

Casual Friday Presents

Respiratory Function

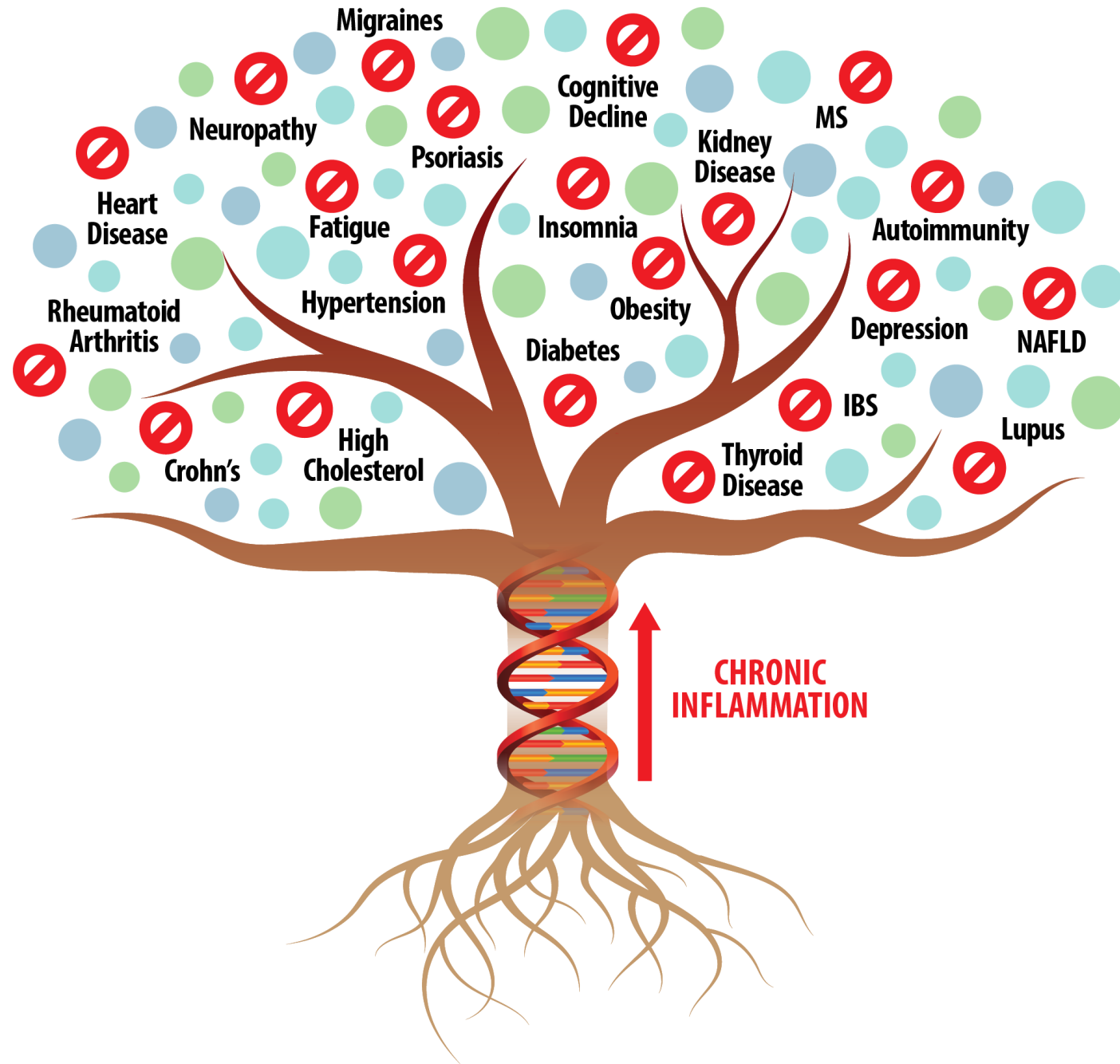
FM Strategies: Physiology, Pathology, Interventions.

