Blood Typing

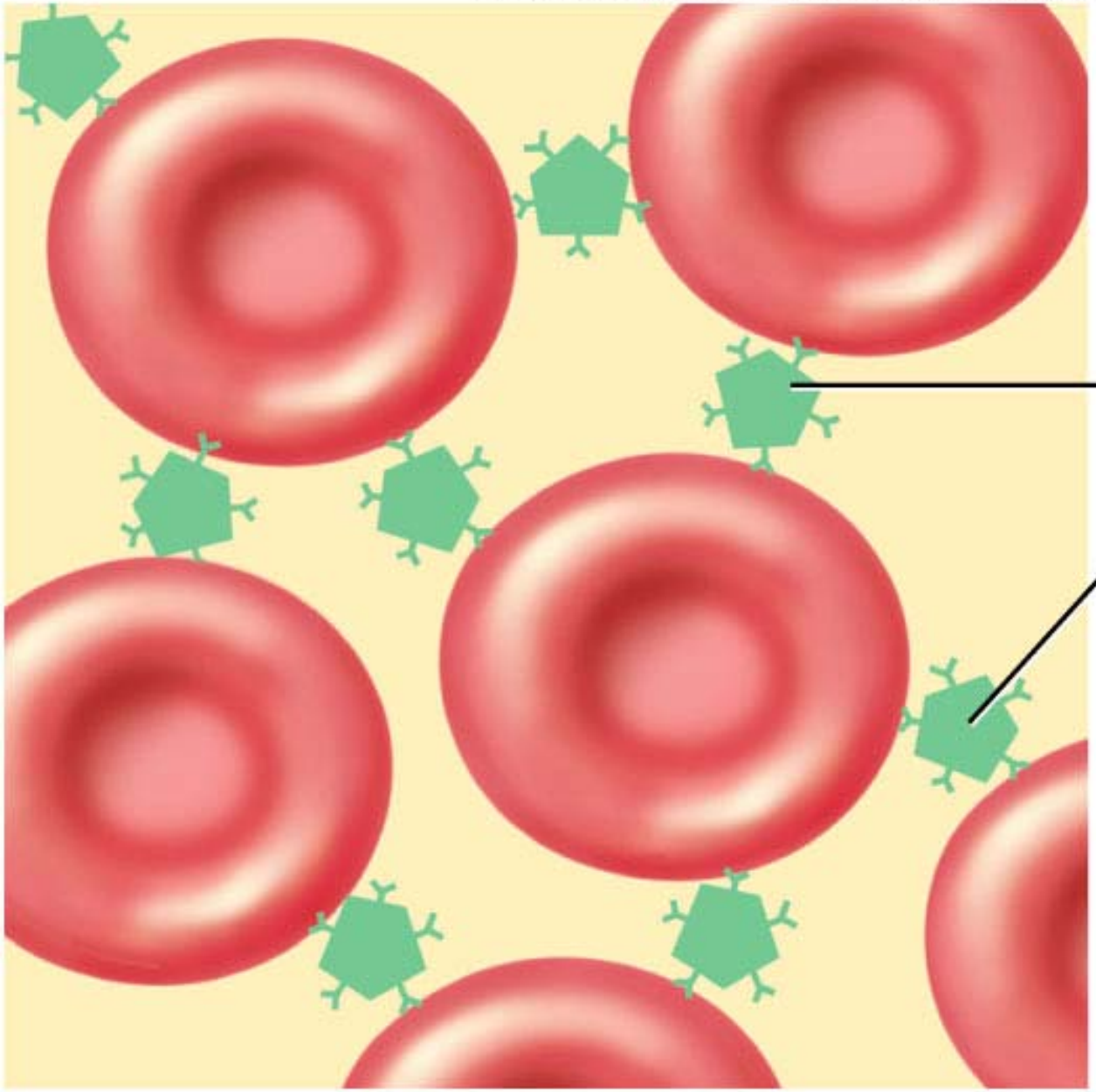
Antigens and Antibodies

- **Antigens**
  - **unique molecules on cell surface**
    - **used to distinguish self from non-self (foreign)**
    - **foreign anti*gens generate* immune response**

- **Antibodies**
  - **secreted by plasma cells (B cell precursor to plasma cell)**
    - **Protein macromolecule (i.e. immunoglobulin / Ig)**
    - **“Humoral” Immune response to foreign matter (i.e. antigen)**
    - **Five classes (IgM / IgG / IgA / IgE / IgD)**

- **Agglutination**
  - **antibody molecule binding to antigens**
  - **causes clumping together of RBCs**

