Allergies

Etiology: genetic predisposition, in utero exporures, biochemical individuality (more permeable gut or sensitive immune responses), toxic load, poor nutrition or digestion, greater need for nutrients not provided by the diet, stress or chronic illness, gut dysbiosis, leaky gut

Signs and Symptoms: inflammation (swelling, heat, redness and pain), edema, runny nose, watery eyes, itching, hives and bronchoconstriction Pathophysiology: the inflammatory response damages cells and tissues, immune system becomes hypersensitive launching an attack against harmless things a) Type 1 reaction: IgE reactionimmediate response (can be localized, general, mild or severe) b) Type 2 reaction: IgG reaction-IgG's react to surface antigens on whole cells (can be ones' own cells resulting in autoimmune disease). Cells become injured and no longer function properly or are inappropriately stimulated causing hypertrophy and hyperfunctioning c) Type 3 reaction: IgG reaction- free soluable antigen (rather than attaching to the cells) like proteins, DNA or self antigen (autoimmune) released inappropriately into the blood)-food sensitivities (typically delayed)/threshold level of allergen that is required to illicit symptoms/ IgG memory for the allergen may only last about three months, typically delayed

d) Type 4 reaction: cell mediated immunity-delayed hypersensitivity/T cells react to an allergen and cells are destroyed in the process eg: contact dermatitis (poison ivy, toxins attach to cell), some autoimmune disease, transplant rejection/ response is 1-3 days/ body views previously recognized cells as foreign

Recommendations: text p. 26-28

Autoimmune Disease

Etiology: Antibodies and sensitized Tcells are produced against one's own tissues (attacking the body)/can be a type 2, 3 or 4 hypersensitivity <u>Signs and Symptoms</u>: all the same kinds of symptoms aforementioned in the signs and symptoms of allergy, plus the diagnosis on p. 28 of your text.

Pathophysiology: inefficient lymphocyte programming making it so that their is a misunderstanding of what is foreign/changes in the HLA antigens on cell surface where lymohocyte can no longer recognize the antigen/cross reaction where an antibody is made for something foreign, which HLA markings look similar to and are attacked/free circulating immune complexes cause inflammation in surrounding tissues and then other antibodies are created in response to these abnormal ones causing even more damage in connective tissues (rheumatoid arthritis) /for the same reason as Type 2 sensitivity T cells are implicated in the destruction of body cells

Lupus

Etiology: genetic predisposition, UV light, canavanine (found in insufficiently sprouted alfalfa, hydralazines or hair dyes), possibly viral(retrovirus, adenovirua) or bacterial triggers, aspartame makes it worse

Signs and Symptoms: arthritis, arthralgia, anemia, pleurisy, photosensitivity, fever, skin rashes, kidney involvement, butterfly rash, hair loss/early symptoms: fatigue, weakness, weight loss, fever and polyarthritis/version that mainly affects the skin called discoid lupus Erythematosus

Pathophysiology: immune complexes can deposit in the kidneys and interfere with normal kidney function/ profuse collagen formation in the dermis and much healing with fibrosis resulting in induration of the skin and a mask look/blood vessels dilate below the skin surface into spidery telangiectasia/fingers appear claw like because of scaring (sclerodactaly), Raynaud's syndrome (because the blood supply to the fingers is interrupted by scarring), difficulty swallowing because of fibrosis in the esophagus Recommendations: p.31-33 of the text

Edema

Etiology: inflammation in which proteins and fluid escape from the blood into the tissues/poor circulation and pooling of the blood, pressure in the vessels pushing fluid out of blood into tissues/low protein (low water holding potential in the blood and then it leaks out into the tissues)/ lymphatic blockage (by infection or parasite) and the lymph fluid builds up between the cells

Signs and Symptoms: swelling and puffiness of tissues, skin appears stretched and shiny, pitting of the skin

Pathophysiology: failure of the lymphatic system to maintain and balance body fluids (movement of fluids from the capillaries exceeds the lymphatics ability to drain tissue resulting in accumulation in the tisues)

Recommendations: p.41 & 42 of text

Acne Vulgaris

Common acne breakout on the face, upper back and chest consisting of comedones, cysts, papules and pustules.

