

# OHIO PA Insider

Special December 2023 Issue

## A Senior Encourager

By Reverend Dr. R. Frank Lee

"What a difference access to appropriate care and love can make!"

Dear Andi,

I will never forget your story. When a life is saved most medical professionals say they were just doing their job, giving the credit to their support teams. But what if they did not have a support team? It's a tale as old as time; but this one is different. This story is about impossible battles won, established programs learning new ways, and two suffering children whose lives were saved. This story is about a young Ohio University Physician Assistant, desperately trying to find a place to work with the poor because she "now knows this was what she wanted to do with her life." After being turned away by bureaucratic red tape, she offered to work without pay if the chance to serve people experiencing poverty was available. The doors closed again. And so, against my sternest warnings, she signed up for a medical mission to Uganda. Upon arrival, she discovered the U.S. "Medical team" was Her! An even bigger surprise revealed sick children were seen two days per year, and with the ink barely dry on her new degree, she was alone in Uganda and the sole U.S. provider for one of those days. Like all recent graduates, she wondered how good her PA training really was. She was about to find out.

To quote your notes, "Alice presented with complaints of feeling unwell. The patient never had a diagnosis due to finances. The grandmother was unsure of how long this episode had been occurring but stated it was something the child had to endure constantly. The patient was scared as she gingerly moved about. After being examined, she was found to have an enlarged liver, an enlarged spleen, scleral icterus, and ascites."



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Figure 1: Class of 2025 students and Dr. Cacchillo auscultating heart sounds during a cardio lab

The providers' hearts were broken when they realized the extent of her condition. Due to the lack of resources, no medicines they had available would be able to touch her condition. It is difficult to come to terms with the possibility of leaving the child to suffer without proper care. What happened next? Again, from your notes, "The patient's story was brought to the attention of the remainder of the mission team. Despite the lack of funding, it was decided the patient needed to be transferred to the hospital. She was taken to Kampala, where her diagnosis of sickle cell was confirmed. She received blood transfusions and was given medication for pain. In addition to her sickle cell diagnosis, she was found to have pneumonia and an enlarged spleen, for which she will possibly have surgery. It was scheduled for her to return to the hospital in one week for a follow-up. For this visit, it was \$2,000. For the next \$ 3,000. She was sent home with a smile on her face and was given a new dress. What a difference access to appropriate care and love can make!"

As you explained, in sickle cell disease, the misshapen blood cells can break, become trapped within blood vessels, and obstruct blood flow. This leads to severe pain, something many sickle cell patients have become accustomed to experiencing. Although this is a terrible disease, it can be managed with medication. However, in Uganda, children constantly deal with the agony associated with this disease due to the inability to receive proper care. Most of these patients pass away at a young age and never make it to adulthood. Alice's continued management will cost around \$100 a month. So, the PA's, nurses and Ugandan teammates found a way to explain to the Mission Group that their policies were jeopardizing children's lives and needed change was begun. Any experienced executive will confirm that convincing a long-existing institution that they need profound change is difficult even for the most seasoned consultant. To do so in a way that breaks down protecting turf and inspires rather than confronts may be the biggest miracle of all. Andi, I have no idea how you and your Ugandan counterparts did this. I do know that today they are striving to establish an actual clinic to serve these desperate people. Andi, that is galaxies away from "two children's clinic days per year."

I have now learned your team diagnosed a young boy who requires immediate surgery. And you also instituted fundraising to get this accomplished as well. When I offered help in setting up a vehicle to fund Alice's treatment - "Oh, Father Frank, that has already been done." I should have known. You see, this was no ordinary "mission member." This was the Physician Assistant from Ohio University who working with her Ugandan provider teammates saved two children and inspired change in an organization because they were "called to serve the poor."

Mother Teresa said, "We can cure physical diseases with medicine, but the only cure for loneliness, despair, and hopelessness is love."

Miss Lamb, Ohio University taught you medicine well; your family taught you compassion, and your faith taught you to serve those in need with great love.

You have made us all proud! Andi, I cannot wait to see how this story ends!