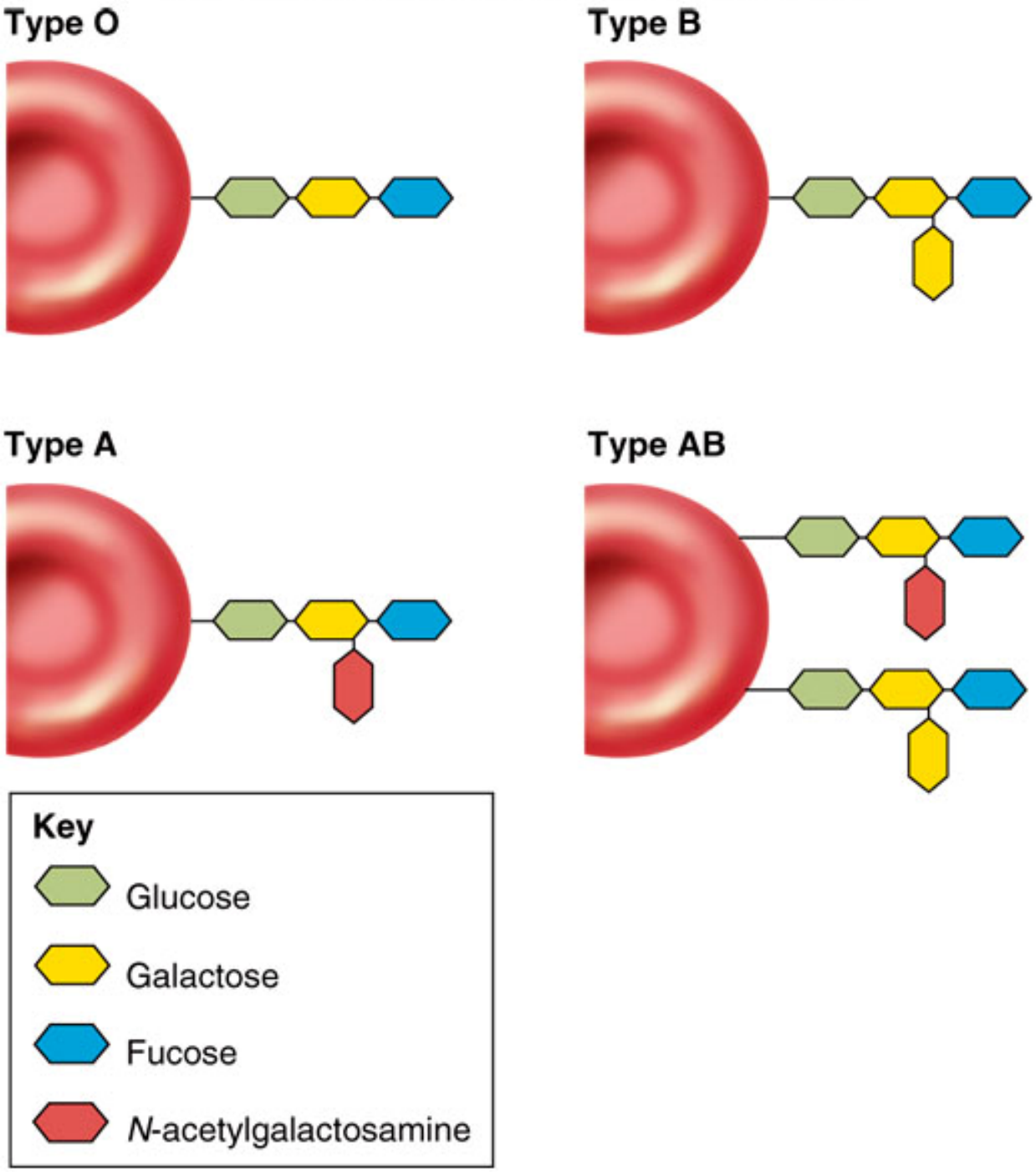
Agglutination of Erythrocytes



Antibodies do not “kill” cells. Antibodies “tag” foreign cells and toxins so other cells of the immune system can remove the pathogen.

ABO Group

- **Your ABO blood type is determined by presence or absence of antigens (agglutinogens) on RBCs**
  - **type A person has A antigens**
  - **type B person has B antigens**
  - **type AB has both antigens**
  - **type O has neither antigen**
  - **most common = type O**
  - **rarest = type AB**



Blood Types

- **RBC antigens**
  - **agglutinogens**
  - **A and B**
  - **on RBC surface**

Our cells are “sugar coated”. The surface of our cells are coated with many different types of sugars. These sugars are responsible for the ABO blood types.

