BIOCHEM #2: SURFACTANT.

- RDS → ↑ ST @ Term. Resp. units
  - ↳ Atelectasis & V-Q Mismatch.

- **Surfactants** (Amphiphilic)

Polar Head (Hydrophilic)

- charged (cationic or Anionic)
- dipolar (zwitterionic)
- Non-charged (nonionic)

Non-polar tail (Hydrophobic)

- usually: long chain hydrocarbon
- less usually:
  - Halogenated or Oxygenated Hydrocarbon or Siloxane chain
  - (Fatty Acid or Fatty Alcohol)

- e.g: SDS (Sodium dodecyl sulfate)
  DTAB (Dodecyl Trimethyl Ammonium Bromide)
  usually:
  - Ethylene Oxide
  - Dioctanoyl phosphatidyl Choline (DOPC)
  - Polyhydric Alcohol

- Surfactants Must have both Moieties to be "Surface Active"

- Classification of surfactant:

Anionic	SDS Sodium Dodecyl Sulfate
Cationic	CPB cetylpyridinium Bromide
Amphoteric	DPPC / Lecithin dipalmitoyl phosphatidyl Choline
Nonionic	Polyoxyethylene 4- Lauryl ether

- f(x) of Surfactant
  1. ↓ ST
     - > ↑ compliance & ↓ work
  2. Stability of Alveoli
     - > ↓ forces that cause Atelectasis
     - > Assist parenchyma "interdependant" support.
  3. Prevent transudation to Alv
     - > ↓ surface Hydrostatic pr. effect
     - > Laplace (Prevent ST from drawing fluid in from cap.)
  4. Expansion of Lung @ Birth.

IRDS

- Causes:
  - developmental insufficiency of surfactant
  - Neonatal infection
  - genetic (problem w/ surfactant proteins development)
- 1% of Newborn infants
- Leading COD of preterm infants.
- More common in: Males
  Caucasians
  2nd twin (premature).

α1-Antitrypsin Deficiency (aka α-1-proteinase inhibitor)

- protects tissues from enzymes of inflam. cells.
  esp. Neutrophil elastase.
- reference range: 1.5-3.5 g/L (in Blood)
  (@ Acute inflam. → ↑ concentration)
- Deficiency: Neutrophil elastase → Lung damage → Emphysema
  α-1-Anti-Trypsin → Liver damage (bc trapped) → cirrhosis
- Heredity: Autosomal Recessive or Codominant. (rare)
  Mutations in SERPINA1 gene @ Chr. 14 long arm
- Structure: Serine protease inhibitor
  - 2° → Beta Sheets & Alpha Helices
  - their Mutation ⇒ Non-functional proteins ⇒ polymerise & Accumulate @ liver.
  - "Infantile Hepatic Cirrhosis"
- 25,000 ppl in UK have the genetic condition but Most remain healthy & few diagnosed.
- Mutation → Z gene → if Both parents are carriers → 25%
- Made Mainly in liver

COPD

- a leading COD, rising DR.
- Smoking ⇒ Emphysema:
  1) destruction of lung tissue → obstruction of air flow
  2) Inflam. & Irritation of airways → obstruction ∴ ∴ ∴
- tar → no protective APT → damage to lung tiss.

Cystic fibrosis

- Lung congestion
- Cf gene ID'ed in 1989 → 7q31.2 (CFTR)
- usually Dx'ed in childhood
- epidemiology:
  - 1/3300 White infants
  - 1/15300 Black " (White > Black > Asians)
  - rare in Asians
  - ♀ = ♀
- prevention: Not possible
  - ID all carriers: Not possible
  - Gene Therapy progress.
  - Not all CFTR Mut are dangerous.
- Inheritance: Autosomal Recessive
- @ Lungs: Mutation: Blocked channels & H_2O & Cl^- can't flow out of cells
  - ⇒ Build-up of thick mucous / Bac infection / widened airways. w/ Blood
- Sx: Oral signs → disorder of saliv. gl.
  - Swelling of lips
  - gingivitis
  - dryness.
  - Sinusitis / Nose polyps
  - Salty sweat
  - Blocked panc. duct → Malabsorption / gallstones
  - Malabsorption @ Intestines
  - Sexual complications

↳ **DX** : - Sweat test (Most imp) (NaCl in Sweat)
- IRT (Immunoreactive Trypsinogen Test)
(Trypsinogen in Blood) 2-3 days after birth
- Chemical Tests (gene)
- other tests assist DX:
Chest X-ray
lung f(x) test
sputum culture
Stool exam. → digestive abnormalities

↳ **Tx**: only symptomatic & prophylactic
postural drainage
chest percussion
inhaled saline / distilled water.