May God Bless and Protect You,

Father Frank +



Figure 2: Father Frank & Andi Lamb, PA-C

# Alumni News

## Submitted First Jobs of 2023 graduates:

**Alex Ancona, PA-C, AT**

Emergency Medicine  
Mid-Ohio Emergency Services

**Katie Boehner, PA-C**

Emergency Medicine  
Mid-Ohio Emergency Services

**Emilia Bowers, PA-C**

Emergency Medicine  
Mid-Ohio Emergency Services

**Haily Cisneros, PA-C**

Emergency Medicine and Urgent Care  
Emergency Physicians Professional Association  
Minnesota

**Joe Delatore, PA-C**

Emergency Medicine  
Ashtabula County Medical Center

**Matthew Eng, PA-C**

Head and Neck Oncologic Surgery

**Haley Guggenheim, PA-C**

Pediatrics  
Upper Arlington Pediatrics

**Kate Kinley, PA-C**

Surgical and Medical Oncology Fellow  
The James Cancer Hospital and Solove Research Institute  
The Ohio State University Medical Center

**Andrew Kraly, PA-C**

Surgery PA Resident  
The Johns Hopkins University Medical Center  
Baltimore, Maryland

**Andi Lamb, PA-C**

Emergency Medicine  
Mid-Ohio Emergency Services

**Meredith Mayfield, MPAP, PA-C**  
Observation and Emergency Medicine  
Elkhart General Hospital  
Indiana

**Natalie Molnar, PA-C**  
Family Medicine  
Colorado Springs Family Practice

**Miranda Moore, PA-C**  
Family Medicine  
Ohio Health Primary Care  
Hilliard

**Nicholas Musico, PA-C**  
Neurosurgery  
Grant Medical Center  
Columbus

**Thomas Nasr, PA-C**  
PA Hospital Internal Medicine Fellow  
Mayo Clinic  
Arizona

**Cassie Nemeth, PA-C**  
Heart Failure and Heart Transplantation  
Ross Hospital  
The Ohio State University Medical Center

**Rachel Oliverio, PA-C**  
Emergency Medicine  
Mid-Ohio Emergency Services

**Lydia Stang, PA-C**  
Hospital Medicine  
MedOne Healthcare Partners  
Riverside Medical Center  
Columbus

**Jennifer Vu, PA-C**  
Dermatology  
Veterans Affairs Medical Center  
Chillicothe

**Abigail Willette, PA-C**  
Orthopedic Trauma  
Grant Medical Center  
Columbus

## Anniversaries

Reilly and Brian Candow celebrated 5 years of marriage on December 22, 2023.



Figure 3: Reilly and Brian Candow with their 6 month-old daughter, Reagan



# Weddings

- Alicia Pierse and Justin Cotton, May 21, 2022
- Katey and Zeke Liston, December 4, 2021
- Rachel Oliverio and Matty Pyles, August 20, 2023
- Samantha Roe and James McDonnell, August 19, 2023



