

FOOD AND NUTRITION NEWS

Acharya N.G. Ranga Agricultural University

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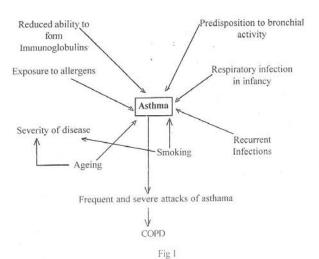
NUTRITION IN RESPIRATORY DISORDERS

The number of cases of respiratory problems are increasing due to increased atmospheric irritants, pollutants, allergies, malignant diseases and pulmonary accidents. WHO has recognised allergy and its manifestations due to air pollutants as a universal problem for most of the respiratory disorders. In 1996, the incidence of respiratory diseases in South East Asian countries was reported to be around 3.5 million cases, accounting for 40 per cent of the global disease occurance. Of the 3.5 million cases, 2.3 million were from

India alone.

About 11 per cent of the population in India suffers from respiratory problems with Acute Respiratory Tract Infection (ARI) being the leading cause of mortality during childhood (15.9% in infancy, 30.0% in under 5 age and 23.9% during preschool age). Nutrition plays a vital role in maintaining normal lung function and the immune mechanism and consequently in the susceptibility to respiratory diseases.

Hypothetical asthmatic pathway to the development of COPD



Etiology of Respiratory Disease:

The main causes of diseases of the respiratory system are infections, inhaled irritants, allergy, vascular accidents and malignancies. The consequences of infection are bronchitis, pneumonia and tuberculosis; exposure to irritants leads to chronic bronchitis, other allergic manifestations and the increasingly prevalent asthma; and vascular accidents result in pulmonary embolism and pulmonary malignancies. The mechanism involved in respiratory failure include depletion of respiratory muscle caused either by malnutrition or atrophy and dysfunction chemoreceptors due to hypoxia.

Whatever the type of pulmonary disease, it is accompanied by acute respiratory distress, compromised lung function with a disposition to respiratory muscle fatigue. Bronchial asthma is a disease of the bronchioles, the small tubes conveying oxygen rich air to the lungs. Certain trigger factors which differ from patient to patient cause spasms of the tiny muscles surrounding the bronchioles, making each respiratory movement a struggle. With treatment, the narrowed bronchioles are restored to normal. This restoration. however, is not permanent. Next exposure to the triggering factors can precipitate the next asthmatic attack. Asthmatic pathway to the development of chronic obstructive pulmonary disease (COPD) include predisposition to bronchial reactivity, leading to infection or decreased ability to form immunoglobulins leading to exposure to allergens and finally to asthma. The severity of the disease is enhanced by

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DEPARTMENTAL ACTIVITIES



Participation of Staff and Students in "Rytu Utsav"

RYTHU UTSAV-1999

The staff and post graduate students of the department of Foods and Nutrition, along with other staff from the faculty of Home Science participated in the Rythu Utsav-1999 from 18-4-99 to 22-4-99 at Nampally exhibition grounds, Hyderabad. Live samples and blowups of the technologies for mushroom cultivation, processing and utilisation of lesser known foods like rajmah, amaranth grains and red palm oil, jowar, papaya and other solar dried products were exhibited. Suitable diets for the aged and infants were also displayed.

Seminar on "Soybean Nutrition"

One day seminar on "Soybean Nutrition" was conducted by the American Soybean Association (ASA) in collaboration with the Department of Foods and Nutrition, College of Home Science, Hyderabad on 7th May, 1999, at College of Home Science, Saifabad, Hyderabad. Ms.Seema Dixit, Technical Director and Mr.Singhal, Consultant, ASA, New Delhi, attended the Seminar. Nutritive value, therapeutic effect, supplementary value and utilisation of soybean were discussed. Towards the end, a panel discussion on popularization of soybean was held in which dietitians from local hospitals, staff from National Institute of Nutrition, Director CAS, and other departmental staff participated.



