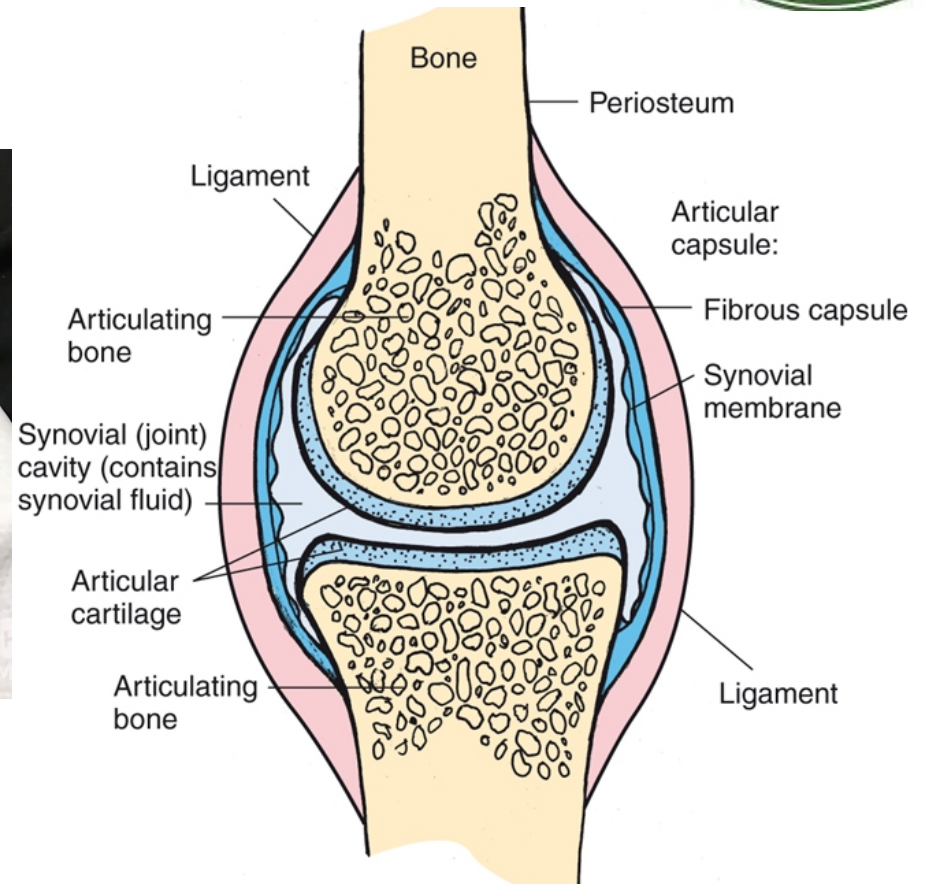
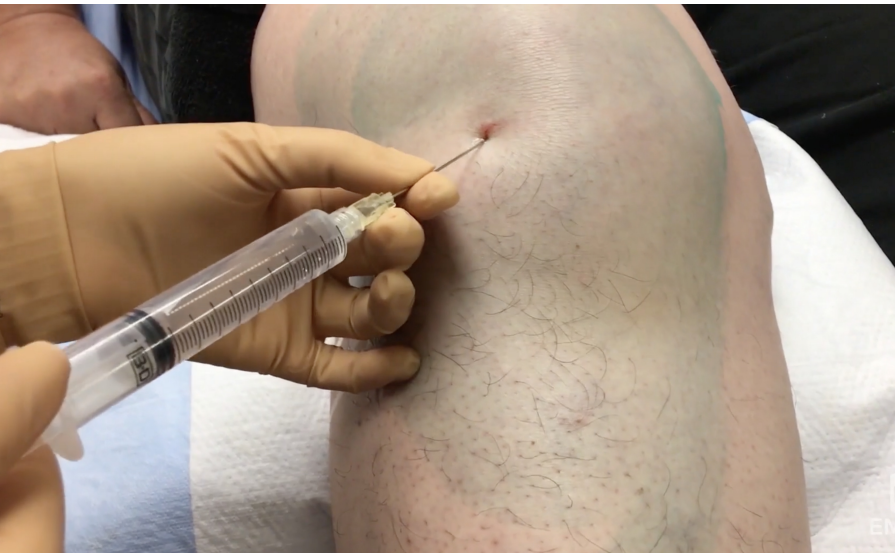