Figure 4: Alicia Piefse & husband, Justin Cotton

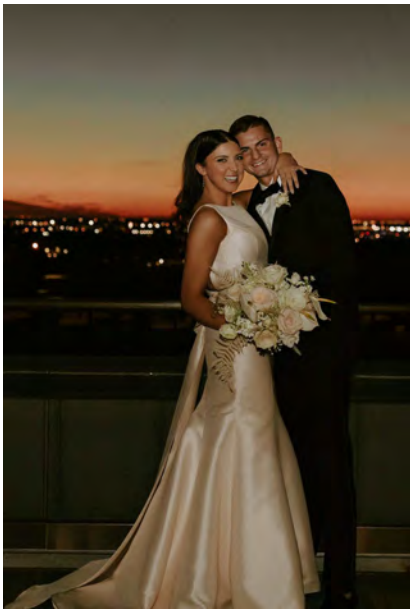


Figure 5: Rachel Oliverio & husband, Matty Pyles



Figure 6: Samantha Roe & husband, James McDonnell



Figure 7: Alicia Pierse & husband, Justin Cotton



Figure 8: Mackenzie Vaclav & Ryan Kusilek

# Engagements

- Mackenzie Vaclav and Ryan Kusilek
- Hannah Shortridge and Carter Rudek

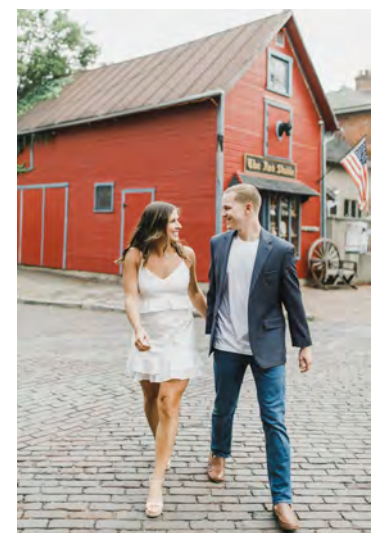


Figure 9: Hannah Shortridge & Carter Rudek

# Function of the Immune System

*By Dr. Jeffrey Vasiloff, M.D., MPH*

The purpose of the immune system is to defend the body against invasion by pathogenic microbes (“infection”). To accomplish this, the immune system must be able to recognize that the body’s “perimeter” has been breached. One of the main ways our immune system recognizes invasion is through the detection of unique cell-surface makers on microbes. For example, rhinovirus has “rhinovirus markers” and the causative agent of Strep throat has Streptococcus pyogenes markers.

“Identity” markers are called antigens

Antigens not only exist on microbes, but also on our own body cells. The markers on our body cells are called self-antigens. On the other hand, the markers on microbes are called non-self-antigens (or foreign antigens). Our immune system is “trained” to be able to distinguish non-self-antigens from self-antigens.

The “discernment” between self- and non-self is essential to health

This allows our immune system to create immune weapons to attack microbial invaders, while, at the same time, refraining from making weapons against our own cells and tissues. Autoimmune diseases arise when our own immune system mistakenly attacks our cells and tissues. More about this will be discussed later.

Microbes are not the only source of non-self antigens encountered by the body

Many environmental proteins and other inanimate substances have foreign identity markers as well. These include pollen, pet dander (flakes of dead skin), bee sting venom, shellfish proteins, peanut proteins, and so on. But environmental proteins and other inanimate substances are NOT microbes, and they cannot ever infect our bodies.

Allergic diseases arise when our immune system attacks inanimate proteins and substances

Thus, allergic diseases are distinct from autoimmune diseases. In allergic diseases, the immune system attacks non-self molecules that we “eat” or “breathe in” or “touch our skin.” But these molecules are not a part of our bodies. They are derived from the outside of our bodies. In autoimmune diseases, it is our own body cells and tissues that are attacked.

Both allergic and autoimmune diseases arise from errors committed by our immune system

But they are due to different errors. In allergic diseases, our immune system correctly identifies environmental or dietary molecules as foreign or non-self, but it incorrectly judges these molecules as threats to our bodies. In autoimmune diseases, our immune system incorrectly identifies our own cells and tissues as foreign or non-self AND incorrectly judges these essential components of our own bodies as threats. Before we discuss the details of allergic and autoimmune diseases, let us review the key functions of the healthy immune system.

The normal immune system has three parts or layers

The first component consists of anatomic surface barriers and related physiologic deterrents. Together, these are simply called “barriers.”

Unbroken or intact skin is a robust barrier against infection

Intact skin blocks invasion by almost all viruses, bacteria, protozoans, fungi. Some superficial fungi can, however, infect the epidermis (like “athlete’s feet”); however, most of the deadly fungi that are capable of deep tissue and bloodstream infections cannot “breakthrough” intact skin.

## Helminths or worms are exceptional as some can “burrow” through intact skin

In the U.S, fortunately, exposure to helminths is limited.

## Mucus membranes are barriers, but are far less impenetrable than skin

Mucus membranes make use of antimicrobial peptides, like defensins and secretory IgA. Secretory IgA is one of five classes of antibodies that can bind to (and thereby inactivate or hinder) invading microbes. However, many pathogenic microbes can overcome this nonspecific immune system weapon.

## Gastric acid creates a deadly milieu many ingested microbes

This is why we usually do not develop “food poisoning” every time we ingest contaminated foods like unwashed fruit. However, some microbes and microbial toxins can survive the low pH within the stomach.