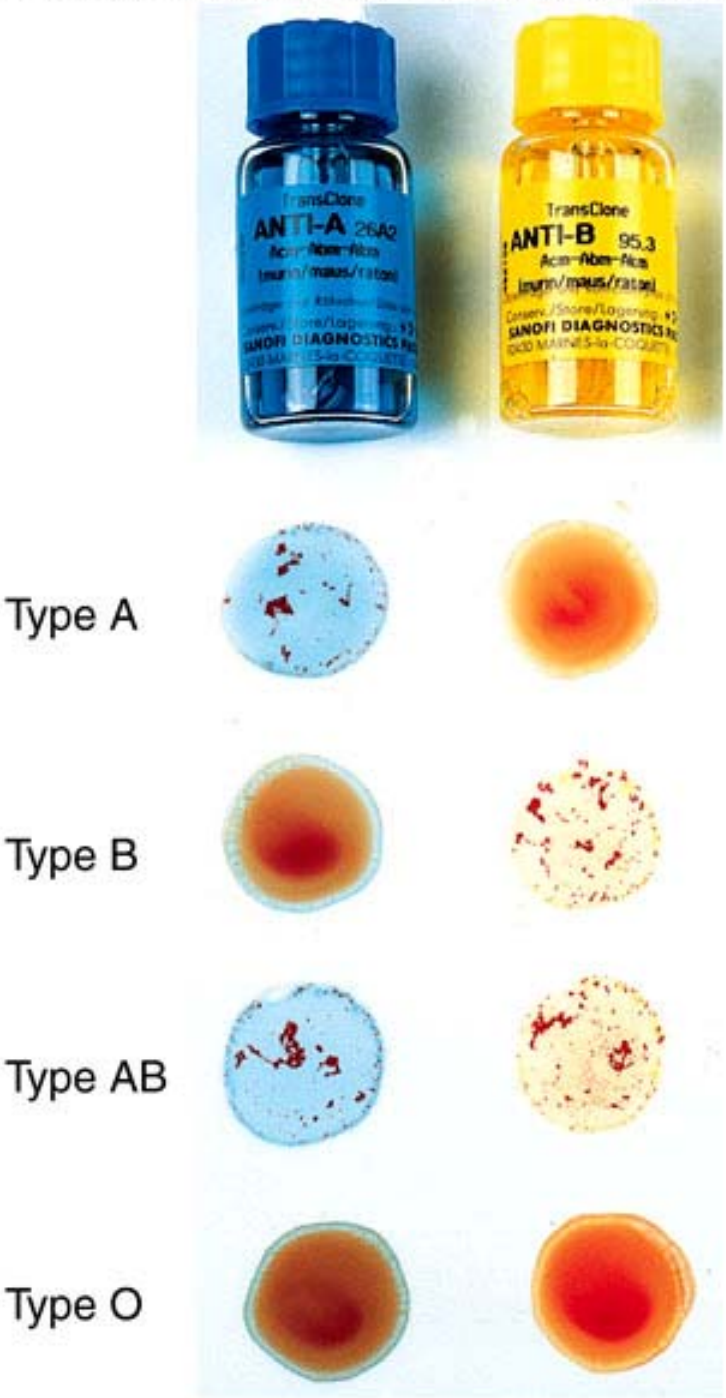
Plasma Antibodies

- **Antibodies (agglutinins)**
  - **Anti-A**
  - **Anti-B**

- **Antibodies appear 2-8 months after birth**
  - **maximum concentration at 10 yr.**
  - **Anti-A and/or Anti-B (both or none)**
  - **circulate in plasma**
    - **you will not form antibodies against “your antigens”**
    - **form antibodies against the “Anitgen NOT PRESENT”**

- **Agglutination**
  - **each antibody can attach to several foreign antigens at the same time**

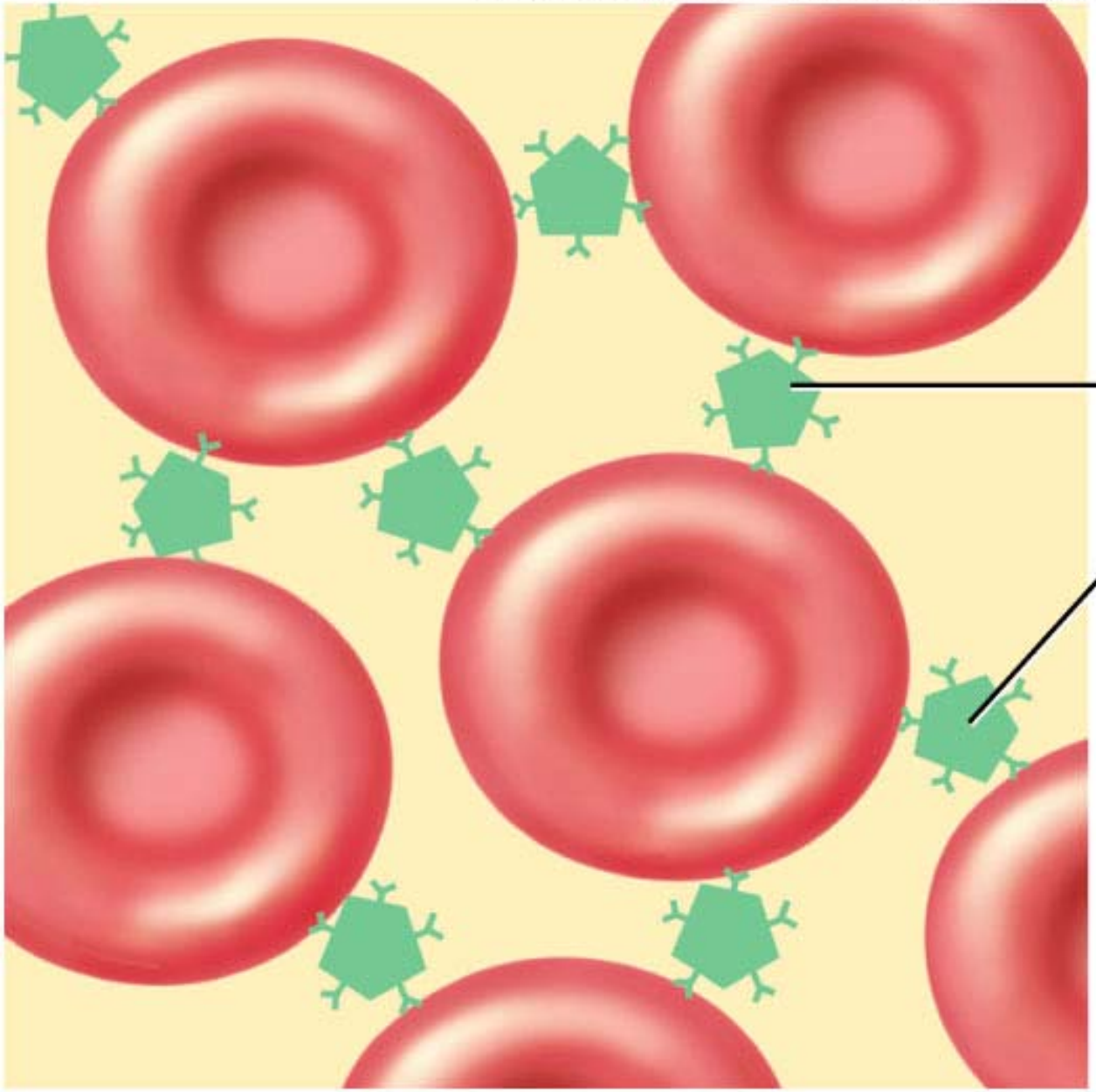
- **Responsible for mismatched transfusion reaction**