# Body Fluid Analysis-II

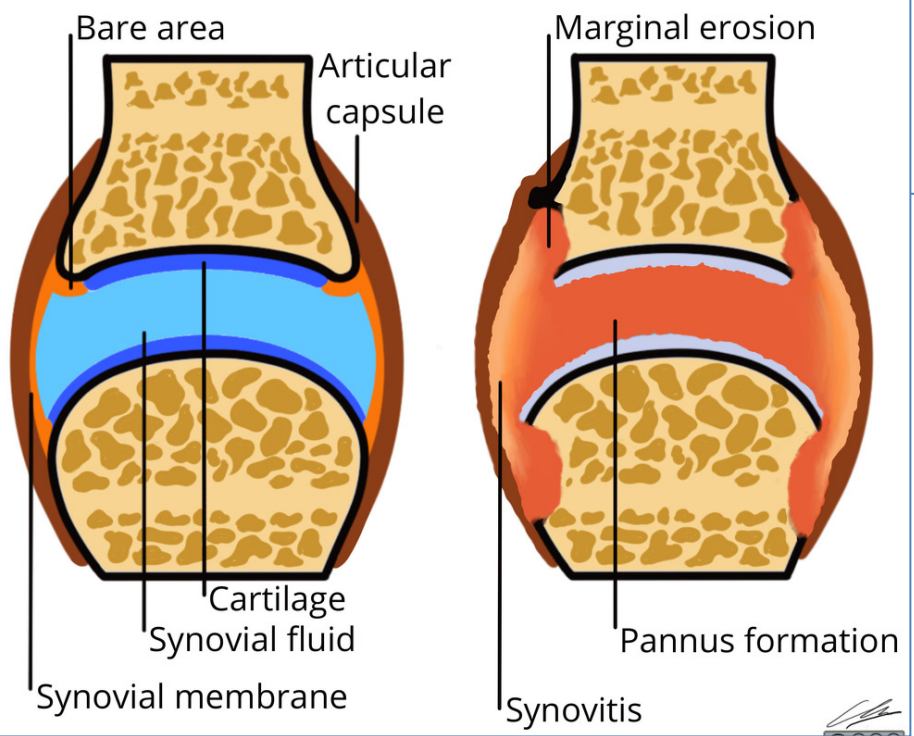
## Synovial Fluid



By

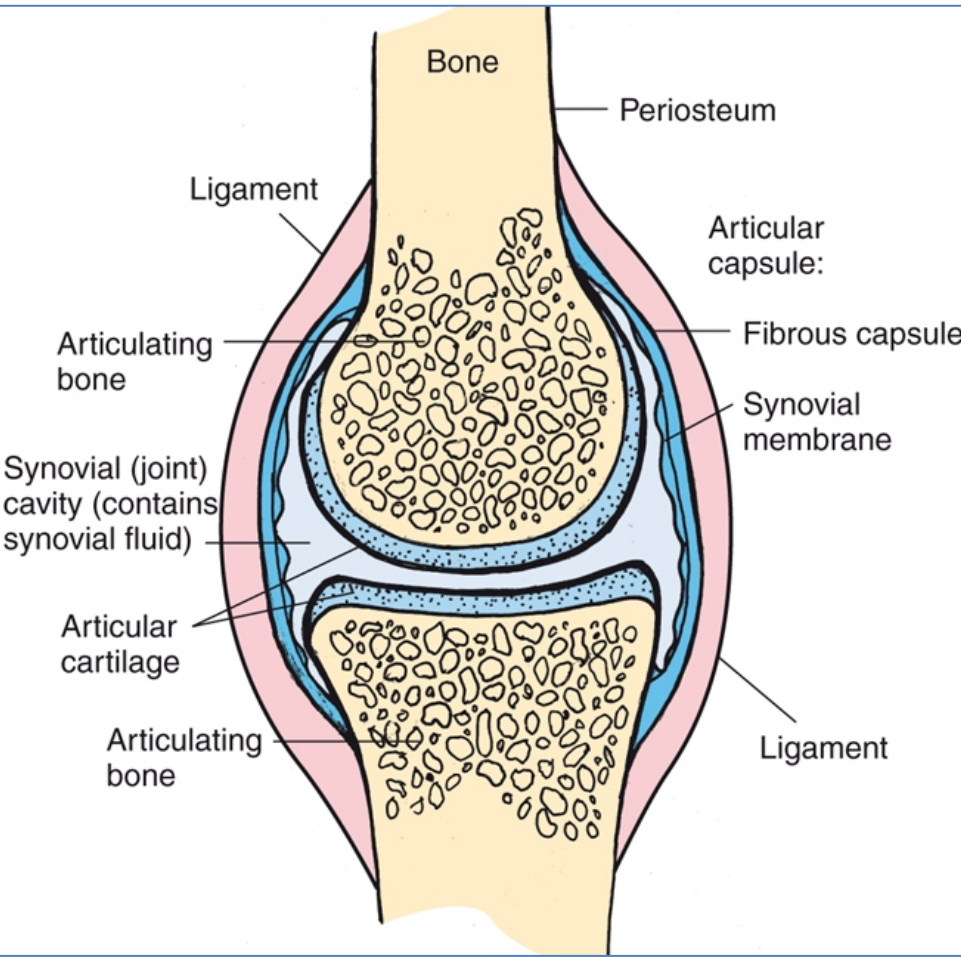
**Asst Prof Dr. Hastyar Hama Rashid Najmuldeen**