## The acidity of urine and vaginal secretions is also a barrier to some infections

However, the degree of acidity is much less than within the stomach.

## The trachea and larger airways provide surprisingly good protection against infection

These large airways are “lined” with a ciliated pseudostratified columnar epithelium. Cilia are in constant motion “beating” upward. This continuously propels a mucus layer that overlies the epithelium. For example, when near a campfire, soot is often inhaled.

These particles “stick to” airway-lining mucus, which prevents them from penetrating more deeply into the lungs, and, in addition, moves them upward where they can be swallowed or spit out or coughed out. The same happens when pathogenic microbes enter these airways. In this way, many potential cases of pneumonia are prevented. It should be noted that the toxicity of tobacco smoke triggers ciliated stratified columnar cells to “morph” into squamous cells. This “metaplasia” is damaging because it impairs the function of the “mucociliary escalator.”

## The “microbiome” is an essential defense barrier against infection

What is the microbiome? It is a collection of viruses, bacteria, and fungi that live on, or in, the body. Each of us has an extensive microbiome. For example, there are about 10 microbial cells on and within our body for every single body cell. The microbiome consists of a wide array of different microbial species and strains or variants of species.

## Different people can have vastly different arrays of these microbes

In addition, there are differences among people in the quantities of various microbes. Why are there differences? This is not completely known, but some known or suspected factors include: a) genetics; b) immune system characteristics; c) age; d) general health status; e) diet; f) former diets; g) body weight; h) current and past antibiotic exposure; i) other medication exposures; j) smoking; k) alcohol; l) illicit drugs (exposure to substances); m) characteristics and health of family members; n) occupation; o) location; p) travel history; q) current and former infectious diseases; r) current and former diseases of all kinds, among others.

## And individual's microbiome can decrease or increase susceptibility to infection

That is, there are microbiomes that are more healthy and those that are less so. Further, microbiomes can change many times or continuously through life.

## Dysbiosis refers to the development of a less healthy microbiome

In the colons of healthy people, there are small numbers of pathogenic bacteria. These bacterial species, such as *Clostridioides difficile*—if “given the chance”—would replicate freely and cause infection of the colon, sometimes leading to life-threatening bloody diarrhea. However, a healthy microbiome—in ways we do not fully understand—“keeps these small numbers of trouble-making bacteria in check.”

Unfortunately, when certain antibiotics are appropriately prescribed to treat infections outside of the bowels, these same antibiotics “kill off” some of the healthy microbiome. And in some cases, this change—this dysbiosis—can remove the inhibitory effect on resident *Clostridioides difficile* microbes so that they replicate and infect the colon.

## Fecal transplantation can be used to improve a patient's microbiome

Amazingly, in *Clostridioides difficile* infections, transferring an array of normal microbiome bacteria from a healthy person into the colon of a patient with colitis is often curative. Whether improving the microbiome by fecal transplantation to cure or prevent other infectious or other diseases is an active area of research.

## The second component or layer of the immune system is called the innate immune system

This layer of the immune system consists of four main immune system “weapons”: a) acute inflammatory response, including phagocytosis; b) complement system of proteins; c) natural killer cells (NK cells); and d) interferons;

Acute inflammation has already been discussed in a prior chapter. The importance of this part of the innate immune system cannot be overstated. Think of all the times you have gotten a cut (laceration), scrape (abrasion), or insect bite (puncture wound). In these cases, skin bacteria, including some pathogenic species, are “allowed entry” into vulnerable deeper tissues.

However, it is uncommon for an infection to ensue. That is, it is uncommon for an infection to “take root” following most cuts, scrapes, or punctures of the skin. This is primarily due to the “swarming” of neutrophils, and later, monocyte-macrophages, which aggressively and effectively phagocytize the wound-contaminating bacteria.

## Patients with insufficient neutrophils greatly weakens the innate immune response

This can occur, for example, in patients receiving cytotoxic chemotherapy. It can also occur when a cancer of white blood cells (called leukemia) develops within the bone marrow. In this situation, the excessive replication of abnormal (“leukemic”) cells “crowds out” the production of normal white blood cells, including neutrophils.