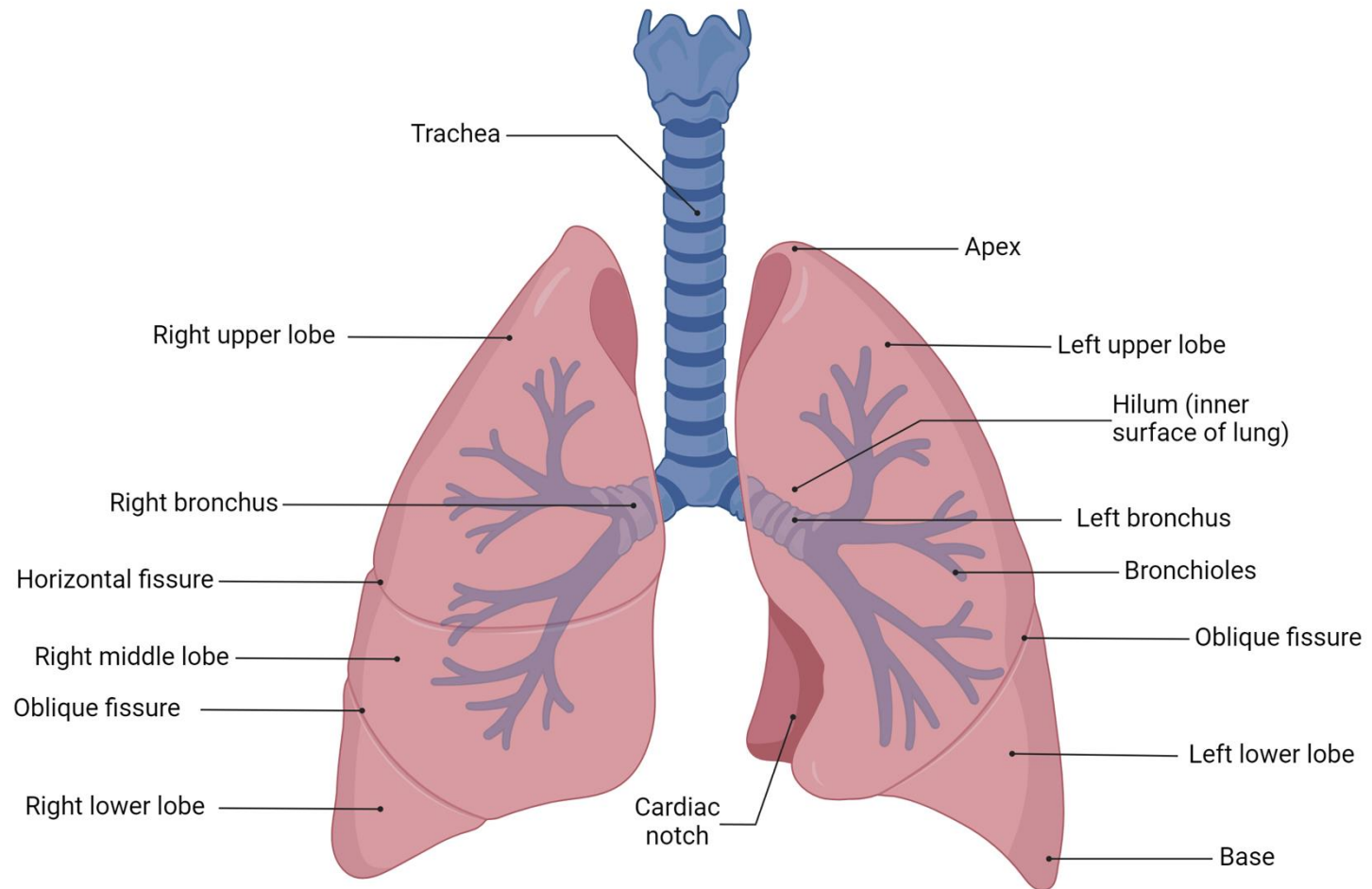
A BIOGENETIX CLINICAL PRESENTATION

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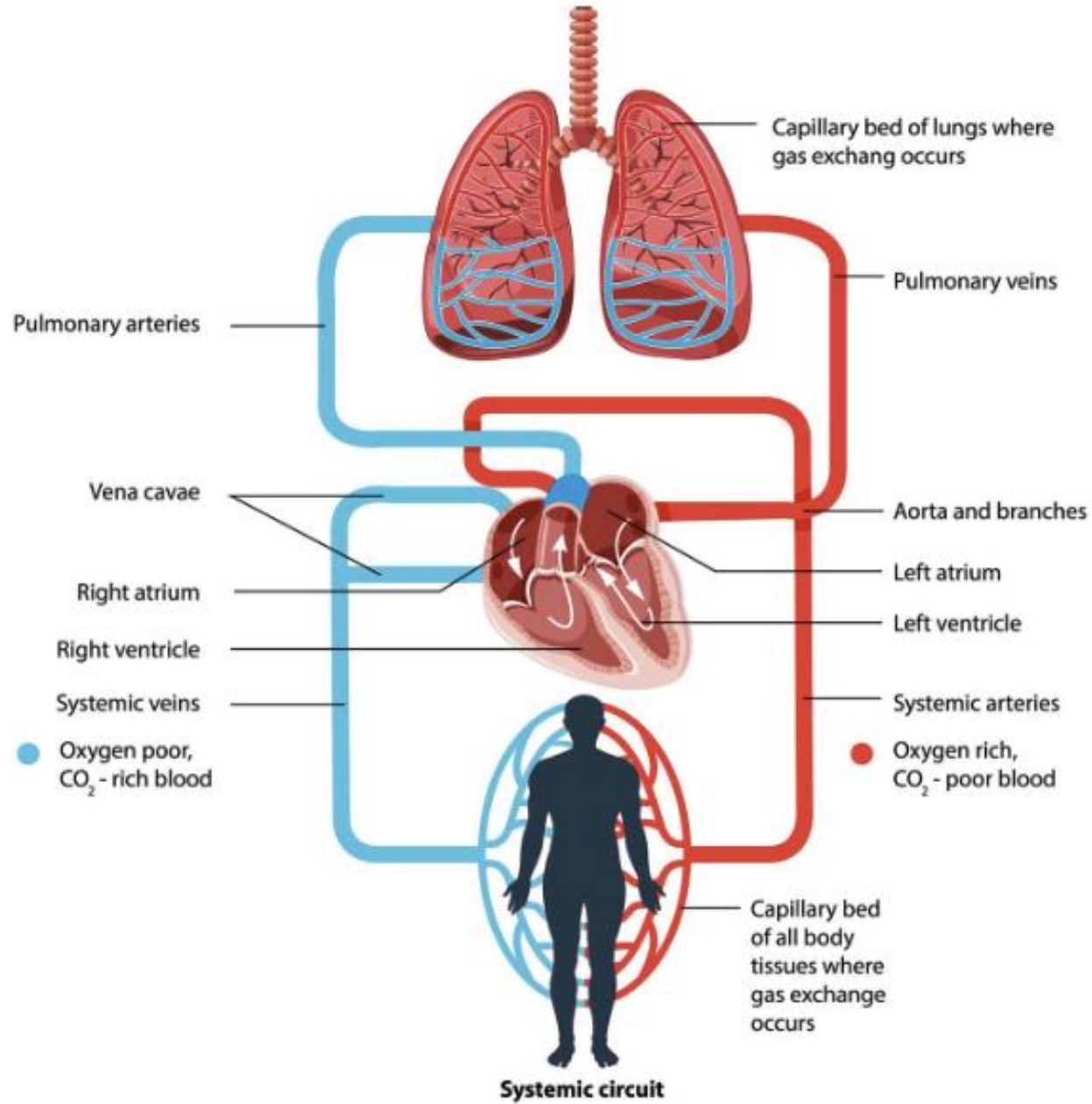