Etiology: hormonal fluctuation (puberty and menopause), response to toxic accumulation when liver and kidneys are too overloaded,

dysbiosis, stress, excess iodine, low levels of zinc, vitamins A, B6, high arachidonic acid levels or low essential fatty acid levels, excess dietary fat

Signs and Symptoms: breakout on the face, upper back and chest consisting of comedones, cysts, papules and pustules.

Pathophysiology: overproduction of sebum blocking the skin follicles. The sebum is broken down by skin bacteria into irritating volatile fatty acids that cause inflammation and pus formation, bacteria overgrowth (propionibacterium- bacteria that causes acne thrives in blocked skin follicles).

Recommendations: p. 86 & 87

Acne Rosacea

Chronic rose coloured eruption on the face (usually localized to the middle of the face, including the nose). Women are affected more often than men and it is usually first observed between the ages of 30-50.

Occurs more often in fair skinned women who flush or blush frequently. Etiology: low Hcl, Vitamin B complex deficiency, reduced pancreatic lipase. Menopause, medications like topical steroids or vasodilators and cosmetics that contain alcohol, witch hazel, fragrances, hydro-alcohols or acetone can worsen the condition. Foods that aggravate: liver, yogurt, sour cream, cheese, chocolate, vanilla, soy sause, yeast extracts, vinegar, eggplant, avocado, spinach, beans, citrus, tomatoes, bananas, raisins, figs, spices and foods high in histamine.

Signs and Symptoms: acne with spidery-like blood vessels in appearance, papulopustular (bumpy lesions), nasal scarring and deformity, or inflammation extending to the eyes (lids, lashes or conjunctiva)

Pathophysiology: Worsened by heat, sun, strenuous exercise, stress, alcohol, hot drinks, strong winds, cold and humidity. Over time the nose can thicken (often looks like an alcoholic's nose.

Recommendations: p. 88-89



Eczema

Inflammatory rash marked by itching and redness (acute or chronic). <u>Etiology</u>: allergies, food sensitivities/ intolerances, irritating chemicals or medications, depressed immunity, exposure to antibiotics *Atopic dermatitis*-found in individuals with a family history of eczema *Contact dermatitis*- skin contact with allergens or irritation substances Common Allergens: wheat, dairy, soy, chocolate, citrus, eggs and

peanuts

Nutrient deficiencies: zinc, selenium, Vit. A & essential fatty acids Signs and Symptoms:

Atopic-skin lesions that are reddened and cracked with thickened skin can become crusty from scrathing, *Contact*-blisters that can 'weep', redness and local edema with intense itching

Pathophysiology: elevated IgE, can start as early as 3 months of life, runs in families (80% chance), allergic contact dermatitis is caused by T-cell mediated hypersensitivity reaction to environmental allergens, either natural or synthetic. These combine with skin proteins altering the normal self-antigens so that new foreign antigens are created. Non allergic contact dermatitis is caused by exposure to detergent, soap or other skin irritant.

Recommendations: p. 90-91

Psoriasis

Eruption of reddish, silvery-scaled maculopapules. Etiology: incomplete protein digestion results in microbial putrefaction of amino acids with the formation of ptomaines like putrescine, spermidine and cadaverine (these inhibit the formation of Camp and upset the balance in cell turnover). Dysbiosis. impaired liver function, alcohol, excess consumption of saturated fats, nutritional factors (it may be associated with a deficiency of essential fatty acids) and stress. Signs and Symptoms: skin lesions that appear as circular red patches covered by thick, silvery dry scales

(condition waxes and wanes). <u>Pathophysiology:</u> upset balance of cell turnover (replication of cells is too fast for the top layer of the skin to shed normally. Increased metabolism of nucleoproteins loads the liver and kidneys. High uric acid levels can cause gout.