## Our bloodstream contains rapidly-acting microbe-fighting proteins

There are several dozen of these proteins, which together, make up the complement “cascade” or complement system. Some of these proteins will recognize and “stick to” microbes, for example, within the bloodstream.

“Opsonization” is the process whereby microbes are “marked for destruction.”

As these “coated” bacteria circulate in the blood, and especially through the tortuous sinusoids of the spleen, macrophages recognize them as invaders. The macrophages are positioned along the lining of the sinusoids so that they can “swallow” and destroy them by phagocytosis. (phagocytosis).

In a different way, complement proteins can attach to virus particles in the blood—and in this way—render them incapable of invading body cells. This process is called “neutralization.”

Complement proteins can “work together” to form a “membrane attack complex” (MAC)

This “assembly” of the following activated complement proteins--C5b, C6, C7, C8, and C9—attaches to the surface of a bacterium, for example, and actually “drills a hole” into it, killing it. Some patients have genetic deficiencies of one or more complement components and as a result, are more susceptible to infection or death from infection. For example, those with deficiencies in any of the membrane attack complex proteins are at increased risk for infection and death from *Neisseria meningitides*.

Complement proteins, as well as other “weapons” of the innate immune system require a way to recognize invading microbes as threats to be destroyed. Fortunately, the molecular structures of most microbes have various “molecular patterns” that are recognized by the “pattern recognition receptors” (PRRs) of certain complement proteins and cells of the innate immune system.

The various molecular markers of microbes are called “pathogen-associated molecular patterns” (PAMPs) which are not present on body cells—only on microbes. These markers are general markers that are shared by many different microbes. This is in contrast to the very unique and specific markers that all microbes possess—specific markers (called antigens) that are used by the more powerful adaptive immune system (discussed below).

Natural killer (NK) cells are another weapon of the innate immune system

NK cells are a type of lymphocyte that can recognize when body cells are stressed or infected by intracellular microbes, such as viruses. Healthy body cells have major histocompatibility complex (MHC) class I molecules on their cell membranes. These markers prevent the “attack” by NK cells.

However, in body cells that lose these markers--stressed cells, damaged cells, malignant cells, and cells infected with intracellular microbes--NK cells recognize them and “connect to them.” Then the NK cell releases “perforins,” which can “bore a hole” in an infected body cell. This is followed by the NK cell release of destructive enzymes called “granzymes”—which enter the hole and trigger cell death by apoptosis. The dying cell causes any intracellular microbes to perish as well.

## Interferon proteins released by infected cells diffuse to nearby uninfected cells to protect them

This innate immune system weapon is counterintuitive. This is because the attacked body cell acts as if it has “given up” on saving itself, and instead “tries” to save other cells. When a body cell is infected by an intracellular microbe, like a virus, this triggers the production of interferon proteins, which enter nearby uninfected cells. Once inside healthy cells, interferon proteins trigger changes within these cells that “strengthen” or “harden” these cells against succumbing to infection themselves.

Released interferons also “strengthen” the virus-killing function of circulating NK cells. In fact, there is evidence that patients with deficiencies in interferon production—or inactivation of interferon by autoantibodies—have worse outcomes from Covid-19 infection.

## The third part or layer of the immune system is the adaptive immune system

The word, “adaptive” refers to the fact that the corresponding immune system weapons are “adapted” or “specifically created” against the exact causative microbe. That is, adaptive immune system weapons are “perfectly shaped” to “seek and find” and “bind to and destroy” infecting microbes only—identified by their specific antigen markers. Because these weapons are specifically created for the specific causative microbe, they are the most powerful immune system weapons.

## Unfortunately, it takes a few days for these weapons to be “built” or “created.”

This is because the antigen markers of an invading microbe must be captured and presented to the immune system—followed by the “mass production” of the specific adaptive weapons. Thus, some rapidly progressing infections can kill before these weapons are made.

## There are two “arms” of the adaptive immune system

The humoral arm results in the production of the important weapon of antibodies, while the cellular arm results in the production of “cytotoxic T cells.” The humoral arm relies on various B cells, plasma cells, antigen-presenting cells, soluble antibodies, and helper T cells, along with the cytokines that helper T cells release. The cellular arm relies on various T cells, including helper T cells and the cytokines that they release, antigen-presenting cells, such as dendritic cells, and cytotoxic T cells.