Panel discussion on utilization and popularization of soyabean

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ageing, smoking and recurrent infection (Fig. 1). The causes of bronchial asthma are multidimensional. Heredity plays an important part in its genesis and past history of allergy or skin disease is generally present. Anxiety, worry, emotional disturbances, abdominal disorder, constant flatulency can precipitate an attack. The attacks start

generally in the middle of the night or in the early hours of the morning. It may be associated with wheezing, coughing, sneezing and sweating. Cough is dry at the beginning and aggravates towards the end of an attack. The allergic triggering factors are innumerable. Some common examples are housedust which includes particles of wool, cotton, fibres from furnishings, carpet, bird feathers, flakes of animal skin, fur, dust mites. Cold water baths, cigarette smoke, certain drugs, strong odours, frying fumes, spray paint, agarbathis, mosquito coils can also be trigger agents. Among foods, sour curds, tamarind, cold beverages, ice creams, deep fried foods and banana can precipitate an attack. Constipation also can initiate an asthma attack.

Pulmonary disease and metabolism

Pulmonary disease can cause either acute or chronic alteration in the normal lung function. Lung injury can be a simple localised infection or diffused alveolar damage. The metabolic response and requirements of severe lung injury are similar to sepsis, trauma, major injury or burns and are associated with hypercatabolism. Muscle proteolysis develops to maintain steady glucose supply to the brain and other glucose dependent tissues, leading to negative nitrogen balance. Hyperglycemia results from increased glucose turnover caused

by relative insulin resistance with expanded hepatic gluconeogenesis and an excess of counter regulatory hormones (glucagon, epinephrine, cortisol). Fat oxidation, being the main energy source is preferred.

Chronic lung disease presents patho-physiologically a fixed obstructive and/or restrictive defect, the most common being chronic obstructive pulmonary disease (COPD), encompassed by emphysema, chronic asthma and bronchitis. The hallmark of the disease is air flow obstruction during

exhalation due to either airway smooth muscle inflammation and edema, bronchial gland hypertrophy with mucus plugging, loss of elastic recoil or accentuated dynamic collapse of airways as lung volume decreases. These changes result in hyper inflation and trapping, elevation of residual volume and flattening of the diaphragm. The diaphragm is then at a mechanical disadvantage by shortening of its resting length prior to inspiration contraction. The patients, thus becomes both inspiration and expiration limited.

Nutritional substrates and respiratory disease

Macronutrients

Carbohydrates: In any type of respiratory distress, there is an increased utilisation of oxygen, accompanied by restricted CO2 elimination, leading to hypoxia. Metabolic state and the underlying nutritional status determine the amount of CO2 produced. A high carbohydrate load in such a condition would increase CO2 production per unit of O2 consumed creating further stress on the system. A high carbohydrate load (resulting in RQ of more than one) exceeding its utilisation would also result in lipogenesis. Contrarily a high fat diet would produce less CO2 per unit of O2 consumed and perhaps be beneficial.

Protein: Increased protein intake enhances the ventilatory response to CO2. The effect is more so with branched chain amino acids as these reduce the brain levels of tryptophan and its metabolite serotonin a vaso depressant. Lipids: Lipid emulsion provide a dense calorie source and prevent fatty acid deficiency. They also provide a nutritional option in patients with hyperglycemia with decreased CO2 production. Lipid infusions also result in increased levels of vasodilatory prostaglandins. The long chain fatty acids

connected to vasodilatory prostaglandins, unblock the hypoxic vaso constriction of the lungs.

Micronutrients

Vitamin A: Low serum levels of vitamin A are associated with higher incidence of respiratory infection. The deficiency causes atrophy of the epithelium and formation of stratified keratinised epithelium in the respiratory tract, aggravating the obstruction and process of breathing. Mild Vitamin A deficiency and ozone exposure are also associated with poor cell proliferation of epithelial tissue in the bronchioles and the alveoli of the lungs, suggesting the vitamin's role in maintenance of lung epithelial cells. Its deficiency also affects the epithelial cell differentiation and therefore serum levels of this vitamin are the best indicators of pulmonary function.

Vitamin E: Pulmonary function improves on long term intake of fish oil by lowering the nasal blood flow stimulation after an allergen challenge. This oil, rich in Vitamin E inhibits the synthesis of cyclooxygenase products that restrain leukotriene synthesis, alleviating allergic symptoms (allergic nasal congestion). Vitamin E being an antioxidant, protects pulmonary nuclei by suppressing lipid

peroxidation. Antioxidant capacity of the lungs, which is directly damaged by oxygen inhalation is protected by this vitamin

Ascorbic acid: Respiratory function is related to plasma Vitamin C levels in that its levels are correlated with forced expiratory volume per second and the forced vital capacity of the lungs, an indicator of lung function. Supplementation with vitamin C reduce the incidence of post race symptoms of URTI in ultramarathon runners and help in faster recovery of patients suffering from acute respiratory tract infection.