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PULMONARY CIRCUIT

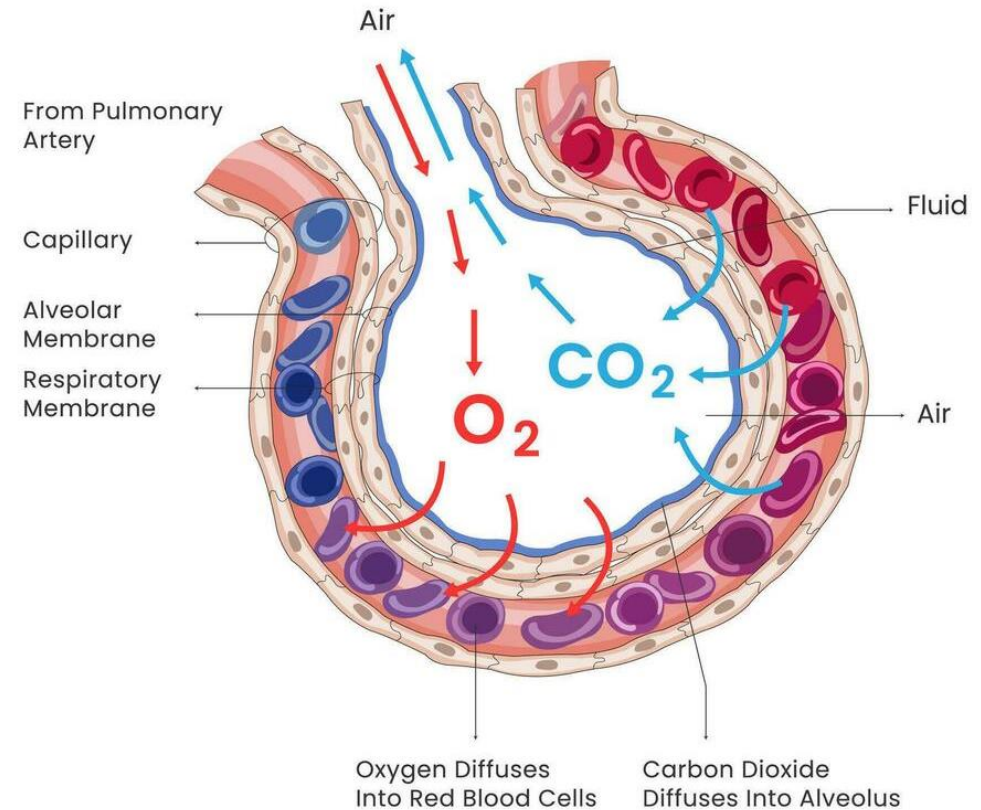


Lung Physiology and Functional Overview

Primary:

Gas Exchange

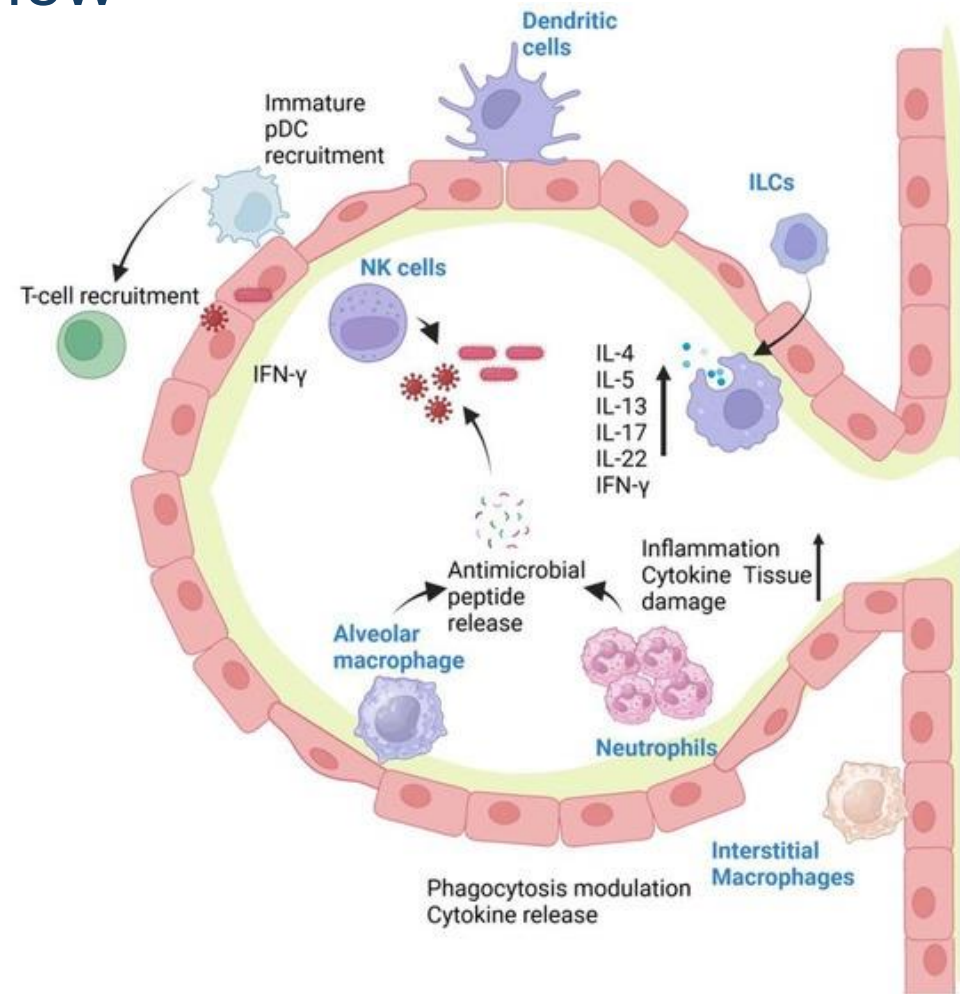
- Oxygen uptake
- Carbon dioxide elimination
- Acid-base regulation



Lung Physiology and Functional Overview

Immune Defense

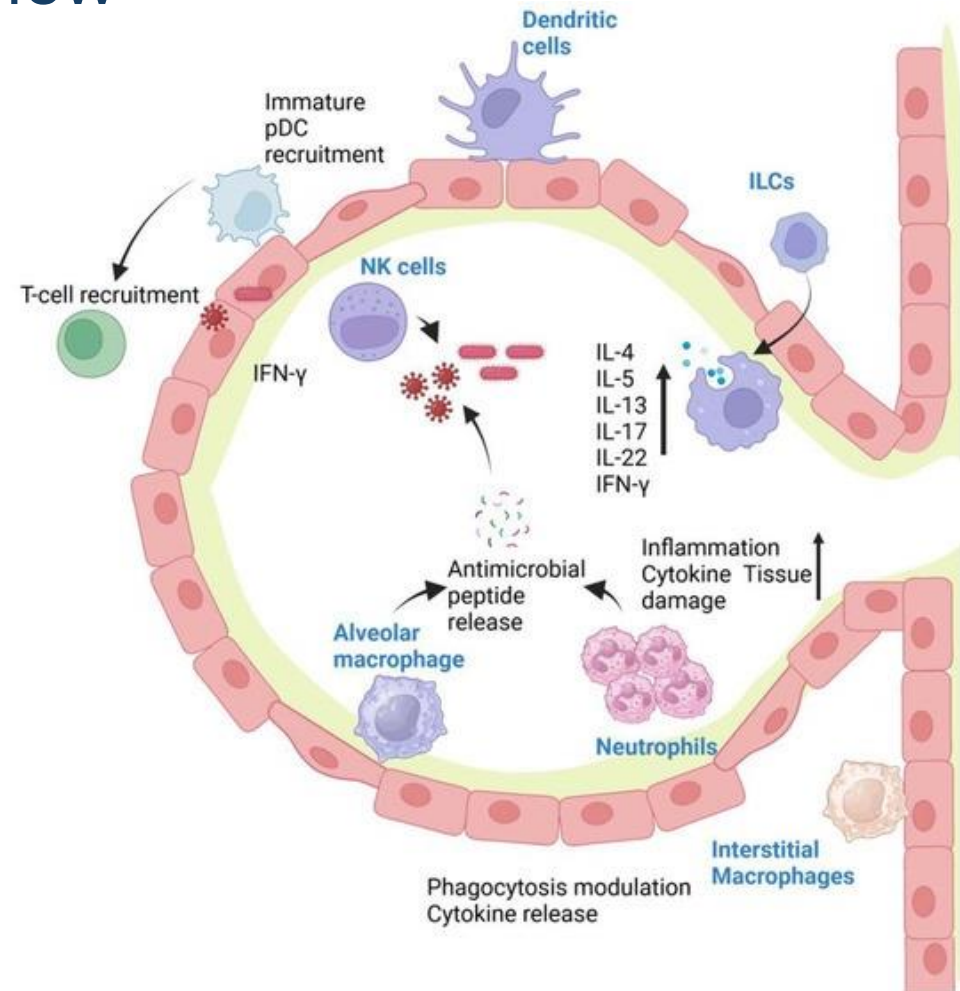
- Mucociliary clearance
- Secretory IgA
- Alveolar macrophages
- Lung microbiome