These “bare” areas refer to **bone within the synovial space which is not covered by articular cartilage.**



# Joint Anatomy

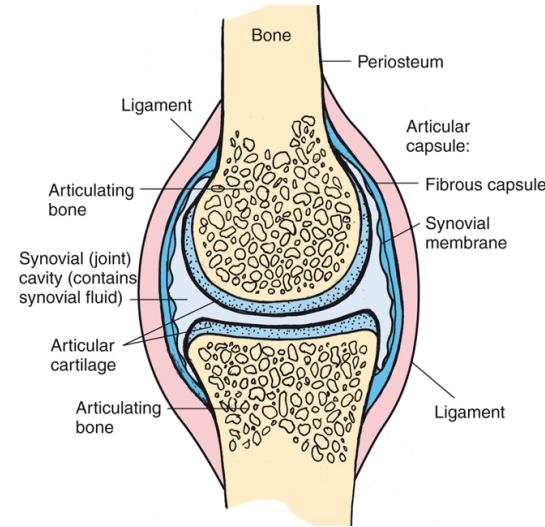
Articulated joint.



bare area of a joint where synovium is in direct contact with bone, the site of marginal joint erosion in some inflammatory arthritides

# Formation and Composition

- Synovial = syn (like) + ovia (egg).
- Secreted by cells of synovial membrane
- Very viscous, clear ultrafiltrate of plasma
- Contains:
  1. Protein lower than plasma
  2. Glucose & uric acid levels equivalent to plasma.
  3. Hyaluronic acid
  4. Muco-polysaccharides (Moistens and lubricates joints).



- [https://www.youtube.com/watch?v=0\\_fxPCQYUiA](https://www.youtube.com/watch?v=0_fxPCQYUiA)
- <https://www.youtube.com/watch?v=qf0NsTkUhKI>

- **Functions of synovial fluid:**

1. Supplies nutrients
2. Lubrication of joint: Mucinous substance that lubricates most joints to reduce friction between the articular cartilage of synovial joints during movement

**SYSTEMIC LUPUS  
ERYTHEMATOSUS (SLE)**

- Chronic, multi-system inflammatory disease with protean manifestations and remitting course
- **Clinical manifestations**
  - Musculoskeletal (joint and muscle pain)
  - Dermatological (malar rash)
  - Renal (glomerulonephritis)
- **Female to male ratio of 9:1**
- **Etiology is unknown**
  - Genetics, race, hormones, environment



**Reasons for S.F analysis:**

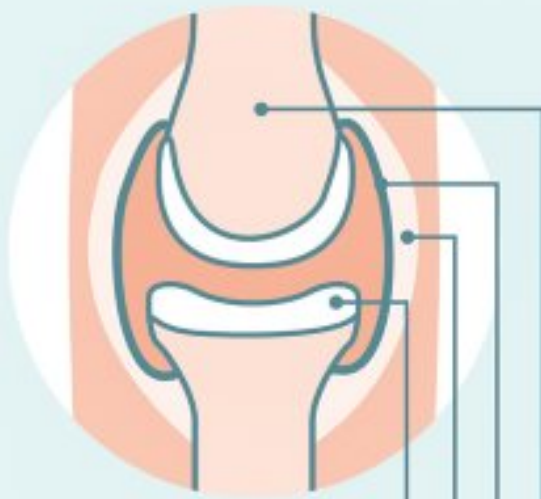
1. Infection (RA)
2. Hemorrhage (Trauma)
3. Degenerative disorders (**arthritis**)
4. Inflammatory disease **Systemic lupus erythematosus (SLE)**

# Classification of Joint Disorders

1. Normal
2. Non-Inflammatory
  - Degenerative joint diseases **Osteoarthritis**
3. Inflammatory
  - Immunologic disorders ( Lupus, Rheumatoid arthritis , gout crystals)
4. Septic
  - Microbial infections
5. Hemorrhagic
  - Traumatic injury, tumors, hemophilia, anticoagulant overdose, etc.