ABO Blood Typing

The Anti-A and Anti-B are solutions with antibodies A and B. If the droplet of blood contains RBCs with a “matched” antigen, then an antibody-antigen complex forms. One antibody can bind to several RBCs. This is known as agglutination. These “clumps” can get stuck in vascular biforcations and smaller vessels.

Agglutination of Erythrocytes

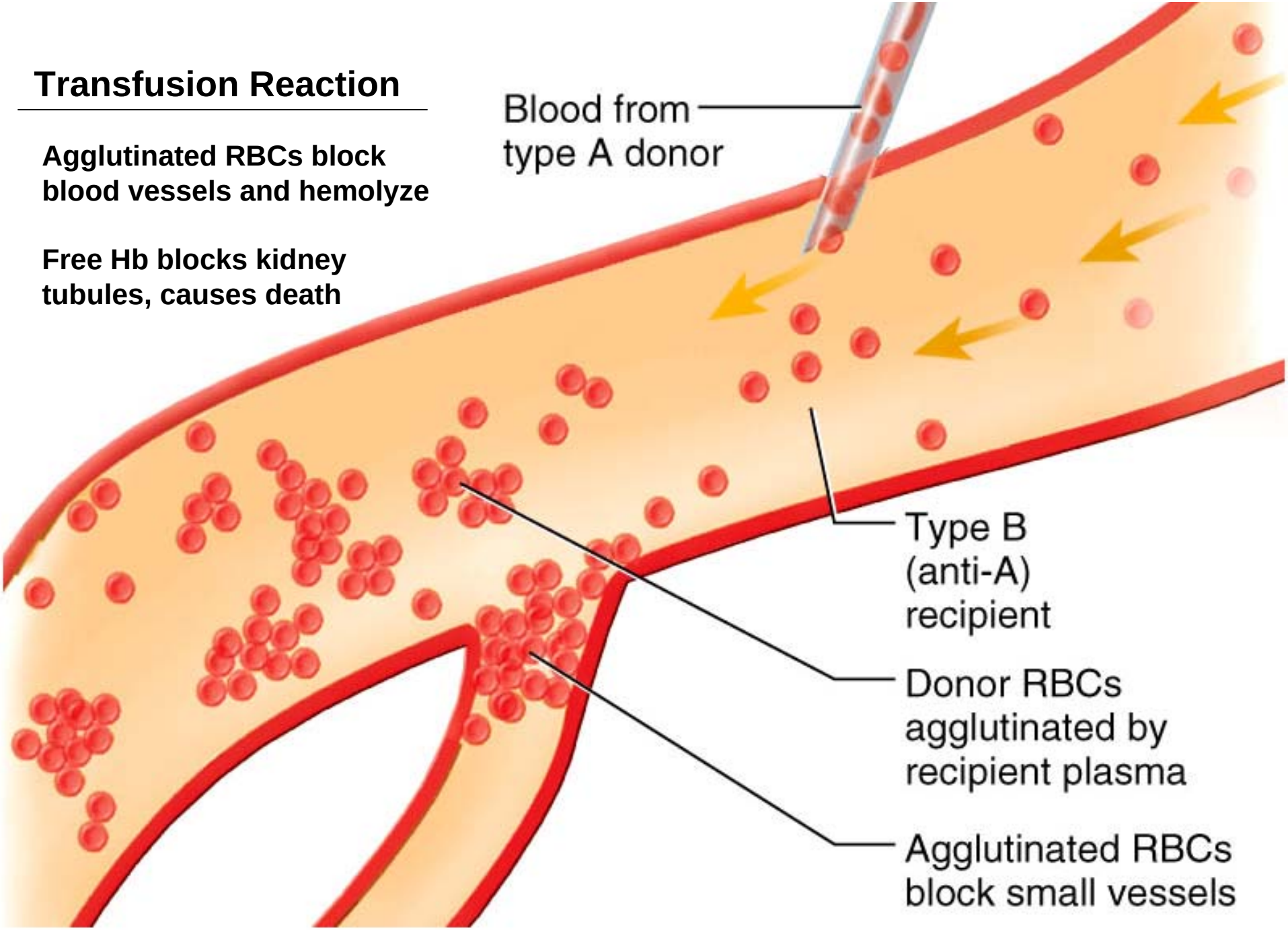


The IgM antibody associated with ABO Blood Typing has “ten active sites” on each antibody (i.e. green object). Some antibodies are monomers (e.g. IgA) and these antibodies have only two active sites.