## Helper T cells act like a “choreographer” or “stage director” of the immune response

Helper T cells—while not direct-weapons themselves, secrete various cytokines that signal various cells of both the innate and adaptive immune system to “ramp up” their capabilities and the strength of their weapons. That is, helper T cells cytokines “supercharge” other cells of the immune response.



In fact, without the participation of helper T cells, the entire immune system “falls apart.” It is counterintuitive that the loss in helper T cell function alone prevents the “work” of so many immune system “effectors” or weapons. Without stimulation by helper T cells, all of the following are rendered less effective: a) phagocytosis by neutrophils and macrophages; b) replication of other classes of T cells, B cells, and plasma cells; c) production of antibodies by plasma cells; and d) cytotoxic T cell destruction of cells harboring intracellular microbes, to name a few.

The central role of helper T cells was seen vividly in the past in Acquired immunodeficiency syndrome (AIDS) due to infection by HIV. Ironically, the HIV virus selectively attaches to helper T cells, enters them, and destroys them (if not prevented by current antiretroviral treatment). Almost all of the many deaths from AIDS were due to the selective destruction of the essential “choreographer” or “coach” of the immune system, the helper T cell.

### Dendritic cells that are “on the lookout” for invading microbes

Dendritic cells exist throughout the body at sites where microbes are likely to invade—for example, in the dermis of the skin and underlying the epithelia of the oropharynx, bronchial airways, alveoli, intestinal tract, urogenital tract, and elsewhere.

### Dendritic cells are always “on the lookout” for microbes

When invasion by microbes occurs, nearby dendritic cells “swallow” or phagocytize them. In this process, they “collect” the microbe’s unique markers or antigens. It is interesting that antigens can be broken down into smaller antigen fragments called “epitopes.” These are also unique for each microbe.

### Dendritic cells place digested microbe antigens and epitopes on their cells surface

This is why dendritic cells are called “antigen-presenting” cells or APCs. Dendritic cells are not the only cell type that can present antigens, but they are the most common one. Antigen-presenting cells “advertise” or “reveal” the identity of the invading microbe to circulating T cells. The majority of both T and B lymphocytes circulate throughout the body within the bloodstream, lymphatic channels, and structures like lymph nodes and the spleen. In addition, some B and T lymphocytes reside in lymph nodes and the spleen.

### There is a natural subset of circulating T cells that is a perfect match for any microbe’s antigens

From very early life, a vast assortment of T cells is produced. In most ways, these T cells are identical in appearance and function. However, they are different with respect to one of their cell-surface receptors, the so-called T cell receptor. Although it may be hard to believe, by extensive and random genetically-programmed processes in early life, there are innumerable subsets of T lymphocytes, each with a unique T cell receptor.

For example, there is a natural subset of T cells with a T cell receptor that has a “perfect fit” for the antigen or epitope of the causative agent of influenza. Let us say a person develops influenza, what happens with respect to the immune response?

First, some invading influenza virus is phagocytized and digested by dendritic cells of the oropharynx. Influenza antigens and epitopes are then “presented” on the dendritic cell’s surface to notify that the body has been invaded (and infected) with influenza virus. However, within the patient’s body, for example, within the blood, lymph, and lymph nodes, there is a subset of T cells who have a receptor that is a match for the presented or displayed influenza virus antigen or epitopes.

### The matching of a T cell’s receptor with a microbe’s antigen kick-starts the immune response

That is, it kick-starts the body’s adaptive immune response. This is the part of the immune system that requires a few days to make powerful weapons against influenza virus--to continue our example.

## T cells replicate wilding when a match occurs, creating a clone of T cells

This results in the creation of many millions of identical T cells. However, these T cells with “mature” into a few different types of T cell (but all retaining the “exact fit” for influenza virus antigens and epitopes—to continue our example)

Some become: a) helper T cells, that will stimulate and “supercharge” the cells of the entire immune system; memory T cells, which will “be ready” to form immune system weapons quickly in the future if the microbe is encountered again; c) cytotoxic T cells that are able to destroy invading microbes, especially those that “live” inside body cells, like influenza virus.