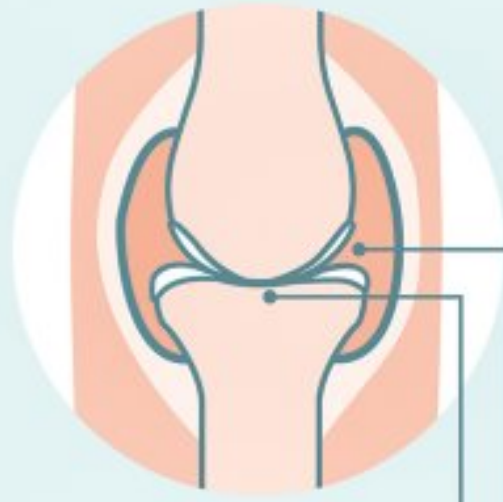
**Rheumatoid arthritis** is an autoimmune disorder that produces inflammatory joint symptoms throughout the body. **Osteoarthritis** is a degenerative condition that is the result of increased wear and tear on joints.

NORMAL JOINT



CARTILAGE  
JOINT CAPSULE  
SYNOVIAL MEMBRANE  
BONE

OSTEOARTHRITIS



BONE ENDS  
RUB TOGETHER  
THINNED CARTILAGE

RHEUMATOID  
ARTHRITIS



SWOLLEN INFLAMMED  
SYNOVIAL MEMBRANE  
BONE EROSION

**Rheumatoid arthritis (RA)** is a chronic systemic autoimmune disease that occurs more frequently in females than males, and it also predominantly observed in the elderly.

- Early diagnosis remains challenging as it relies mainly on the **clinical information** from the patient's history and **physical examination** supported by **blood tests**, and **imaging analysis**.
- There are two major subtypes of RA according to the presence or absence of **anti-citrullinated protein antibodies (ACPAs)**.
- **ACPAs** can be detected in approximately 67% of RA patients and serve as a useful diagnostic reference for patients with **early, undifferentiated arthritis**.

**RA** can be triggered in the potential trigger sites (lung, oral, gut) by the interaction between the genes and environmental factors, which is characterized by the onset of **self-protein citrullination** resulting in the production of **autoantibodies** against **citrullinated peptides**.

1. Lung exposure to noxious agents, infectious agents (*Porphyromonas gingivalis*, and Epstein-Barr virus)
2. Gut microbiome, and dietary factors may induce the **self-protein citrullination** and **maturation of ACPA**.

**Citrullination:** is catalyzed by the calcium-dependent enzyme PAD (**peptidyl-arginine- deiminase**), changing a positively charged arginine to a polar but neutral citrulline as the result of a post-translational modification.

In RA, PAD can be secreted by the **granulocyte** and **macrophage**.



