Nutrition Considerations with Hormone Balance

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May, 2011
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Receiving his Bachelor of Science degree in Nutrition from North Dakota State University, he went on to be awarded his Doctor of Chiropractic degree from Texas Chiropractic College in Pasadena, Texas in 1989 where he became a professor and postgraduate diplomate lecturer. His dietitian experience includes tenure at some of the leading hospitals in the nation - The New York Hospital, Memorial Sloan Kettering in New York City (affiliated with Cornell Medical Center), Memorial Care System and the University of Texas M.D. Anderson Cancer Center in Houston, Texas.

Dr. Grabowski has served on the State of Texas Governor’s Childhood Obesity Taskforce and is a member of the American Dietetic Association, American Chiropractic Association and the Endocrine Society. In addition to his chiropractic practice, he has developed numerous vitamin and mineral formulas for supplement companies. Professional athletes, including those of Olympic standing, seek his expertise in nutrition consultation.

His research interests include nutritional support of the athlete and the use of supplements in clinical practice for the prevention and treatment of chronic diseases such as diabetes, heart disease, arthritis, fibromyalgia and gastrointestinal disorders.
Thyroid Gland
and
Nutritional Considerations
Thyroid Gland

- Synthesizes two major hormones
  - Triiodothyronine (T3)
    - main biologically active thyroid hormone
  - Thyroxine (T4)
    - precursor of T3
- The synthesis of T4 and T3 occurs within thyroglobulin at the cell-colloid interphase.
Hypothyroidism

- The depressed metabolic rate in hypothyroidism is accompanied by a poor appetite, decreased gastric secretions, constipation, and a decrease in intestinal absorption.

- The myxedematous patient often retains fluid and develops a peculiar type of non-pitting edema, rich in mucopolysaccharides, which amounts for some of the weight gain.

- **Protein**
  - Hypothyroidism decreases the synthesis of proteins from amino acids.
  - Nitrogen excretion is decreased.

- **Lipids**
  - Associated with an elevation of the serum cholesterol.
  - Although the rate of incorporation of acetate into cholesterol is reduced, so is its breakdown.
  - Thyroid deficiency affects the entire lipoprotein spectrum causing elevation of the triglycerides as well as the serum cholesterol.
Hypothyroidism and Carbohydrates

- Glucose is absorbed slowly from the intestine resulting in a flat glucose tolerance curve.
- Hypothyroidism ameliorates existing diabetes mellitus and lowers insulin requirements.
Iodine in Thyroid Disease

- Its main function is concerned with the formation of the thyroid hormones.
- After absorption from the intestine, iodine is transported in the blood as the iodide.
- Thyroid cell for iodide causes it to be trapped there, oxidized to elemental iodine, and then incorporated into an amino acid, tyrosine, which in a series of coupling reactions becomes the active hormones, triiodothyronine and tetraiodothyronine (thyroxine).
- The optimal adult requirement for iodine is approximately 200-300 ug/day.
- In the United States, iodized salt contains 0.01% potassium iodide.
  - Thus the requirement could easily be satisfied on an intake of 3-4 g salt daily.
- Conditions such as pregnancy, lactation, infection, puberty, and acromegaly increase requirements considerably above those of the normal adult.
Iodine and the Thyroid Gland

- Crucial for the formation of the hormones at the thyroid gland.
- ~ 59 - 65% of total body iodine is contained in the thyroid hormones.
- Severe iodine deficiency causes goiter and cretinism.
- Milder degrees of deficiency have detrimental effects on energy metabolism, cardiovascular function, bone turnover and neuromuscular alterations, among others.
Iodine Status

- There has been a high prevalence of both suboptimal iodine and zinc status among the children concomitant with the relatively high levels of anemia and marginal vitamin A deficiency (Thurlow et al. 2005).
- Mild to moderate iodine deficiency can have detrimental effects in children, causing impairments in both neuropsychological and physical development (van den Briel et al. 2000; WHO/UNICEF/ICCIDD, 2001).
Iodine Sufficiency

- The presence of more Zn atoms at the thyrocyte can lower the endonuclease activity.
- Production and release of TSH are stimulated by the lowering mainly of T3, but also T4 concentration.
- In all I-deficient groups, TSH levels are often significantly greater than in controls.
Iodine, Selenium and Zinc

- Current evidence indicates that the simultaneous occurrence of nutritional deficiencies of more than one of these micronutrients could be more common than previously considered. (Diplock 1992, Vanderpas et al. 1993).

