ascorbic acid, showed fewer wound and general infections, and shortened length of stay/percent burn. However, Saffle et al. (1997) studied patients fed with a qualitatively similar diet (Impact diet, Sandoz Nutrition, Minneapolis, MN) and found that administration of an immune-enhancing diet provided no advantages over a less expensive high-protein, enteral diet (Replete, Clintec, Deerfield, IL).

In summary, data support the basic concept of pharmacological nutrition that specific supplements of normal components of the diet may improve the clinical course in some cases of burn injury. However, studies demonstrating the efficacy and cost effectiveness of these interventions remain to be conducted. Further studies are needed to enhance understanding of the mechanisms and interactions whereby dietary components influence inflammatory and immunologic responses, the barrier function of the intestinal mucosa and other pathophysiological consequences of burn injury.

LITERATURE CITED