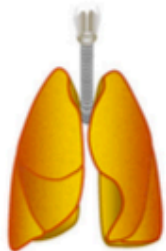
## Gene factors



Susceptibility genes

Epigenetic modifications

Post-translational modifications



Noxious agents



Periodontitis



Gut microbiome  
dietary factors

## Potential trigger sites and environment factors

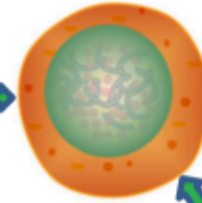
Granulocyte



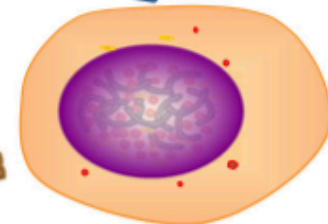
Macrophage



T cell



B cell



PAD

ACPA

Target protein

PAD+Ca<sup>2+</sup>

Citrullinated protein

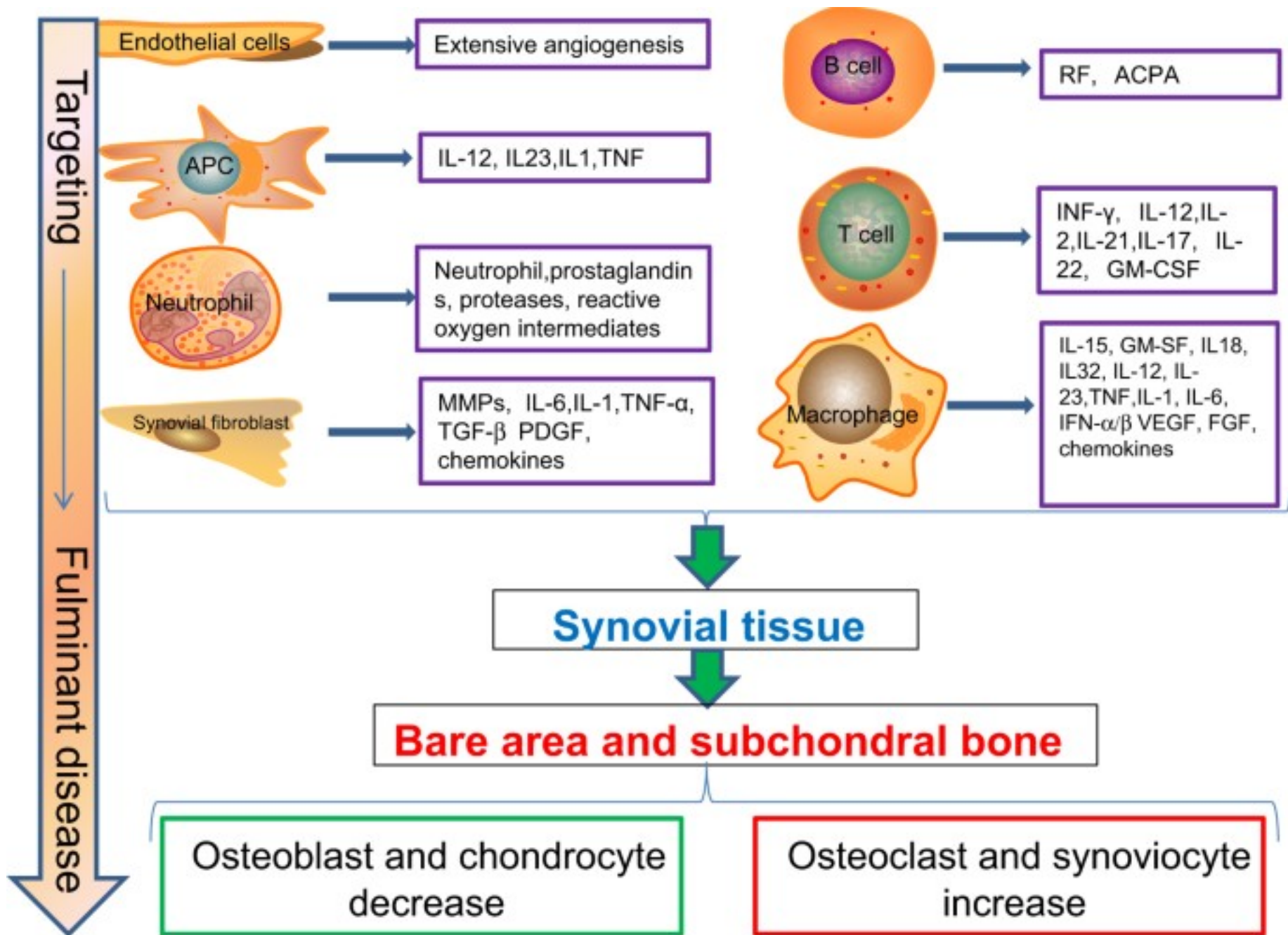
RF

Fibrin, vimentin, fibronectin, Epstein-Barr nuclear antigen 1,  $\alpha$ -enolase, collagen type I, collagen type II, histones, osteoclast precursor