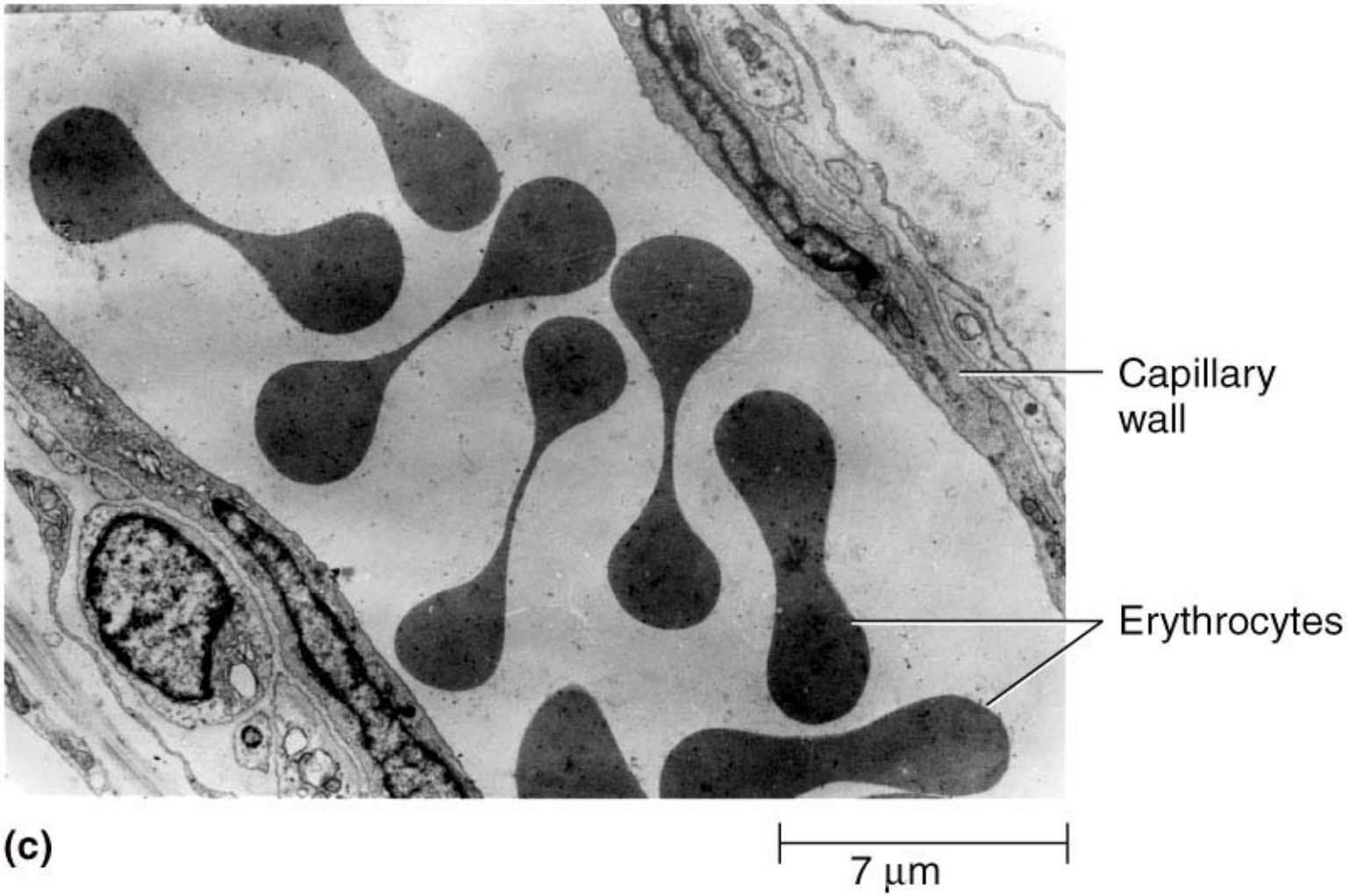
Transfusion Reaction

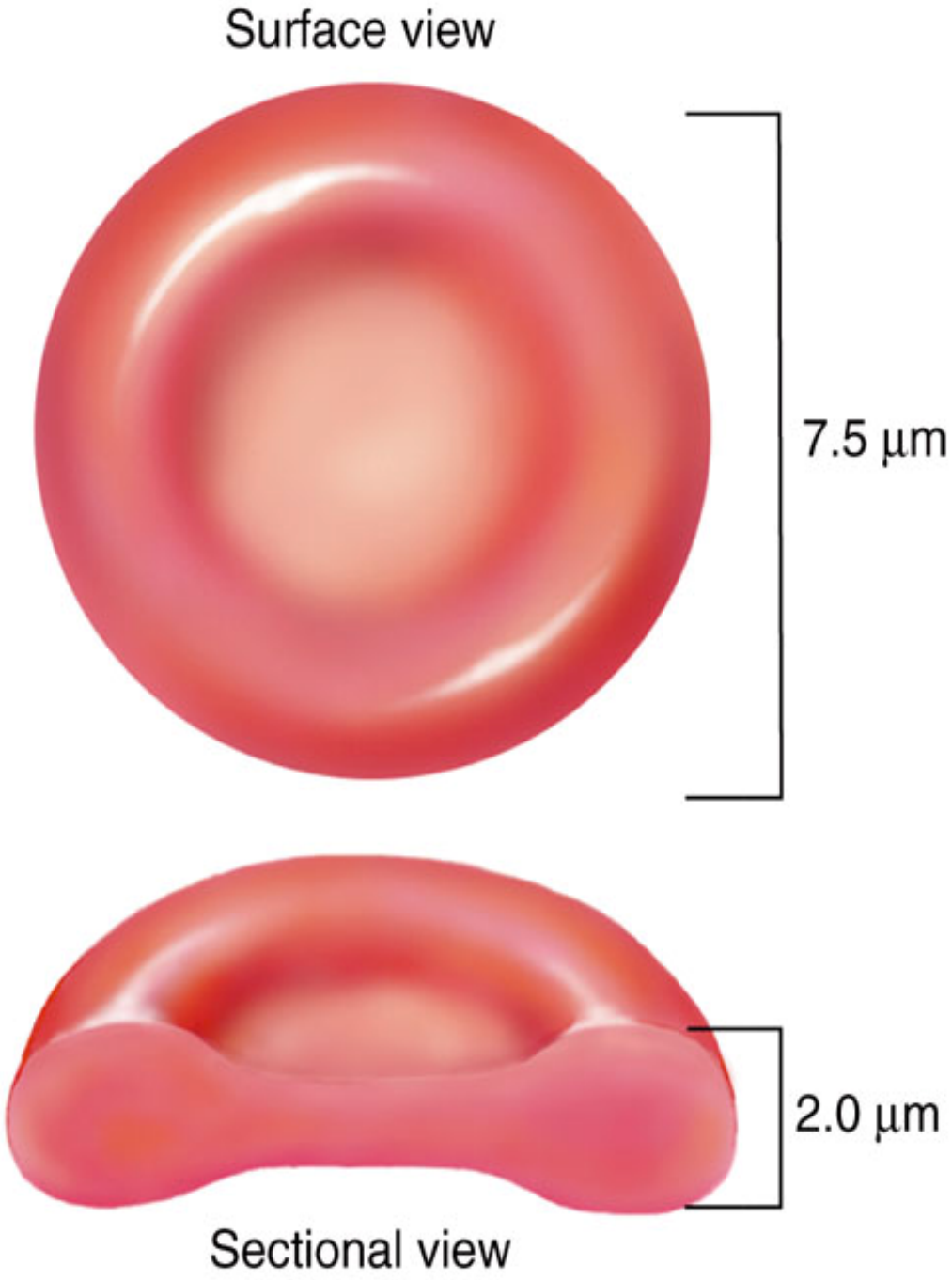
Agglutinated RBCs block blood vessels and hemolyze

Free Hb blocks kidney tubules, causes death



Erythrocytes





The diameter of some capillaries in the spleen are only 2um. RBC must “squeeze” through these capillaries. After 120 days, the RBC membranes become fragile and easily break. This is why the spleen is often called the grave yard for old RBCs.

Lining the inside of the RBC plasma membrane are protein fibers referred to as the “cytoskeleton”. Actin and spectrin are two protein fibers of the cytoskeleton. Actin confers strength and spectrin confers flexibility. Since RBCs lack a nucleus and other organelles to repair and replace cytoplasmic proteins, as the RBC ages these proteins start to break down. The lose of strength and flexibility results in the rupture of the aged RBC.