- Combined deficiency of selenium and iodine has been suggested as a potential determining factor in the development of the myxedematous or nervous form of endemic cretinism. (Goyens et al. 1987, Vanderpas et al. 1990).

- Two-and three-way nutrient interaction may have distinct manifestations in thyroid function.
Selenium and the Thyroid Gland

- Participates in the extrathyroidal deiodination of T4 to the active form T3 (Arthur et al. 1993).
- Component of deiodinase type I, which transforms T4 into T3 in liver, kidney, muscle and thyroid.
- Evidence suggests that deiodinases type II and Type III may also be selenium dependent (Arthur 1997).
- Deiodinase type II plays an important role in providing intracellular T3 to the brain and pituitary.
- Selenium also plays a role in oxidative stress control at the thyroid as a component of the enzyme glutathione peroxidase.
Histologic Evaluations

- Thyroid glands of selenium-deficient animals showed an active fibrotic process in which the inflammatory reaction and excess of transforming growth factor-b play a key role. (Contempre et al., 1996)

- The most severe morphologic alteration of the thyroid cell were caused by Zn deficiency alone or in combination with the lack of Se.

- When Type I deficiency was simultaneously present, cellular changes were not as severe as seen in conditions of adequate Type I.
Zinc and the Thyroid Gland

- Protein synthesis
- Involved in T3 binding to its nuclear receptor (Miyamoto et al. 1991).
- In a zinc depletion-repletion study conducted in humans, Wada and King (1986) observed that circulating TSH, total T4 and free T4 tended to decrease during the depletion phase, returning to control levels after zinc repletion.
- Zinc deficiency can indirectly affect thyroid hormone status by decreasing energy intakes.
Mechanisms by Which Zinc Deficiency May Affect Cell Structure:

- **1st Mechanism**
  - Role of zinc in a number of enzymes involved in nucleic acid metabolism and protein synthesis (Valle 1990).

- **2nd Mechanism**
  - Related to the critical role of zinc as a regulatory agent in apoptosis.
    - In apoptosis, activation of an endonuclease is responsible for chromatin cleavage and major nuclear morphologic changes, as typically observed during this process (Arends et al. 1990).
  - An intracellular pool of zinc atoms is crucial for modulating the activity of this endonuclease (Perry et al. 1997).
Zinc and Nuclear Receptor

- A possible explanation, the nuclear receptor for T3 is partially Zn dependent.
- Zinc participates in the formation and mechanism of action of thyrotropin-releasing hormone (TRH).
- Processing of pre-TRH to form TRH is zinc dependent via post-translational processing enzymes such as carboxypeptidase H. (Pekary and colleagues-1991.
- The mechanism of action of TRH at the hypophysis is bimodal.
  - First - it stimulates the release of stored hormone.
  - Second - it stimulates transcription.
  - The intimate mechanism involves an increase in intracellular Calcium and an activation of protein C kinase.
  - Protein C kinase is a Zn-metalloenzyme; specifically, the regulatory domain coordinates four atoms of zinc (Quest et al. 1992).
Zinc Status

- Serum retinol has been positively associated with serum zinc.
- In some studies, children with serum retinol <1.05 umol/L had an almost two-fold greater risk of being zinc deficient compared to those with serum retinol levels indicative of adequate vitamin A status.
- Serum albumin concentrations have also correlated positively with serum zinc concentrations.
  - Approximately 70% of zinc is transported in the serum bound to albumin.
- Males have shown to have an almost two-fold greater risk of being zinc deficient than females, possibly a reflection of the higher zinc requirements of males than females because of their higher lean body mass and growth rate (Hotz and Brown 2004).
Zinc Absorption and Excretion

- Studies have shown that phytate interferes with zinc absorption both from vegetable and animal protein sources.
- In addition to phytate, vegetables contain other organic compounds, e.g., phaeophytin, a degradation product of chlorophyll, which may bind metals and make them unavailable.
- Calcium may increase the inhibitory effect of phytate on zinc absorption.
- The normal zinc content of sweat is approximately 115 ug/100ml.
- A normal individual might lose 5 or more mg of zinc/day in sweat.
Thyrotoxicosis

