Beebe Healthcare
Margaret H. Rollins School of Nursing
N101 – Foundations of Nursing

	OA	RA
Patho	Gradual loss of articular cartilage with formation of boney outgrowths at the joint margins. Can be cause by a known event or condition that directly damages cartilage or causes join instability. Other causes can include decreased estrogen at menopause, obesity (contributes knee and hip), anterior cruciate ligament injury from quick stops in sports (increase risk for knee). Genetic, metabolic, and local factors interact to cause cartilage deterioration from damage at level on chondrocytes. The normally smooth white translucent articular cartilage becomes dull, yellow, and granular as OA progresses. Affected cartilage becomes soft and less elastic. The body attempts to repair the cartilage cannot keep up with destruction of OA. As the collagen structure in the cartilage changes, articular surfaces become cracked and worn. As the central cartilage becomes thinner, cartilage at the joint edges becomes thicker and osteophytes form. Joint surfaces then become uneven and affect the distribution of stress across the joint and cause reduced motion. Synovitis may occur when phagocytes try to rid the joint of small pieces of cartilage torn from the joint surfaces. These kinds of changes cause the early pain and stiffness, whereas later in the disease pain comes from bony joint surfaces rubbing	Likely results from combination of genetics and environmental triggers, but no know exact cause. Autoimmune cause is most widely accepted theory. This suggests that RA begins when a genetically susceptible person has initial immune response to an antigen. Said antigen, which is most likely not the same in all patients triggers formation of abnormal immunoglobulin G. autoantibodies are known as rheumatoid factor (RF). These combine with IG to form immune complexes. Complexes first deposit on synovial membranes or superficial articular cartilage in the joints which leads to completement activation and inflammatory response as they grow to a complex formation. Neutrophils are attracted to the site of inflammation then release proteolytic enzymes that damage articular cartilage and cause the synovial lining to thicken. Activated CD4 cells cause monocytes, macrophages, and synovial fibroblasts to secrete the proinflammatory cytokines interleukin-1 (IL-1), IL-6, and tumor necrosis factor. These drive the inflammatory response in RA.
Risk Factors	Obesity, tobacco use, drug use, skeletal deformities, trauma, joint instability, age, post menopausal, weight bearing, hormonal influences, minerals, low protein	Age, sex, family history, smoking, obesity, infections, periodontitis (advanced gum infection)
S&S	Pain, stiffness, swelling, tenderness, limited range	Joint pain, joint swelling, joint stiffness, fatigue, weight loss, fever,

	of motion, instability, grating sensation, bone spurs, joints appearing larger, muscle weakness	loss of appetite, deformities
Dx	x-ray, MRI, joint fluid analysis, bone scan, CT, CBC, uric acid, liver/renal function tests, ESR (erythrocyte sedimentation rate; for levels of inflammation)	CBC, ESR, CRP, rheumatoid factor, xrays, synovial fluid analysis,
Tx	Drug therapy (NSAIDs, intraarticular corticosteroids) surgical therapy (hip/knee replacements)	Drug therapy (DMARDS, NSAIDs,) surgical therapy (synovectomy, arthroplasty)
NI	health promotion, ambulatory care, heat and cold applications, nutrition therapy, exercise, rest and joint protection	rest, joint protection, cold and heat therapy, exercise, nutrition