Universal Donors and Recipients

- **Universal donor**
  - **Type O**
  - **lacks RBC antigens (no antigen on membrane!)**
  - **donor's plasma may have antibodies against recipient's RBCs**
    - **to minimize this risk**
    - **give packed cells (minimal plasma)**

- **Universal recipient**
  - **Type AB**
  - **lacks plasma antibodies**
    - **no anti - A**
    - **no anti - B**

Rh Group

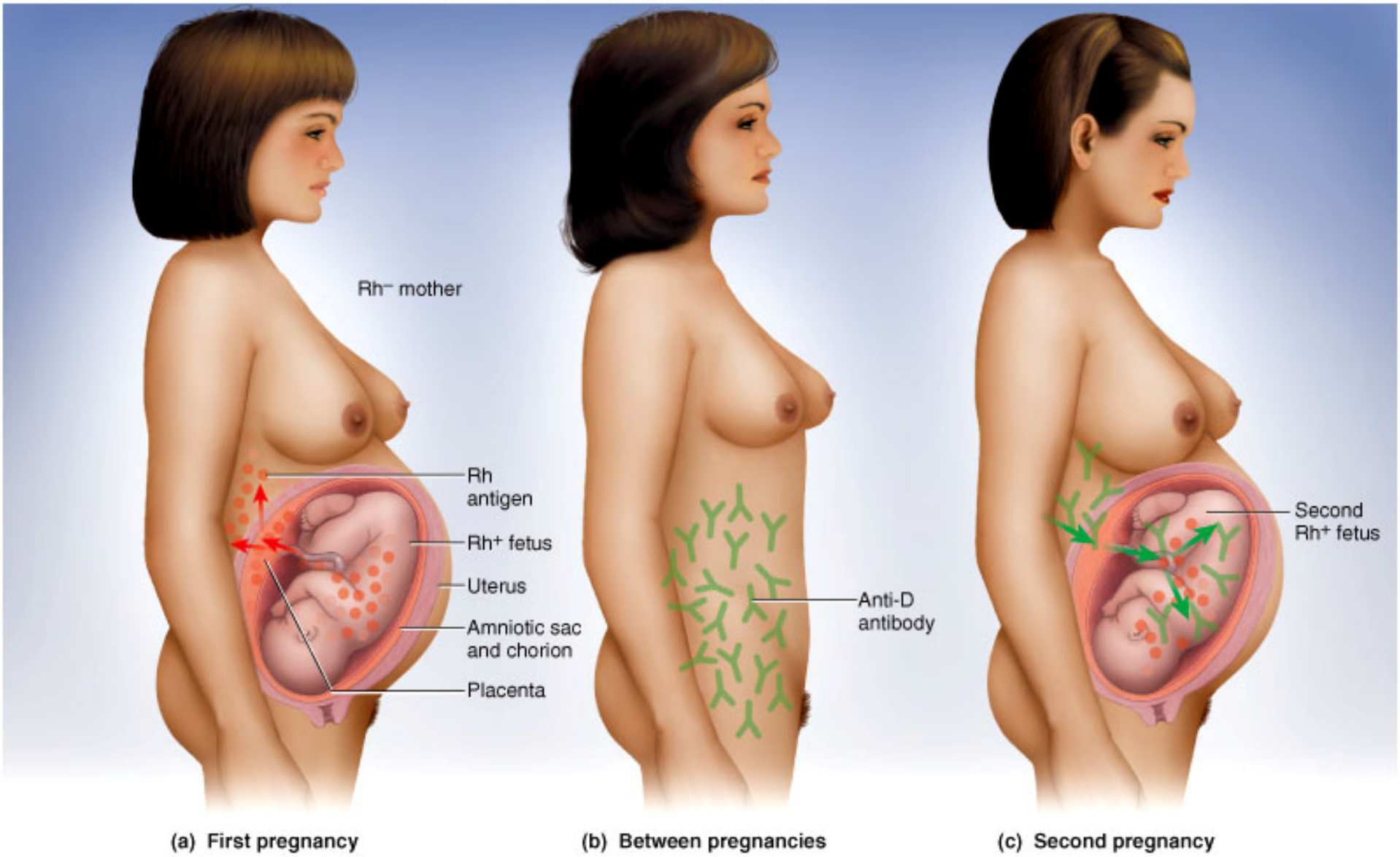
- **Rh (D) agglutinogens discovered in rhesus monkey in 1940**
  - **Rh^+ blood type has D agglutinogens on RBCs**
  - **Rh frequencies vary among ethnic groups**

- **Anti-D agglutinins (antibodies) not normally present**
  - **Rh negative person will not have anti-D (D antibodies)**
    - **This is different than what we saw in the ABO system**
  - **Anti – D antibodies only form in Rh^- individuals after being exposed to Rh^+ blood**
    - **Rh^- woman with an Rh^+ fetus**
    - **transfusion of Rh^+ blood**
    - **no problems with first transfusion or pregnancy**