- It increases the need for all dietary nutrients.
- Hypermetabolism occurs in almost all organs except the spleen, brain, and tests.
- Hyperthyroid patients may require 2-3 times the normal daily calorie requirement in order to maintain body weight.
- Excessive amounts of thyroid hormones, result in greater catabolism of protein with gradual wasting of the lean body mass.
  - Kekki reported that both synthesis and catabolism of albumin are accelerated.
- Fat is mobilized from depot stores, plasma free fatty acids may initially increase.
  - Excessive thyroid hormone lowers serum cholesterol, phospholipids, and triglycerides.
  - The fall in serum cholesterol level is best explained by increased degradation and biliary excretion of cholesterol.
Thyrotoxicosis and Vitamins

- Increases the need for almost all the vitamins especially pyridoxine, pantothenic acid, and thiamin.
  - A deficiency of pyridoxine in a normal animal depresses the uptake of radioactive iodine by the thyroid gland.
- If pyridoxine deficiency exists, gastric secretion decreases with a subsequent decline in vitamin B12 absorption.
- Mohamed and Roberts have reported that seven of ten thyrotoxic patients showed an abnormality in the breakdown of histidine to glutamic acid.
  - This reaction has been shown to be folate dependent and suggests that a relative deficiency of folic acid exists in the thyrotoxic patient.
- Excessive thyroid secretion has been demonstrated to increase the need for vitamin A, riboflavin, ascorbic acid, vitamin D, choline, and perhaps vitamin E.
Thyrotoxicosis and Minerals

- Serum and urinary calcium levels are mildly elevated.
- Urinary losses of phosphorus are even greater than calcium.
- Total body magnesium is low in spite of normal or high serum levels.
- Losses of potassium in the urine depend on the extent of protein catabolism.
- Mineral absorption from the gut can compensate for excretory losses if adequate amounts are in the diet.
- Hypercalcemia stimulates thyroid parafollicular cells to secrete calcitonin resulting in a lowering of the calcium concentration.
Zinc and Hyperthyroidism

- High serum Zn values have been reported in hyperthyroidism and serum Zn concentrations have been noted to correlate well with T3 and T4 levels.
- The present study confirms that the red blood cell Zn concentration is decreased in thyrotoxic patients and increased in hypothyroid patients.
- Thyroxine administration to rats significantly enhanced the rate of Zn-turn-over and altered its distribution in various organs leading to an almost 50% depletion in soft tissue Zn.
- A higher than normal urinary Zn excretion in hyperthyroidism has been reported by Bremner and Fell and Nishi et al; this may result from an increased extracellular fluid Zn loss.
- Excess thyroid hormone may result in the mobilization of Zn from tissues such as erythrocytes, bone, and muscle by increased catabolism.
It is well known that Zn, RBP, and T4 to some extent, exist in blood complexed to PA which is a carrier for both thyroid hormone and Zn in the plasma.

Zn is considered to play a regulatory role in the release of RBP from the liver.

Morley et al noted that serum Zn levels correlated positively with T3 but not with T4 and that RBP and PA levels correlated positively with T3 in patients with alcoholic cirrhosis.
Copper and the Thyroid Gland

- Most plasma Cu (approximately 93%) is bound to ceruloplasmin and a small fraction to albumin (6 to 7%) or is chelated to amino acids (<1%), which is diffusible.

- Thyroid hormones enhance the synthesis of lysozymal enzymes in muscle and are necessary for the catabolic response and increase the concentration of free amino acids in plasma.
  - A general increase in plasma amino acid concentrations in hyperthyroid rats has been reported, and an increased plasma ceruloplasmin level in patients with hyperthyroidism.
Manganese and the Thyroid Gland

- There has been statistically significant correlation between erythrocyte Mn concentrations and T4 or T3 levels.
- These findings suggest that the thyroid hormone may in part, also chronically affect Mn metabolism.
- Mn is responsible for superoxide dismutase.
Overfeeding

