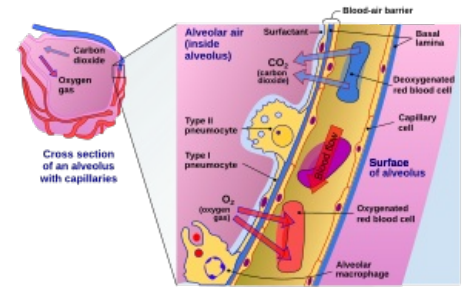
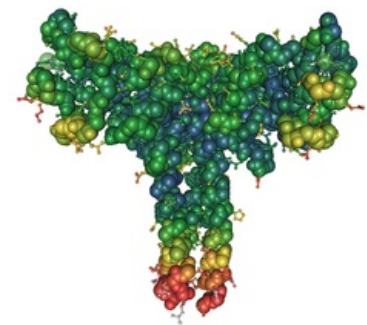


Surfactant

A surfactant is a surface-active substance that reduces the surface tension in the lungs. In the human body, surfactant is produced by **pneumocytes II. type**. It lowers the surface tension at the fluid-air interface in the alveoli, thereby increasing lung compliance. Surfactant consists of a mixture of phospholipids (e.g. lecithin dipalmitate), proteins and ions. Absence or insufficient secretion of surfactant leads to the collapse of alveoli with subsequent respiratory failure, e.g. danger for premature babies (surfactant production begins in the 6th-8th month of pregnancy).^[1]



Cross section of an alveolus and capillaries showing diffusion of gases



Lung Surfactant Protein D

- **SURFace ACTIVE Agent**
- **value:** reduces surface tension and promotes stability of alveoli during exhalation → prevents atelectasis, reduces work of breathing, prevents fluid transudation from capillaries into alveolar spaces;
- **composition:** phospholipids, neutral lipids and surfactant proteins A, B and C (SP-A, SP-B, SP-C) – hydrophilic and hydrophobic components;
 - phospholipids: mainly phosphatidylcholine (PC or lecithin, L), phosphatidylglycerol (PG) and to a lesser extent other phospholipids;
 - neutral lipids: cholesterol, triacylglycerols (TAG), free fatty acids (FFA);
 - sphingomyelin (its concentration does not change during pregnancy), glycolipids and carbohydrates form a very small fraction of surfactant;
 - SP-A is a large glycoprotein;
- **biosynthesis of surfactant** (phosphatidylcholine) takes place in the endoplasmic reticulum of pneumocytes II. type, then it is transported to the lamellar bodies and from there secreted into the alveoli;
- **surfactant recycling** takes place in alveoli – surfactant degradation in alveoli and small airways → surfactant components are absorbed by pneumocytes and recycled;
- **regulation of surfactant production:** stretch receptors, stimulation by the entry of gases into the lungs (distension of the alveoli); β-adrenergic receptors;
- pneumocytes I. and II. type can be detected histologically around the 22nd week of pregnancy;^[2] pneumocytes II. type makes up about 2% of the surface of the alveoli;
- in pneumocytes II. type, lamellar bodies gradually appear - intracellularly stored surfactant (around the 24th week of pregnancy);
- **factors affecting lung maturation:**
 - glucocorticoids – endogenous cortisol is an important physiological stimulus of fetal lung maturation;
 - beta-adrenergic drugs;
 - thyroid hormones – thyroxine increases surfactant production and lung maturation; thyroxine (T₄) does not cross the placenta, unlike TRH and triiodothyronine (T₃);
 - prolactin – unclear significance in the regulation of surfactant production; epidermal growth factor; fibroblast pneumocyte factor;
 - insulin – slows down the maturation of pneumocytes II. type and surfactant formation, inhibits SP-A gene expression;
 - testosterone – slows down lung maturation and surfactant production.^[3]

Links

Related articles

- Induction of fetal lung maturity
- Alveolo-capillary membrane
- Lung
- Lung (histological preparation)
- Fetal lungs
- Respiratory Distress Syndrome

References

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2. JANOTA, Jan – STRAŇÁK, Zbyněk. *Neonatologie*. 1. edition. Mladá fronta, 2013. pp. 90. ISBN 978-80-204-2994-0.

3. RENNIE, JM, et al. *Textbook of Neonatology*. 5. edition. Churchill Livingstone Elsevier, 2012. pp. 459-464. ISBN 978-0-7020-3479-4.