Vitamin B6: Decreased activity of pyridoxal phosphate dependent erythrocyte aspartate amino transferase in asthamatics results in altered B6 metabolism and low levels of circulating PLP. This decreases the release of oxygen from haemoglobin resulting in localised hypoxia and oxidant stress in tissues. Asthamatics are more sensitive to this oxygen deprivation and therefore supplementation with B6 could decrease the frequency and intensity of wheezing and the asthamatic attack.

Riboflavin: The measured energy expenditure in COPD patients exceeds the calculated energy expenditure due to

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plasma inorganic phosphate, reflecting

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increased O2 consumption of the respiratory muscles, increasing the need for riboflavin. Elevated levels of riboflavin and magnesium supplementation helps in recovery from this problem.

Iron: In respiratory distress, there is an increased respiratory rate, accounting for increased need for haemoglobin. As iron is a structural part of haemoglobin, it plays a vital role in respiration. Iron deficiency can lead to reduced gaseous exchange, creating more stress in an already hypoxic stressed system.

Copper and zinc

Copper is an important component of tissue development in lungs and zinc is an integral part of several respiratory enzymes, particularly carbonic anhydrase, which helps in removal of CO2 by combining it with haemoglobin and carrying it to the alveoli. Copper is essential for fetal lung development and foetal deficiency of this nutrient results in incidence of respiratory distress syndrome. Copper is an important cofactor for lysyle oxidase, which catalyses the cross linkage of collagen and elastin. Therefore, copper deficiency weakens collagen. Lung injuries relate to free radical oxidant damage when the lung's natural antioxidant defence system, of which

Hypermetabolism is normally associated with any critical illness, needing additional nutritional support. Oxygen consumption increases with increased nutrient administration, which increases the energy expenditure upto 25 per cent. This necessitates avoidance of overfeeding. To maintain normal Co2 level, increased mechanical level of breathing results in physical exhaustion. Around 40-50 per cent of patients with respiratory disease are malnourished leading to histological and functional copper is an integral part, becomes overwhelmed (smoking) or deficient. Flavin also provides protection against oxidative damage caused by oxidised forms of haeme protein in respiration.

Calcium: Cell mediated secretions, membrane permeability, smooth muscle contraction and airway gland secretions are calcium dependent processes. All these functions are abnormal in asthmatics suggesting an increase in calcium ion transport across the diaphragmatic muscle contraction in COPD patients. Calcium deficiency also inhibits airway smooth muscle contraction to a histamine challenge and reduces antigen induced release of mediators from the lungs.

Calcium and cortico steroids: Steroids are used in the management of severe or chronic respiratory problems. Glucocorticoid treatment can result in impaired intestinal calcium absorption and increased resorption of the bone. Increased levels of parathyroid hormone due to corticosteroid induced low calcium levels, could lead to development of osteoporosis. Glucocorticoids also lead to impairment of tubular reabsorption of calcium by the kidneys resulting in hypercalciuria. Elevated levels of parathyroid hormones improve serum calcium levels at the expense of

Malnutrition and Respiration

changes in respiratory muscles, increased susceptibility of the respiratory muscle to fatigue, decreased surfactant production, decreased protein and collagen synthesis, increased proteolysis, decreased tissue elasticity and impaired immune function.