- Increases T3 production.
- Energy restriction decreases serum T3 concentrations.
  - Mediated by a reduction in the extrathyroidal conversion of T4 (Katzeff et al. 1990).
Hypothyroidism and Anemia

- In humans, a mild anemia is found in the majority of patients with thyroid deficiency.
- The anemia may be hypochromic, megaloblastic, normochromic, or normocytic.
- Hypothyroidism reduces iron and vitamin B12 absorption and at least some of the associated anemias can be corrected by the appropriate nutrient.
Iron and Iodine

- Iodine deficiency is also exacerbated by coexisting iron deficiency (Zimmerman et al. 2000).
- In subjects with iron deficiency anemia:
  - Levels of plasma thyroxine (T4) and triiodothyronine (T3) are lower.
  - Conversion of T4-T3 is slowed
  - Concentrations of TSH are elevated (Beard et al. 1998).
Iron and Thyroid

- The 2 initial steps of thyroid hormone synthesis are catalyzed by thyroperoxidases and are dependent on iron.
  - Iodine incorporation into tyrosine residues of thyroglobulin and covalent bridging of the residues—are catalyzed by heme-containing thyroperoxidases.
  - Theoretically, severe iron deficiency could lower thyroperoxidase activity and interfere with thyroid hormone synthesis.
- Animal and human studies suggest that iron deficiency impairs thyroid metabolism.
- Iron deficiency anemia decreases plasma thyroxine (T4) and triiodothyronine (T3) concentrations, reduces peripheral conversion of T4 to T3, and may increase concentrations of thyrotropin.
- Compared with healthy control subjects, iron-deficient adults have lower circulating T4 and T3 concentrations and higher thyrotropin concentrations.
Hypothyroidism and Vitamin A

- Thyroid hormones facilitate the conversion of the carotenes of vitamin A.
- When thyroid insufficiency exists, hypercarotenemia may develop imparting the yellow discoloration to the skin of myxedematous patients.
Vitamin A and Iodine

- Coexisting suboptimal vitamin A status (<1.05 umol/1) can also exacerbate iodine deficiency.
- Mechanism (proposed)
  - Involves inhibition of thyroid-stimulating hormone (TSH) secretion by the pituitary and thyroid hormone transport, mediated in part through retinol-binding protein and transthyretin.
Parathyroid Disorders

- **Parathormone**
  - Chief function is to control calcium levels in body fluids.
  - Secretion of the hormone results in an increase in the serum calcium.
  - Increases bone resorption.
  - Calcium absorption from the gut.
  - Renal tubular calcium reabsorption.

- Demineralization of bone results, which produces the clinical picture of rickets in children and osteomalacia in adults.

- Calcitonin is a polypeptide hormone from the thyroid gland that lowers serum calcium and phosphate.

- Hypocalcemia causes parathormone to be released from the parathyroid to effect a rise in the level of calcium.
Overproduction of parathormone results in a rise in serum and urinary calcium at the expense of bone demineralization.

Peptic ulcer, as well as the classical but increasingly less frequent, bone lesions are also seen.

Appetite, absorption, and utilization of food in hyperparathyroidism may be altered due to the gastrointestinal disturbances.

Pancreatitis, which has been reported to be associated with hyperparathyroidism could affect nutrition by causing anorexia, abdominal pain, intolerance to certain foods, malabsorption, and weight loss.

Elevated serum calcium levels result in inhibition of the action of antidiuretic hormone on the renal tubule with resultant excessive water excretion.

Excessive secretion of parathormone has a marked catabolic influence on body protein.
Parathyroid Hormone and Metabolic Syndrome

Cardiovasc Diabetol., 2011
Hyperparathyroidism and Minerals

- Excessive excretion:
  - Inorganic phosphate
  - Calcium
  - Potassium
  - Magnesium
Hypoparathyroidism

Deficiency in parathormone results in:
- Hypocalcemia
- Decreased urinary excretion of calcium
- Elevated serum phosphorus.

Tetany is the most prominent clinical manifestation of acute hypocalcemia.

Hypoparathyroidism is associated with decreased intestinal absorption of calcium.

