**B cell response**

Macrophage and helper T cell involvement with initiating a B cell response:

When specific B cells are activated, they multiply

Some cells become **memory cells**, stored in case of a subsequent infection

**Immunological memory from vaccines**

Vaccines introduce antigen (dead or weakened) to induce production of memory B & T cells, antibodies

Memory cells are activated on real exposure to bacteria, virus. Antibodies already present to label

**Vaccines and the quest to eliminate infectious disease**

Lung cancer vaccination
What about T cells?

T cells recognize **virus-infected** or **cancerous body cells** (cell-mediated).

When triggered, T cells with specific ‘self-antigen’ multiply. Killer T cells contact and release chemicals to kill the cells with the self-antigen marker.

What triggers T cells?

Macrophages “wear what they eat” (in this case, self marker plus antigen from pathogen, or self markers on cancerous cells).

Helper T cells are triggered and activate killer T cells and memory T cells.
How Killer T-cells kill body cells

Viral antigen is displayed on surface of host cell with self-antigen

Virus invaded host cell

Killer T cell recognizes and binds with a specific foreign antigen complex

Killer T cell

Host cell with virus

How Killer T-cells kill body cells

Killer T cell releases chemicals that destroy cell

How Killer T-cells kill body cells

Helper T cells

Helper T cells do not kill cells, but amplify effects of other WBCs:
- Enhance production of T and B cells, make chemotaxins for phagocytes
- “Master switch” for immune response
The **MHC** is a set of genes that code for glycoproteins on cell membranes and mark cells as “self.”

Matching MHC markers is important when transplanting organs.

**Combining non-specific and adaptive immune response**

**Bacterial infection:**
- At first: phagocytes, histamine release, inflammatory response
- Inflammation brings phagocytes, plasma proteins (complement system, clotting proteins)
- Bacteria antigen stimulates helper T cells, B cells get activated: antibodies
- Bacteria get labeled w/antibodies, killed by complement, macrophages, killer cells.

**Viral infection:**
- Virus inside body cells do not trigger macrophages, B-cells, or complement.
- Virus-infected or cancerous cells release interferon, signaling neighboring cells and attracting natural killer cells, macrophages, complement. Virus ‘out in the open’ can be attacked.
- Self-antigen combination triggers T-helpers, which help stimulate killer T cells (takes days) and attract macrophages to the area.

This slide is just another way to organize things for immune response to help study. I won’t use in lecture.
Why are there no better solutions to fight common colds and flu?

Blood groups

ABO blood types are named by antigens on the surface of RBCs: A, B, AB, or O (neither antigen).

People acquire antibodies for the blood antigens they do not have on their RBCs.

Blood type O: universal donor (no antigens).
Blood type AB: universal recipient (no antibodies)

Allergies: adaptive immunity gone wrong

Reactivity to a harmless substance in environment

Common triggers: pollen, molds, bee stings, dust, fur, mites, penicillin

Allergies: adaptive immunity gone wrong

- Hives - allergens on skin
- Hay fever - allergens in nasal passages
- Asthma - allergens in airway
Allergies involve a particular type of antibody – IgE antibodies

IgE antibodies trigger mast cells and basophils to produce histamine and other chemicals at the site of the allergens.

How does the immune system react differently for different allergies?

**Skin**: Besides IgE response, can be a T-cell response to substance (ex: urushiol oil)

**Airway**: besides histamine, leukotrienes are released – airway constricted

**Gut**: traditional food allergies are IgE (egg, milk, wheat, nuts, shellfish, etc.) Histamine dilation, leukotriene constriction. Some are T-cell allergies w/delayed effects (gluten, milk).

Anaphylactic shock

**Systemic anaphylaxis**: when large amounts of histamine and inflammatory signals are released all at once to blood.

- Widespread dilation - hypotension. Airway constriction. Victim can die within minutes. Often due to penicillin, bee venom.

Allergy medications

- They generally reduce the histamine signal
- They can be bronchodilators, reduce leukotrienes, decongestants (constrict capillaries), injectable epinephrine, anti IgE
- Corticosteroids inhibit expression of cytokines and other signals of inflammation
Hygiene hypothesis

Keeping a child’s environment ‘too clean’ may prevent proper development of immune system

**Recent study:** Exposing ‘high-risk’ infants to peanuts reduced later allergies by 80%

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What are autoimmune diseases?

Immune system wrongly attacks body cells, often caused by production of **autoantibodies**

- Rheumatoid arthritis – autoantibodies attach to joints and induce inflammation & attack by complement, and WBCs

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A few immune related disorders:

- Diabetes Type I
- Crohn’s Disease
- Multiple Sclerosis
- Pernicious anemia
- Addison’s disease
- Lupus

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Triggering of autoimmune disease

AI diseases often have an environmental trigger, those with certain genotypes can be more susceptible to it

A trigger can be an infection w/antigen that is molecularly similar to markers on body cells

Possible environmental agents: silica, mercury, nitrates in drinking water, groundwater pollutants, drugs, many other chemicals…
Respiratory system

External vs. cellular respiration

Airway: from nasal passages down to trachea, bronchioles and alveoli. The trachea and bronchi are reinforced with cartilage

Larynx (voicebox) has vocal cords

Bronchioles can dilate and constrict

Smooth muscle

bronchioles

Alveolus

Pulmonary capillaries

Respiratory system

chest cavity
Lung wall

Transmural pressure gradient
lungs will always expand to fill pleural cavity

Inspiration and expiration: how we change chest volume

What determines airflow?

\[ F = \frac{\Delta P}{R} \]

same equation as blood flow!

Major determinant of resistance is radius of bronchioles

Disease can increase resistance (asthma, bronchitis)

Increasing cavity volume, air enters

If lung pressure is less than atmospheric pressure, air enters the lungs.

[Diagram showing pressure difference across a container with varying volumes to illustrate airflow]
**Surface tension at alveoli**

*Surface tension* – whenever water layer meets air – water molecules are attracted to each other.

Surface tension along the lining of alveoli resists expansion of alveoli.

*Surfactant* reduces surface tension.

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**Gas exchange and partial pressure gradients**

Air is a mixture of gases. Nitrogen is 79% of air. Its partial pressure: $0.79 \times 760 = 600.4$

<table>
<thead>
<tr>
<th>Alveoli</th>
<th>Capillaries</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{O_2}$</td>
<td>100</td>
</tr>
<tr>
<td>$P_{CO_2}$</td>
<td>40</td>
</tr>
</tbody>
</table>

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**Hemoglobin saturation**

Most $O_2$ is carried by Hb - some is dissolved in plasma and determines partial pressure.

Hb saturation is high where $P_{O_2}$ is high (lungs).

Saturation remains high even $P_{O_2}$ is 60.

Small decrease in $P_{O_2}$ makes Hb unload much more $O_2$.

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**Shifting the curve**

Increase in $CO_2$ from tissue shifts the saturation curve to the right.

Increased acidity ($H^+$, carbonic acid) and temperature has the same effect - Bohr effect.
Most $O_2$ carried on hemoglobin

A teensy bit of $O_2$ is dissolved in plasma

Chemoreceptors sense $O_2$ dissolved in blood and signal to brain stem

$P_{O2}$ and $P_{CO2}$ and $H^+$ can be detected in plasma

But $O_2$ saturation is not detected

Hb has higher affinity for CO and will displace $O_2$...but this will go undetected @ chemoreceptors

Spirometry

Measures airflow and volume of inspiration and expiration