Recommendations: p. 93-94

Rickets

Deformation of the bones in children as he result of Vitamin D deficiency. Etiology: Deficiency of vitamin D or failure to form vitamin D. Signs and Symptoms: bones become deficient in calcium and therefore not strong enough to support the growth of the growing child/affects the long bones and typically causes knock knees, bow legs and deformaties of the rib cage/ growth is impeded and the teeth are soft/the ends of the long bones are often swollen, especially at the wrists and the costochondral junctions where rib bones and rib cartilage meet/pigeon chested/scoliosis. Pathophysiology: impaired calcium absorption

Recommendations: p.53-55

Osteomalacia

Adult form of rickets Etiology: Arises from not enough sun exposure, inadequate intake of vitamin D in the diet, gut problems that cause failure to absorb the vitamin (gallbladder removal, biliary obstruction, celiac disease), repeated pregnancy where the diet is inadequate (babies have leached the mother's calcium), drugs such as anti convulsants also affect calcium absorption, renal failure will also affect a person's ability to form active vitamin D

Signs and Symptoms: muscle weakness, waddling or penguin gait, vague muscular aches or rheumatism, X rays show linear areas of translucency that look like fractures, long bones become curved Pathophysiology: Fully formed bones disintegrate because essential nutrients are missing (spine, pelvis and legs (the weight bearing bones). Recommendations: p. 53 & 54

Osteoporosis

Osteoporosis is the loss of bone mass throughout the skeleton, predisposing individuals to fractures. <u>Etiology</u>: Insufficient intake of the nutrients required to build the bone matrix, insufficient gravitational pull on the skeleton, hormonal changes, excess acidity (minerals will be shunted away from the bone and used instead to neutralize an acidic terrain).

Signs and Symptoms: Can persist for years without symptoms , bones easily fracture, bone deformaties can occur over time resulting in kyphosis (dowager's hump), loss of height

Pathophysiology: bone matrix and minerals are substantially reduced, thinning and weakening of the bones (especially in the spine, wrists and hips)

Recommendations: p. 57-59

Fibromylagia

Refers to pain in fibrous tissue. Etiology: possibilities: virus, untreated trauma caused fibrous scarring around muscles or nerves, toxins and acidity from gut dysbiosis, hypothyroidism (same symptoms), chronic stress and chronic inflammation, unmyelinated nerves in deep tissues that send continual messages to the spinal cord irritating it.

Signs and Symptoms: widespread pain in 11-18 spots, muscular pain and malaise (worse with cold, damp & minor trauma) Pathophysiology: Fibrous tissue such

as myofascia over muscle is primarily affected and affects the nerves and muscle groups on which the fibrous tissue covers. The myofascia can become thickened and tightened, leading to chronic pain in the underlying muscle. Recommendations: p. 61-63



Chronic Fatigue Syndrome

Incapacitating fatigue that is not relieved by rest. Etiology: stress, poor nutrition, dysbiosis, liver overload, free radical damage (exposure to pollutants ie: smoke, drugs, cigarettes, heavy metals, dental mercury amalgams, persistent viral infections) Signs and Symptoms: severe chronic fatigue for 6 or more consecutive months not related to ongoing exertions or other conditions, fatigue significantly interferes with daily activities and work, 4 or more of the following 8 symptoms: post-exertion malaise lasting more than 24hours/ unrefreshing sleep/significant impairment of short term memory or concentration/muscle pain, multi-joint pain without redness or swelling, headaches of a new type, pattern or severity/tender cervical or axillary lymph nodes/ a sore throat that is frequent or reoccuring Pathophysiology: elevated IgG levels, decreased natural killer cells Recommendations: p. 65 & 66