Therapy with large doses of vitamin D (50,000-300,000 units daily) and a diet containing 1,000 mg/day calcium usually suffice to maintain serum calcium at normal levels.
Adrenal Disorders

- Adrenal disorders not only affect nutrition, but the adrenals are quite sensitive to nutritional abnormalities.
- Mean cortisol secretion rate in normal adults is 18mg/24hr and 28mg/24hr in grossly obese individuals.
- Ascorbic acid level
  - High in adrenal cortex, some experts have concluded that it is highly utilized in these glands.
  - Many symptoms of scurvy, i.e., fatigue, loss of muscle strength, digestive disorders, and reduced ability to tolerate stress resemble those of adrenal insufficiency.
- Vitamin A deficiency causes both structural and functional changes in the adrenal gland.
  - The interference of gluconeogenesis in vitamin A deficient animals is probably an indirect effect of the vitamin lack and more directly related to adrenal insufficiency.
Hyperfunction of the Adrenal Cortex

- Cushing’s syndrome is essentially excessive glucocorticoids.
- Cushing’s syndrome
  - Hyperplasia of the adrenal cortices.
  - Fluid retention
  - Hypernatremia
  - Hypokalemia, in spite of normal levels of aldosterone.
  - A metabolic alkalosis that is the result of potassium and hydrogen ion losses in the urine is often present.
    - The losses are directly related to the amount of cortisol secreted.
Cushing’s Syndrome and Protein

- Protein metabolism is profoundly affected in Cushing’s syndrome.
- Gluconeogenesis, a major metabolic effect of the glucocorticoids, is enhanced.
  - Results in protein catabolism.
    - Amino acids are then available for conversion to glucose.
    - Increased nitrogen excretion is the result of protein breakdown.
Catabolic Effects of Glucocorticoids

- Striae, due to loss of subcutaneous connective tissue.
- Osteoporosis, due to the loss of protein matrix of bone.
- Wasting of the extremities, due to muscle atrophy.
- Nitrogen losses may be associated with an aminoaciduria.
  - Protein supplementation may decrease the losses.
  - 600 mg L-lysine to the diet of patients taking steroids decreases the catabolic effects if the protein intake exceeds 88 g/day.

- Carbohydrates
  - Cortisol - an increase in blood sugar and glycogen stores occurs.
  - Hyperglycemia causes increased insulin secretion.
    - Eventually islet cell reserves may become depleted and “steroid diabetes” ensues.
  - The hormone that maintains normoglycemia during the fasting state, when secreted excessively, frequently results in diabetes mellitus.
Glucocorticoids and Vitamins

- The conversion of carotene to vitamin A is inadequate in cortisone-treated animals.
- Vitamin B12 and thiamin deficiencies are accentuated when steroids are administered.
- The requirement of pyridoxine, which is the cofactor of one of the enzymes (glutamic pyruvic transaminase) utilized in gluconeogenesis, is also increased significantly.
Adrenocorticoids and Minerals

- Excessive adrenocorticoids cause marked changes in electrolyte requirements.
  - Potassium losses in the urine are excessive and hypokalemic alkalosis results.
  - Sodium is retained due to the mineralcorticoid action of the hormones.
  - Hypertension is common in Cushing’s syndrome.
  - Calcium losses combined with protein breakdown of the bone matrix results in osteoporosis.
  - A higher calcium intake may protect steroid-treated patients from osteoporosis.
Hyperaldosteronism

- Aldosterone has a potent mineralocorticoid influence with very little glucocorticoid function at physiologic levels.
- Aldosterone stimulates RNA synthesis of various enzymes that participate in sodium transport.
- Excessive secretion leads to sodium retention and potassium losses by the renal tubules, sweat glands, and other secreting glands.
- Hypokalemia is associated with a metabolic alkalosis.
- Glucose tolerance is often decreased in hyperaldosteronism.
  - This may be due to the hypokalemic effect on the cell.
- In humans a negative magnesium balance is found in primary aldosteronism.
  - Increased magnesium excretion is due to a direct action of the hormone.
- Potassium and calcium losses occur in the feces and urine, and an adequate intake of potassium water, magnesium, and calcium are required.
Addison’s Disease

- The primary defect in Addison’s disease is a deficiency of all adrenocortical steroids.