Other pathophysiological mechanisms consequent to chronic lung disease lead to weight loss due to impaired gastrointestinal function, inadequate dietary intake, adaptive mechanism to lower oxygen consumption and work of breathing, altered pulmonary

an increase in urinary phosphate. Calcium supplementation reduces the incidence of pathological bone fractions and increases the bone mass of the distal radius in glucocorticoid treated patients. Magnesium: Magnesium plays a multiple dynamic role in pulmonary structure and function. It inhibits vasoconstriction through calcium regulation. The intracellular flux of calcium due to increased parathyroid hormone level, causes smooth muscle contraction. So, magnesium functions as a weak antagonist of calcium entry into vascular smooth muscle cell. Calcium binds to sites that release acetylcholine, initiating smooth muscle contractibility. Magnesium competes for these cites and thus regulates the bronchial activity. Other actions of magnesium include antihistaminic effect on mast cells. In an allergic challenge there is an increase in the number of mast cells, which is a primary stimulus for the release of chemical mediators like histamine and acetylcholine in presence of calcium. Magnesium blocks calcium, thus preventing the release of chemical mediators and the smooth muscle contraction. Smooth muscle relaxation in lungs depend on A-kinase, a magnesium dependent enzyme.

and cardiovascular haemodynamics limiting nutrient supply to tissues and a hypermetabolic state.

Patients with COPD show significant increase in energy expenditure due to increased respiratory muscle activity, inducing a hypermetabolic state and the subsequent weight loss. Impaired gastrointestinal function due to steroidal and nonsteroidal medication generally leads to hyperacidity, followed by maldigestion, malabsorption and the consequent malnutrition.

DYNAMICS OF ENERGY EXPENDITURE

The resting energy expenditure (REE) is high in both acute and chronic respiratory disease. Patients with advanced disease have reduced gas exchange and pulmonary function. As a result even a little deterioration in lung function increases REE and heat loss. In acute respiratory distress, this high level of energy consumption induces a hypermetabolic state resulting in weight loss. The patients also show higher energy expenditure during exercise and poor exercise tolerance, placing excessive demands on the respiratory muscles during excercise and increase the total oxygen consumption.

NUTRITIONAL MANAGEMENT

Nutritional management should aim at relieving respiratory stress. increase exercise tolerance and control complications like decreased oxygen levels in the blood. Dyspnea an obvious feature of the disease, leaves little time between breaths for eating. Chewing and swallowing worsens the feeling of distress. Respiratory distress also causes

(Contd., page 5)

Indian Dietetic Association Annual Meet-99

Annual meeting of the Indian Dietetic Association - AP Chapter was held on 12th June 1999, at Sarojini Devi Eye Hospital. The staff and PG students of the department of Foods and Nutrition participated in the meet. Dr.N.Lakshmi Devi, Assistant Professor, presented a paper on "Nutrition profile of institutionalised elderly in Andhra Pradesh" at the meeting.

Valedictory function for the short course

Valedictory function after the successful completion of a short course on "Recent Advances in Nutrition for Vulnerable Groups" was held on 1-7-99 to the participants. Dr.R. Vatsala, Dean, Faculty of Home Science, ANGRAU presided over the function. Dr.M.V.Shantaram, Dean of P.G. Studies,



Valedictory function of the short course held on 1-7-99

at the Centre of Advanced Studies, Post Graduate & Research Centre, Dept. of Foods & Nutrition, College of Home Science, ANGRAU, Hyderabad. The course was coordinated by Dr.(Mrs)K.Aruna and Dr.(Mrs)K.Uma Maheshwari. Fourteen participants from State Agricultural Universities, traditional Universities and ICAR Institutes participated in the course. Dr.Vijaya Khader, Director, CAS welcomed the gathering. The chief guest of the function Dr.I. V. Subba Rao, Vice-Chancellor, ANGRAU addressed the gathering and gave away the certificates

ANGRAU gave the valedictory address and the course material to the participants. Dr.K. Aruna, presented a brief report about the course, its content, participants profile, host and guest faculty involved. This was followed by the participants remarks. The chief guest and the president of the function complemented the comprehensive coverage and meticulous planning of the entire course. However, inclusion of tribal group as a part of vulnerable population in future courses was suggested. The function ended by vote of thank's proposed by Dr.K. Uma Maheshwari.

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a sense of fulness and pressure after eating small amounts of food, resulting in early satiety. Increased work and energy expenditure of breathing, coupled with decreased appetite and food intake leads to calorie deficit. Loss of intercoastal muscle mass contributes to difficulty in breathing. This leads to a vicious cycle of not eating in order to breath and the increasing difficulty in breathing because of failure to eat.