## Antigen-presenting cells also engage matching B cells

When a specific subset of B cells recognizes a corresponding microbe’s antigen or epitopes, these B cells replicate wildly, just as do corresponding T cells. Many of these B cells differentiate further (or “morph into”) plasma cells.

## Plasma cells function as antibody production “factories”

Antibodies are the powerful weapons of the humoral arm of the adaptive immune response. The most important characteristic of antibodies is that they recognize the causative invading microbe. That is, they are a weapon that was created—over 2-4 days—to bind tightly to the microbe’s cell membrane or virion.

## Antibodies opsonize microbes, but are even more powerful than complement proteins

Further, once antibodies attach to microbes, this often attracts complement to attach as well. In other words, the attack by both complement proteins and antibodies are synergistic. Antibodies are especially effective at neutralizing or triggering the destruction of extracellular pathogens, such as most bacteria.

Antibodies are not cells, but rather, an assemblage of polypeptides chains. There are five different classes: a) IgM; b) IgG; c) IgA; IgE; and IgD.

## Specific IgM antibodies are the “first responders” to most infections

Thus, plasma cells first produce IgM antibodies against the infection’s causative organism. Once produced, IgM antibody levels in the blood fall. Thus, the appearance of specific IgM in the blood can be used to diagnose an acute infection. For example, in the viral infection, hepatitis A, acute infection is diagnosed by the appearance of IgM in the blood.

## Specific IgG antibodies are the second wave of antibodies produced by plasma cells

These antibodies also “attack” the invading microbe, however, in contrast to IgM, their levels persist in the blood for years. While detecting IgM against hepatitis A indicates “current” or “very recent” or “acute” hepatitis A, the finding of the corresponding IgG indicates only that the patient was once infected with hepatitis A. For example, say in January of 2000, a patient develops acute hepatitis A. At that time, the IgM test will be positive, and soon afterward the IgG test will also be positive. But after a few months, the IgM test will be negative, while the IgG test will remain positive. And say the patient’s blood is checked in January of 2010, the IgG test will still be positive.

The fact that it is still detectable in the blood indicates that there are plasma cells within the blood that are still producing IgG antibody against hepatitis A virus. This is why the patient cannot become re-infected with hepatitis A virus. That is, the patient is immune to hepatitis A by the continuing production of (low, but sufficient) levels of IgG antibody against the virus.



## IgA antibodies are secreted by plasma cells at mucus membranes

For example, IgA antibodies are present in: a) tears; b) saliva; and c) the mucus of the: i) nasal cavity and sinuses; ii) oropharynx; iii) airways; iv) esophagus, stomach, and intestines; v) urinary tract; and vi) genital tract.

## People with impaired production of antibodies are at increased risk of severe infections

This is especially true of pathogenic microbes that live and reside outside of body cells. Most bacteria, for example, reside within the blood and body fluids. This means that those with either genetic or acquired defects in the humoral arm of the adaptive immune system are more susceptible to bacterial infections and other extracellular infectious pathogens.

## The main weapon of the cellular arm of the adaptive immune system is the cytotoxic T cell

Unlike antibodies, cytotoxic T cells do “stick” to microbes. Also, unlike antibodies that are potent weapons against extracellular pathogens, cytotoxic T cells are most potent against microbes that “hide out” within body cells, such as viruses.

Cytotoxic T cells approach and bind to infected body cells. Once “in position,” they release two types of molecules: a) perforins; and b) granzymes. Perforins “perforate” the infected cell’s membrane, which provides a portal for destructive granzymes, which kill both the body cell and the contained microbe(s).

Those with impaired cellular immunity are susceptible to both extracellular and intracellular pathogens. However, intracellular microbes can be especially severe. Some of these include: a) a) viruses, especially those with latency, such as: i) cytomegalovirus (CMV); ii) herpes simplex virus (HSV); iii) and varicella zoster virus (VZV) ; b) intracellular bacteria, such as mycobacteria; and c) many fungi, such as Pneumocystis jirovecii.

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