- Signs/Symptoms
  - Weakness, weight loss, dehydration, and the inability to withstand stress.
  - Anorexia is common as well as nausea, vomiting, and diarrhea.
  - Dehydration contributes to the weight loss, which is frequently severe.

- Hypoglycemia is a result of decreased carbohydrate absorption and a deficiency in “new glucose” formation.
Glucocorticoids and Gluconeogenesis

- Increase gluconeogenesis from the products of protein breakdown and inhibiting utilization of glucose by:
  - 1<sup>st</sup>- increasing mobilization of protein by way of amino acids to the liver for later transamination and conversion into glucose.
  - 2<sup>nd</sup>- facilitating liver glucose production from three-carbon precursors.
  - 3<sup>rd</sup>- selectively increasing glucose 6-phosphatase activity resulting in less available glucose 6-phosphate for glycolysis.

- The association of diabetes and Addison’s disease is higher than expected in view of the tendency towards hypoglycemia with adrenal insufficiency.
Addison’s Disease and Nutrients

- Vitamin B complex increases the survival of adrenalectomized rats.
- Ascorbic acid retards the degradation of cortisol and thus increases the availability of the hormone.
- Depletion of sodium and retention of potassium occurs in untreated Addison’s disease.
- Hypercalcemia may be seen, but this finding is not common.
- Hypermagnesemia is associated with Addison’s disease and results in depression of the central nervous system and peripheral neuromuscular function.
Polycystic Ovarian Syndrome (PCOS)

Most common cause of female infertility due to anovulation in the United States.

Affects approximately 6-10% of reproductive-aged women.

There is a genetic susceptibility to the disorder.

- Kahsar-Miller et al. reported that 32% of sisters and 24% of mothers of patients with PCOS were affected by the syndrome, and those who had the syndrome demonstrated significantly higher mean testosterone, free testosterone, and DHEAs values.
PCOS and Insulin Resistance

- PCOS is associated with increased insulin resistance and pancreatic B-cell dysfunction independent of the frequent attendant obesity.
  - Inherited as an autosomal dominant trait, mothers and sisters, as well as brothers, of women with PCOS were found to be more insulin resistant compared with matched control subjects without family history of PCOS and diabetes.
- Insulin may act directly and/or indirectly, through the pituitary, to stimulate ovarian androgen production.
- It has been shown that 82% of women with type 2 diabetes mellitus have PCOS on ultrasound scan but only 52% of them present clinical symptoms of the syndrome.
- Asian women with PCOS and especially those with family history of type 2 diabetes mellitus are more insulin resistant than matched controls.
Metabolic Syndrome

- More prevalent in women with PCOS than in the general U.S. population, even when matched for both age and body mass index (BMI).
- Women with PCOS who have the MBS have a higher incidence of hyperandrogenism.
- In one study, a 20-to 29-yr-old group, women with PCOS had a nearly 8-fold greater prevalence than women in the general population (44.8 vs. 5.9%, respectively).
- Similarly, women with PCOS aged 30-39 had a nearly 4-fold increased prevalence of the MBS.
Hormonal Characteristics

- Women with PCOS and the MBS
  - Found to be more hyperandrogenic than women with PCOS lacking the MBS after adjustment for age and BMI.
  - Levels of serum free testosterone higher.
  - Serum SHBG concentrations were significantly lower.
    - SHBG has been noted to correlate inversely with insulin sensitivity.

- PCOS manifest increased atherosclerosis and a 7-fold increased risk of cardiovascular disease.

- Women with concurrent PCOS and MBS presented more frequently with the phenotypic feature of acanthosis nigricans, a putative biomarker of insulin resistance, than women with PCOS only.