Lung Physiology and Functional Overview

Detoxification

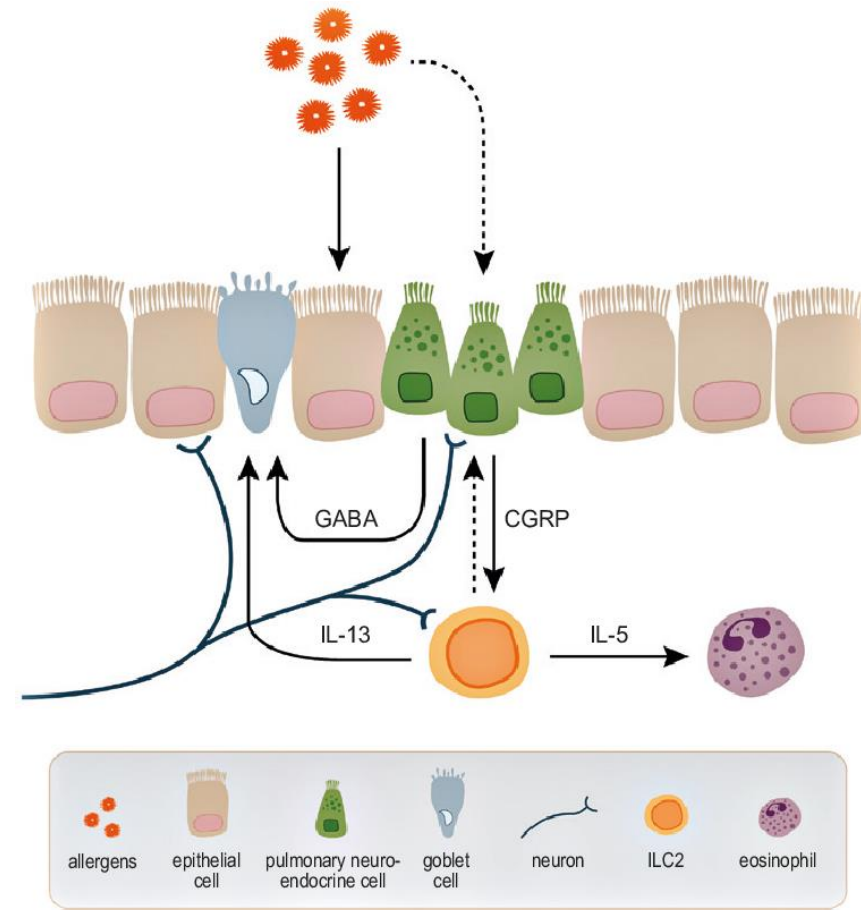
- VOC elimination
 - benzene
 - Ethanol
 - formaldehyde
- Xenobiotic metabolism
 - Drugs
 - Pesticides
 - Cigarette additives
- Air filtration



Lung Physiology and Functional Overview

Neuroendocrine Function

- ACE enzyme activity
- Nitric oxide production
- Stress response regulation



ACE Cycle

Angiotensin-converting enzyme (ACE) is located primarily on the surface of endothelial cells lining the pulmonary capillaries in the lungs.

Blood carrying angiotensin I (an inactive precursor) flows from the right side of the heart into the lung circulation.

As blood passes through the lung capillaries, ACE removes two amino acids from angiotensin I, converting it into angiotensin II.

Angiotensin II is a potent hormone that causes vasoconstriction (narrowing of blood vessels).

- Increases systemic blood pressure.
- Stimulates the adrenal cortex to release aldosterone.
- Aldosterone acts on the kidneys to increase sodium and water reabsorption.
- Increase blood volume.
- Further raise blood pressure.
- ACE also breaks down bradykinin, a molecule that normally: Causes vasodilation (widening of blood vessels).
- Increases vascular permeability.
- Converting angiotensin I to angiotensin II and degrading bradykinin mean ACE helps regulate blood pressure
- Blood volume
- Fluid and electrolyte balance

