- **List specific functions of each type of GAGs:**
- **Heparin Proteoglycan**
  - Anticoagulant: Prevents thrombus (clot) formation by activating antithrombin.
  - Binds to lipoprotein lipase, increasing its release from capillary walls to plasma, aiding in blood lipid clearance.
- **Keratan Sulfate Proteoglycan:** Essential for the transparency of the cornea.
- **Heparan Sulfate Proteoglycans**
  - Primarily associated with the plasma membrane of cells.
  - Play an important role in cell membrane receptors and cell-cell interactions.
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- **Enumerate general functions of GAGs.**

1.Structural Role in ECM: They are important constituents of the extracellular matrix (ECM).

2.Hydrated Gel Formation

The negatively charged carboxylate and sulfate groups on the proteoglycan bind positively charged ions and form hydrogen bonds with trapped water molecules, creating a hydrated gel. This gel:

- ✓ Provides flexible mechanical support for the ECM.
- ✓ Acts as a filter that allows the diffusion of ions (e.g., Ca^{2+}), H_2O, and other small molecules while slowing the diffusion of proteins and cell movement.
- ✓ Functions as a lubricant, especially in synovial fluid.
- ✓ Is compressible: when a GAG solution is compressed, water is squeezed out, reducing its volume. When the compression is released, the molecules regain their hydrated size, giving GAGs shock-absorbing properties in joints and maintaining eyeball resilience.

3. How can you diagnose diabetes mellitus clinically (signs and symptoms)?

Diagnosis of Diabetes Mellitus

Symptoms

1. Polyuria (frequent urination)
2. Polydipsia (excessive thirst)
3. Polyphagia (excessive hunger)

- Often triggered by physiologic stress, such as an infection.
- Usually accompanied by fatigue and weight loss.

4. Enumerate laboratory diagnostic tests of diabetes mellitus.

Laboratory Diagnosis

1. Glucosuria

- Occurs when blood glucose >180 mg/dL.
- Impaired renal sodium-dependent glucose transporters (SGLT) lead to glucose "spilling" into the urine.
- Accompanied by water loss, resulting in polyuria and dehydration-induced polydipsia.

2. Hyperglycemia

- Diagnosis is confirmed by:
  - **Fasting blood glucose (FBG) ≥126 mg/dL** (normal: 70–99 mg/dL).
  - **Impaired glucose tolerance (pre-diabetes):** FBG of 100–125 mg/dL.
  - **Random or 2-hour postprandial blood glucose ≥200 mg/dL** in symptomatic individuals.
  - **Glycated hemoglobin (HbA1c) ≥6.5%** (normal: <5.7%).

Characteristics	Type 1 Diabetes	Type 2 Diabetes
AGE OF ONSET	Usually during childhood or puberty; symptoms develop rapidly	Frequently after age 35 years; symptoms develop gradually
NUTRITIONAL STATUS AT TIME OF DISEASE ONSET	Frequently undernourished	Obesity usually present
PREVALENCE	<10% of diagnosed diabetics	>90% of diagnosed diabetics
GENETIC PREDISPOSITION	Moderate	Very strong
DEFECT OR DEFICIENCY	β-Cell destruction, eliminating production of insulin	Insulin resistance combined with inability of β cells to produce appropriate quantities of insulin
FREQUENCY OF KETOSIS	Common	Rare
PLASMA INSULIN	Low to absent	High early in disease; low to absent in disease of long duration
ACUTE COMPLICATIONS	Ketoacidosis	Hyperosmolar hyperglycemic state
RESPONSE TO ORAL HYPOGLYCEMIC DRUGS	Unresponsive	Responsive
TREATMENT	Insulin always necessary	Diet, exercise, oral hypoglycemic drugs, insulin (may or may not be necessary); reduction of risk factors (weight reduction, smoking cessation, blood pressure control, treatment of dyslipidemia) essential to therapy