- Majority of women with PCOS had at least one abnormality of the MBS present (91%).
Metformin and PCOS

- **Mode of action**
  - Inhibition of hepatic glucose output, which lowers insulin concentrations.
  - Dosages range between 1,500 and 2,500 mg/d
  - Appears to reduce insulin levels and decreases ovarian androgen production.
  - Can induce ovulatory cycles in more than 50% of patients with PCOS.
  - Reported to reduce the incidence of first-trimester pregnancy loss in women with PCOS.
    - Once pregnancy has been achieved, PCOS women are at increased risk for gestational diabetes, pre-eclampsia, and pre-term delivery.
N-Acetyl Cysteine and PCOS

- Nonessential amino acid.
- A mucolytic product with insulin sensitizing properties.
- Study: 150 women diagnosed with CC-resistant PCOS, aged 18-39 years undergoing therapy for infertility were included.
  - The patients were assigned randomly to receive either NAC 1.2 g/d (group I) or placebo (group II) with CC 100 mg/d for 5 days starting at day 3 of the cycle.
  - **CONCLUSION:** The NAC as an adjuvant to CC was more effective than placebo for CC-resistant patients with PCOS. It is safe and well tolerated. (Fertil Steril - 2/2005)
Oral Contraceptives and Nutritional Status

- The use of contraceptive pills has been shown to decrease the physiologic levels of six nutrients--riboflavin, pyridoxine, folacin, vitamin B12, ascorbic acid and zinc (J Reprod Med., 1984).
Oral Contraceptives Agents (OCA)

- Oral contraceptives have been shown to reduce folate storage in the body and results in megaloblastic changes.
- Homocysteine has also been shown to be elevated with OCA usage.
Vitamin B6 and Oral Contraceptives

- OCs alter vitamin B6 tryptophan metabolism in 2 ways.
  - They increase hepatic tryptophan oxygenase activity resulting in a surplus of tryptophan further in the pathway to niacin.
  - OCs also reduce the proclivity for kynureninase, the pyridoxal phosphate of vitamin B6 dependent enzyme of tryptophan metabolism perhaps increasing the need for plasma pyridoxal phosphate (J Egypt Public Health Assoc., 1988).
Insulin and Magnesium

- Research results provide significant evidence that oral Mg supplementation improves insulin sensitivity even in normomagnesemic, overweight, non-diabetic subjects emphasizing the need for an early optimization of Mg status to prevent insulin resistance and subsequently type 2 diabetes. (Diabetes Obes Metab., 2011 March)
Insulin and Vitamin D

- Low vitamin D levels have been correlated with insulin resistance (J Pediatr Endocrinol Metab., 2011).
- Vitamin D not only facilitates the biosynthetic capacity of β cells but also accelerates the conversion of proinsulin to insulin (J Endocrinol, 1999).
- Vitamin D supplementation has been reported to improve insulin secretion in vitamin D–deficient and nondiabetic subjects and in patients with type 2 diabetes (Diabetologia, 1986 and Bone Miner, 1986).
- Research suggests that vitamin D deficiency affects β cell function and that vitamin D supplementation improves β cell function.
Vitamin D and Magnesium Relationship

- Vitamin D may increase the gastrointestinal absorption of magnesium and increase the transport of magnesium from extra- to intracellular space (J. Nutrition).
- Magnesium is required for the hepatic 25-hydroxylation of vitamin D (AJCN).
Growth Hormone and Vitamins

- Growth hormone administration to animals with marginal body stores of vitamin A depletes the vitamin faster than would have otherwise occurred.
- Pantothenic acid-deficient rats injected with growth hormone develop acute deficiency symptoms, and pyridoxine-deficient animals have more severe acrodynia.
Growth Hormone and Minerals

- Retention of phosphorus, magnesium, potassium, and sodium is consistently found with administration of growth hormone.
- Excessive amounts of growth hormone causes hypercalcemia.
- The levels of serum phosphorous and alkaline phosphatase are usually increased when the disease is active.
Hypopituitarism

- Panhypopituitarism is associated with a lowered metabolic rate.
- Anorexia may result in dietary deficiencies.
- Fat stores are preserved due to the normal and unopposed insulin action that also accounts for the susceptibility of these patients to hypoglycemia.
- Absorption of food is impaired with hypopituitarism and gastric secretion is reduced.
- An amelioration of diabetes mellitus is noted in patients who develop hypopituitarism.
- Caloric undernutrition inhibits secretion of the anterior pituitary hormones and may produce symptoms indistinguishable from primary hypopituitarism.
- Decreased metabolism, developmental failure, gonadal atrophy, and intolerance to stress may develop.
- Amenorrhea is common in both conditions.
- Inhibition of anterior pituitary secretions also occurs if there is a lack of essential amino acids, protein, or vitamins.
Commonly asked questions

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