Osteoarthritis

Osteoarthritis is a degenerative synovial joint disease. The inflammation is not nearly as pronounced as in rheumatoid arthritis and is secondary to the joint damage. <u>Etiology:</u> *Primary OA*: decreased ability to replace cartilage tissue either

because of decreased enzyme activity or decreased building blocks for the formation of cartilage *Secondary OA:* predisposition for joint destabalization (inherited

abnormalities in joint structure or function, prior trauma ie: sports injury, previous inflammation like rheumatoid arthritis or gout leading to excess or abnormal wear and tear Signs and Symptoms: early morning stiffness, stiffness after periods of rest, pain that worsens on joint use, loss of joint function, local tenderness, soft tissue swelling, creaking and cracking of joints on movement, restricted mobility Pathophysiology: affects weight bearing joints and joints of the hands, cartilage turnover is slowed down and as a result the cartilage has to last longer and is more susceptible to wear and tear/ as the cartilage ages it becomes dry, brittle and the synovial fluid less thick reducing their cushioning of the joints/ cartilage becomes softened and loses its elasticity, surface becomes worn in spots (as it thins out it can't hold as much synovial fluid), joint space narrows and cartilage becomes so thin that the bones begin to rub together, as the join becomes destabilized, bony spurs grow at the joint margins to try and stabilize the joint causing inflammation, pain, deformity and a diminished range of motion. Recommendations: p. 68-71

Gout

A form of arthritis marked by urate crystals in the joints and other tissues. 95% of cases occur in men. Etiology:

Primary gout- 90% of all cases, genetic defects which increase uric acid synthesis, or reduced ability to excrete uric acid

Secondary gout- uric acid synthesis, or reduced ability to excrete uric acid due to excessive cellular breakdown or turnover as occurs in cancer, chronic hemolytic anemia or the use of cytotoxic drugs, excess sugar consumption/psoriasis, kidney disease, diuretic therapy, low dose aspirin, lactic acidosis or ketoacidosis. Lead toxicity can cause a specific type of gout called saturnine.

Signs and Symptoms: acute onset of intense joint pain (especially in the first joint of the big toe (intermittent symptoms), high blood uric acid, uric acid crystals are detected in subcutaneous tissue, bone, cartilage, joint, fluid and other tissues, uric acid kidney stones/an attack is usually precipitated by eating excess purine (high in meat and organ meats) over eating, fatigue, stress, infection and the administration of penicillin or insulin.

Pathophysiology: deposition of uric acid crystals in joints, tendons, kidneys and other tissues causing inflammation and damage. Associated with bone calcium loss because it is leached out of the bones by the high acidity of the tissues.

Recommendations: p. 73 & 74

Rheumatoid Arthritis

It is a systemic autoimmune disease that is especially common with women, affecting connective tissue accompanied by thickening of the articular soft tissues with extension of synovial tissue over articular cartilages which become erodedleads to deformities and disability. Etiology: possibilities: virus, dysbiosis, leaky gut, poor digestion, Signs and Symptoms: p. 76 Pathophysiology: autoimmunity that affects all connective tissue (cartilage, bone, ligaments, tendons, dermis, periosteum, synovial membranes and basement membranes). Great deal of chronic inflammation and destructive polyarthritis (inflammation of many joints), vasculitis and inflammation of the heart, spleen and lungs, drop in RBC's and WBC's and low grade fever

Recommendations: p.78-80

Ligament & Tendon Damage

Tendonitis is inflammation of a tendon, often due to repetitive strain injury (golfer's elbow, tennis elbow, rotator cuff tendonitis, pitcher's shoulder and swimmer's shoulder) Tenosynovitis is inflammation of the tendon sheath which becomes distended with fluid (commonly affects flexor and extensor tendons of the fingers) Ligament strain is over stretching of the ligament without tearing Ligament sprain is a partial rupture or tearing of the ligament, sometimes with some muscle damage. Etiology: Tendonitis and tensynovitis are commonly due to repetitive strain (overuse) of the affected muscles and tendons, while ligament strain and sprains are mostly due to traumatic injury Signs and Symptoms: pain over the area, usually worse with use. Pathophysiology: weakness leading to inflammation, pulls and tears Recommendations: p.82 &83