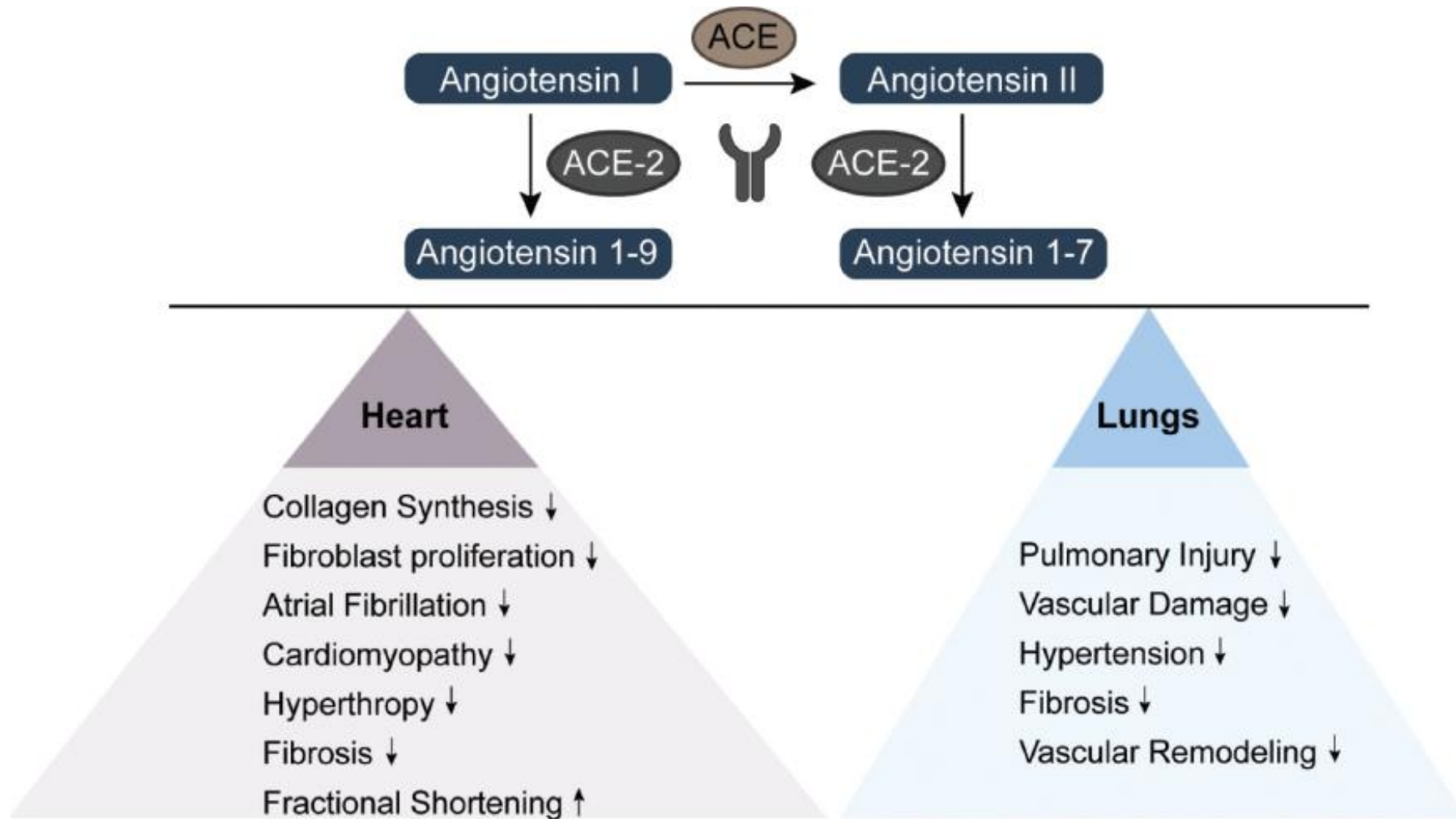


ACE Cycle

1. Liver releases angiotensinogen.
2. Kidney releases renin → converts angiotensinogen to angiotensin I.
3. Lungs (ACE) convert angiotensin I → angiotensin II.
4. Angiotensin II causes vasoconstriction and stimulates aldosterone release.
5. Blood pressure and blood volume increase.



ACE Cycle



Nitric Oxide Cycle

Nitric oxide (NO) is produced by cells lining the blood vessels and airways of the lungs, especially pulmonary endothelial cells. The amino acid L-arginine is converted into nitric oxide (NO) and L-citrulline by the enzyme nitric oxide synthase (NOS).

3 forms of NOS:

- eNOS (endothelial NOS) – primary source in pulmonary blood vessels.
- nNOS (neuronal NOS) – found in airway nerves.
- iNOS (inducible NOS) – produced during inflammation and immune responses.



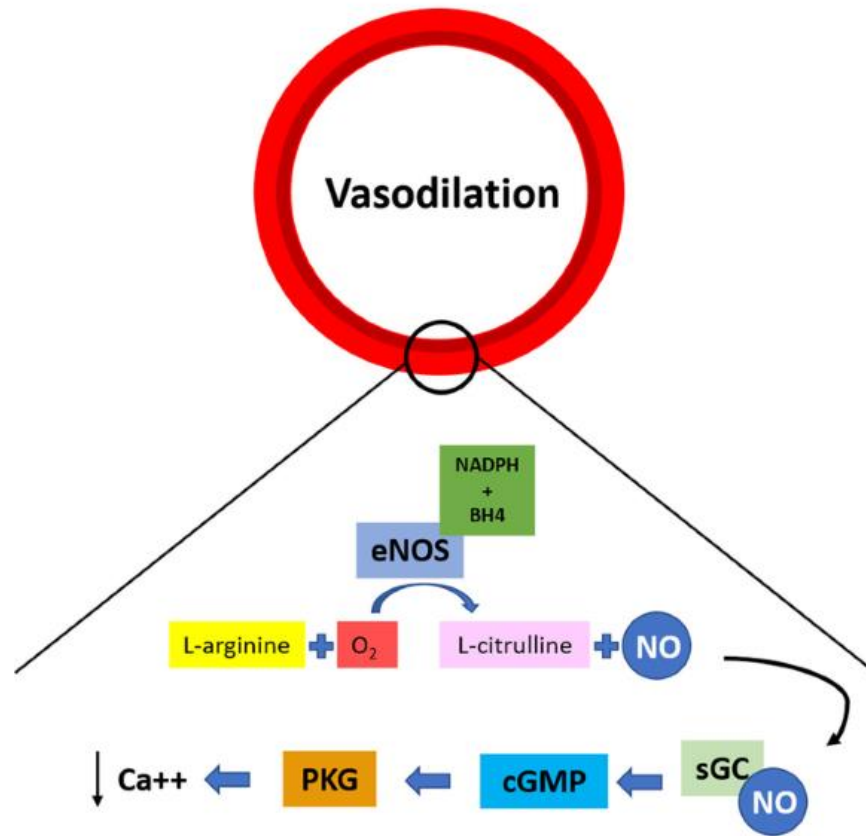
Newly formed NO diffuses rapidly from endothelial cells into nearby smooth muscle cells in the pulmonary arteries. Inside smooth muscle cells, NO activates soluble guanylate cyclase (sGC). sGC converts GTP into cyclic GMP (cGMP). Increased cGMP causes: Relaxation of smooth muscle.