## Self-protein citrullination

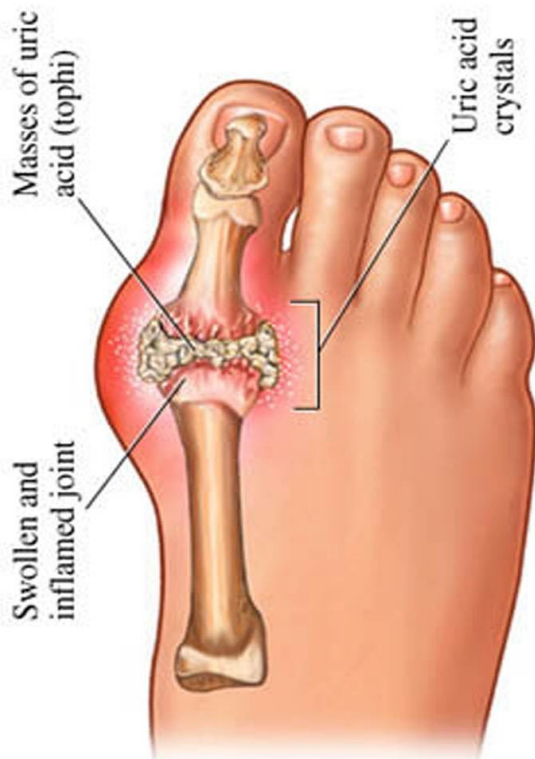
## Maturation

RA rheumatoid arthritis, PAD peptidyl-arginine- deiminase, ACPA anti-citrullinated protein antibodies, RF rheumatoid factor. ACPA occurs as a result of an abnormal antibody response to a range of citrullinated proteins, including fibrin, vimentin, fibronectin, Epstein-Barr Nuclear Antigen 1,  $\alpha$ -enolase, type II collagen, and histones, all of which are distributed throughout the whole body.



# What is gout diseases? and causes of gout?

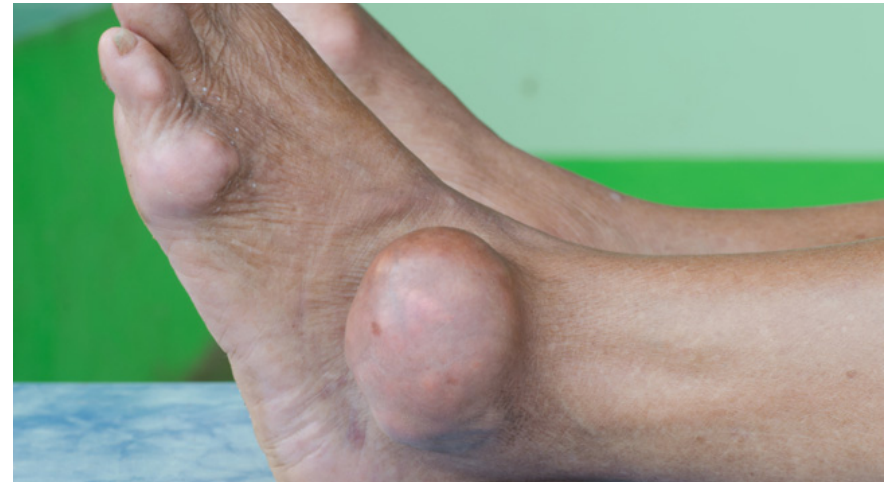
- Gout is caused by a build-up of a substance called **uric acid** in the blood.
- **Uric acid** is produced in the body during the breakdown of **purines** - chemical compounds that are found in high amounts in Alcoholic beverages and certain foods such as meat, poultry, and seafood.



## What are the factors that increase your chances of getting gout include:

1. Having a close relative with gout
2. kidney problems: **insufficiency of kidney function** to filter enough out of uric acid.
3. Eating foods that cause a build-up of uric acid, such as red meat, offal and seafood
4. Drinking too much beer or spirits

If **too much uric acid produced** or, it can build up and cause tiny sharp crystals to form in and around joints. These crystals can cause the joint to become inflamed (red and swollen) and painful.



## Causes of Infectious Arthritis

### Organism

### Clinical clues

Staphylococcus aureus

Healthy adults, skin breakdown, previously damaged joint (eg, rheumatoid arthritis), prosthetic joint

Streptococcal species

Healthy adults, splenic dysfunction

Neisseria gonorrhoea

Healthy adults (particularly young, sexually active), associated tenosynovitis, vesicular pustules, late complement deficiency, negative synovial fluid culture and gram stain

Aerobic gram negative bacteria

Immune compromised hosts, gastrointestinal infection

Anaerobic gram negative bacteria

Immune compromised hosts, gastrointestinal infection

Mycobacterial species

Immune compromised host, recent travel to or residence in an endemic area

Fungal species (sporotrichosis, cryptococcus, blastomycosis)

Immune compromised hosts

Spirochete (*Borrelia burgdorferi*)

Exposure to ticks, antecedent rash, knee joint involvement

Mycoplasma hominis

Immune compromised hosts with prior gastrointestinal tract manipulation