Hemolytic Disease of Newborn

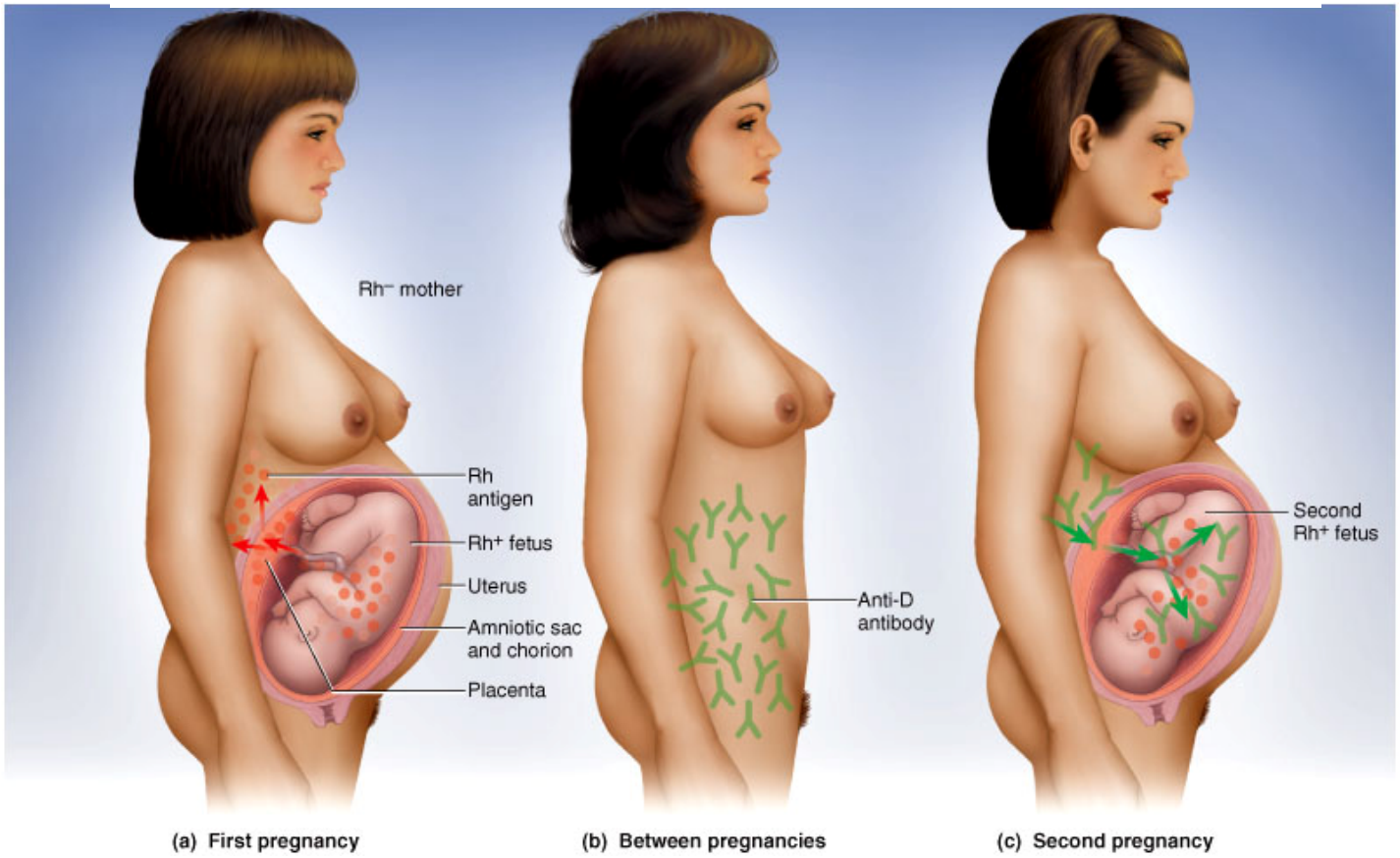
- **Occurs if mother has formed antibodies (anti-D) and is pregnant with 2nd Rh^+ child**
  - **Anti-D antibodies can cross placenta**

- **Prevention**
  - **RhoGAM given to pregnant Rh^- women**
    - **during delivery, fetal antigen D may mix with mothers blood, this would result in stimulating the mother's immune system to make anti-D**
    - **RhoGAM binds fetal agglutinogens in her blood so she will not form Anti-D (D antibodies)**



Rh antibodies attack fetal blood causing severe anemia and toxic brain syndrome

Hemolytic Disease of Newborn



Rh antibodies attack fetal blood

- Hemolyzed RBCs release Hb / Hb converted to bilirubin / high bilirubin causes brain damage that may kill the infant
- HDN can be treated with “phototherapy – exposing infant to UV light degrades the bilirubin as blood passes through the capillaries of the skin. In extreme cases, an exchange transfusion to completely replace the infant’s Rh+ blood with Rh- blood. In time, the infant will make new Rh+ but by then the mother’s antibody will have disappeared from the infant’s blood.

Other Blood Groups

- At least 100 known blood groups
- More than 500 known antigens
- Most common groups.
  - MN
  - Duffy
  - Kell
  - Kid
  - Lewis
- Rarely cause transfusion reactions
- Useful for legal purposes such as paternity, criminal cases, anthropological research, and population genetics.
- Dell, Kidd, and Duffy groups occasionally cause HDN.