- Vasodilation
- Reduced pulmonary vascular resistance.
- Pulmonary vasodilation improves: Blood flow through the lungs.
- Oxygen uptake into the bloodstream.
- NO can also Inhibit platelet aggregation.
- Reduces adhesion of inflammatory cells to blood vessel walls.
- Helps maintain healthy endothelial function.
- NO is rapidly inactivated when it binds to hemoglobin in red blood cells, limiting its effects mainly to the local pulmonary circulation.



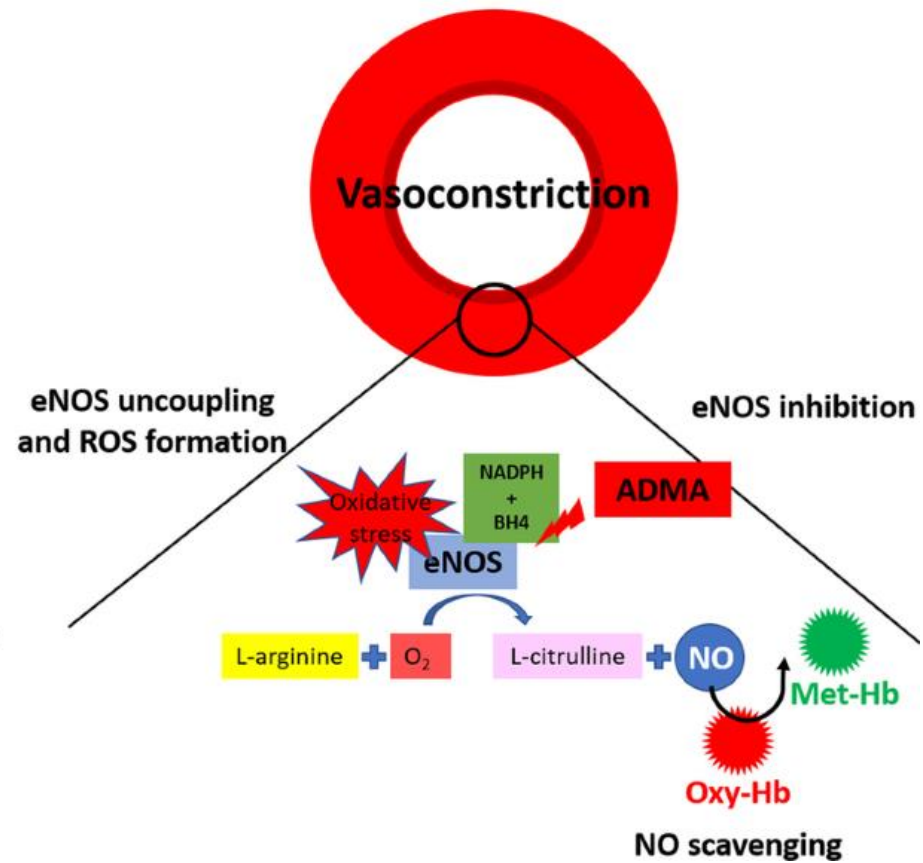
A

Healthy endothelium



B

Endothelial dysfunction

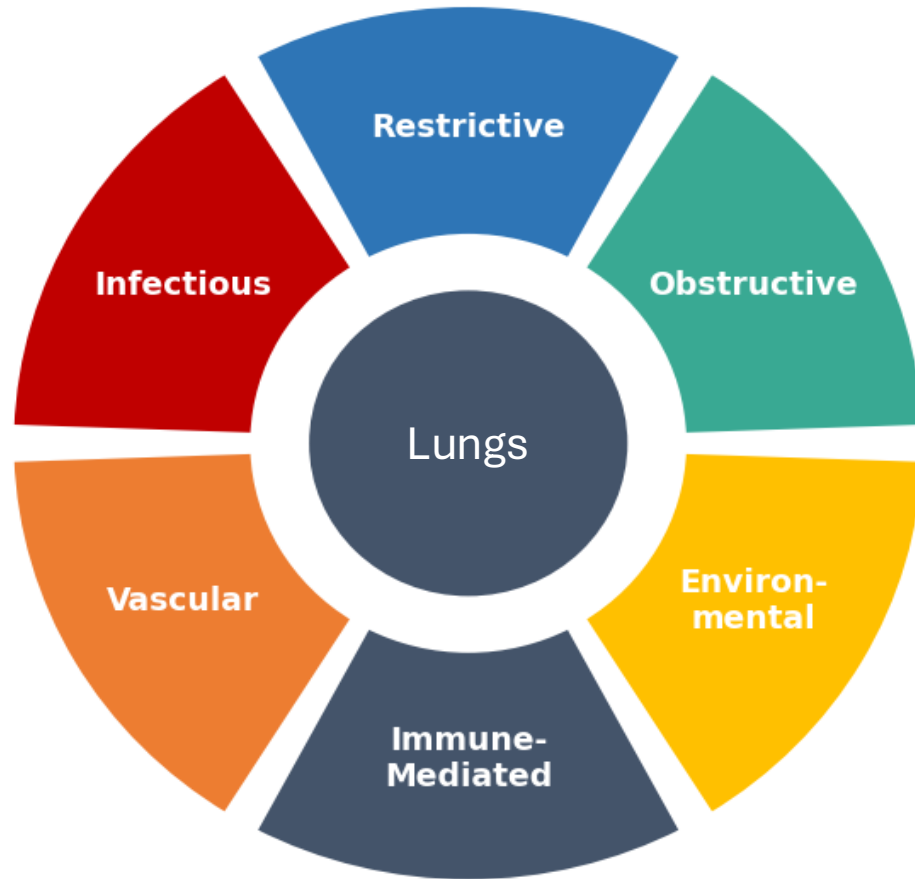


Nitric Oxide Cycle

1. eNOS produces NO from L-arginine.
2. NO should cause vasodilation via cGMP.
3. Some NO reacts with oxyhemoglobin (Oxy-Hb).
4. Oxy-Hb is converted to methemoglobin (Met-Hb).
5. NO is consumed/scavenged.
6. Less NO available → vasoconstriction and endothelial dysfunction.



The Big 6 Pathology Categories



1. Obstructive

Asthma, COPD, Bronchiectasis

2. Restrictive

Fibrosis, Sarcoidosis

3. Infectious

Viral/Bacterial Pneumonia, TB

4. Vascular

Pulm. Hypertension, Pulm. Edema

5. Immune-Med.

Eosinophilic, Autoimmune Pneumonitis

6. Environmental

Mold/CIRS, Occupational, VOCs

Obstructive Lung Disorders

Mechanism: Airflow limitation due to airway narrowing

Examples

- Asthma
- COPD / Emphysema
- Chronic bronchitis
- Bronchiectasis (widened and scarred)
- Alpha-1 antitrypsin def.

Symptoms

- Wheezing
- Shortness of breath
- Chest tightness
- Chronic cough
- Mucus hypersecretion

FM Drivers

- Th2 immune activation
- Mast cell / MCAS
- Environmental toxins
- Food sensitivities (dairy/gluten)
- Mold/CIRS exposure

Obstruction Disorder Diagnostic Measures