Due to limitations in volume intake imposed by the distressed condition, frequent small meals along with high calorie supplement should be given. Ideally 100% to 125% of calcium

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RESEARCH HIGHLIGHTS

Nutritional status and energy expenditure pattern in acute and chronic obstructive pulmonary disease

The most commonly seen pulmonary disorder is the obstructive pulmonary disease. It has been suggested that patients with this condition have significantly high energy expenditure due to increased respiratory muscle activity. This study was taken up to assess the nutritional status and energy expenditure pattern in acute respiratory disease (ARD) and chronic obstructive pulmonary disease (COPD) in both steroid dependent and non-dependent patients.

The results indicated that the food and nutrient intake of both ARD and COPD subjects was significantly (P<0.05) lower than the control group. ARD subjects, particularly steroid dependent showed lower weights for corresponding height and age; and lower height for age than the control group. COPD subjects had significantly low haemoglobin and serum Vitamin A levels and steroid dependent subjects also showed significantly high serum calcium levels.

Energy expenditure during rest as well as exercise in ARD and COPD subjects was higher by 15%, 33% and 15.8%, 26.0% respectively.

Compared to control, the post prandial resting energy expenditure (PP-REE) also increased by 33% in respiratory distressed patients.

V.Jayalakshmi Anurag Chaturvedi

Micronutrient levels in subjects with Respiratory distress:

Energy requirement during any type of respiratory distress is dynamic. More often the requirement increases by 10 per cent. Consequently, the micronutrient requirements, particularly those related to energy metabolism also increase. A study was conducted to assess the micronutrient levels in subjects suffering from acute and chronic respiratory disease, both steroid dependent and non dependent. The results indicated that there were significantly low serum concentrations of Vitamin A. C. B2, Mg. Fe and Zn in both acute and chronic disease conditions. Serum calcium levels were significantly high in steroid dependent subjects.

> P.N.Padma Kumari Anurag Chaturvedi

VISITORS

Dr. Tej Verma, ADG (Home Science) visited the AICRP - Home Science unit at the PG & RC on 20th March, 1999 to discuss issues and problems of the project and help advice the group on developing more

facilitative work environment.

The meeting was attended by the Dean (Home Science) respective Heads of all the five departments, Unit Coordinator as well as Scientists and research staff of all 5 AICRP units.

(Contd., from page 5)

requirement based on ideal body weight should be given.

Patients with bronchial type of COPD loose little or no weight, but show symptoms of edema, lack of appetite, varying degree of malabsorption and sometimes liver dysfunction. They have markedly limited ability to exercise and little dyspnea. They often appear normal or overweight. This necessitates control of edema through sodium restriction, correction of malabsorption and liver dysfunction and nutritional replenishment as needed. Slow weight loss for obese patient is suggested.

In severe COPD, diet low in carbohydrate (30% of total calories) and

high in fat (50% of calories) is suggested. This diet has low RQ and puts less stress on lungs for gas exchange. Proteins of good biological value to ensure repair of injured tissue and compensation for hypercatabolic loss can maintain positive nitrogen balance.

Micronutrients supplement is essential to prevent tissue atrophy and reduce oxidative damage in lungs.

Modifications of nutritional factors may not reverse the underlying disease but can go a long way in supporting optimum lung function. However, it should be remembered that the patients' energy needs are "dynamic" and nutrient estimations which are normal in hypermatabolic phase can become excessive on recovery.

ANNOUNCEMENT

A short course on "Recent advances in Mineral Nutrition" will be conducted from 17-11-99 to 16-12-99 by the centre of Advanced Studies, Department of Foods and Nutrition, Post Graduate and Research Centre, College of Home Science, ANGRAU, Rajendranagar, Hyderabad. TA, Food and lodging expenses for the State Agricultural University staff will be met by the organisers, Staff from traditional Universities have to meet their own TA and DA expenses. Nominations of trainees may be set to:

The Director Centre of Advanced Studies Post Graduate & Research Centre Acharya N. G. Ranga Agricultural University Rajendranagar, HYDERABAD - 500 030.

Last date for the receipt of nominations

: 30th September, 1999

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For any correspondence

Address to Editor:

Dr (Mrs) Vijaya Khader

Director

Centre of Advanced Studies Post graduate and Research Centre ANGR Agricultural University Rajendranagar, Hyderabad 500 030.

Issue Editor:

Dr. (Mrs) Anurag Chatruvedi

Associate Professor -AICRP ON PHT OF HORT CROPS