- **Spirometry:** The gold standard for OAD diagnosis. It measures how much and how fast air moves out of your lungs.
- **FEV₁/FVC Ratio:** The key diagnostic marker. An FEV₁ (Forced Expiratory Volume in 1 second) to FVC (Forced Vital Capacity) ratio below 0.70 after using a bronchodilator confirms airway obstruction.
- **Reversibility Testing:** Administering a short-acting bronchodilator during spirometry to see if airflow limitation improves; significant improvement points more toward asthma, while less reversibility is common in COPD.
- **Additional PFTs (Pulmonary Function Tests):** Tests like lung volumes (plethysmography) and diffusion capacity (DLCO) determine hyperinflation and rule out restrictive lung diseases.

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Toxins Summary



Vibrant Wellness | 3521 Leonard Ct, Santa Clara, CA 95054
1(866) 364-0963 | support@vibrant-america.com | www.vibrant-wellness.com

LAST NAME	FIRST NAME	GENDER	DATE OF BIRTH	ACCESSION ID	DATE OF SERVICE
[REDACTED]					

High (>95th percentile)						
Mycotoxins Heavy Metals Environmental Toxins						
TEST NAME	CURRENT RESULT	PREVIOUS RESULT	CURRENT RESULT	PREVIOUS RESULT	REFERENCE	
Diacetoxyscirpenol (DAS)	7.03		0 2.4 4.27		≤4.27 ng/g	
Gliotoxin	326.49		0 116 207		≤207.87 ng/g	
Ochratoxin A (OTA)	22.63		0 3.83 6.8		≤6.8 ng/g	
Roridin E	3.82		0 0.75 1.33		≤1.33 ng/g	
Sterigmatocystin (STC)	1.84		0 0.3 0.53		≤0.53 ng/g	
Zearalenone (ZEN)	0.92		0 0.38 0.67		≤0.67 ng/g	
Antimony*	3.53		0 0.07 0.16		≤0.16 ug/g	
Cesium*	10.63		0 6.37 10.3		≤10.3 ug/g	

* Indicates NHANES population data reference ranges.



We Want to Hear from You!

Give us your Casual Friday feedback
with this short 5-question survey.



biogenetix.com/survey