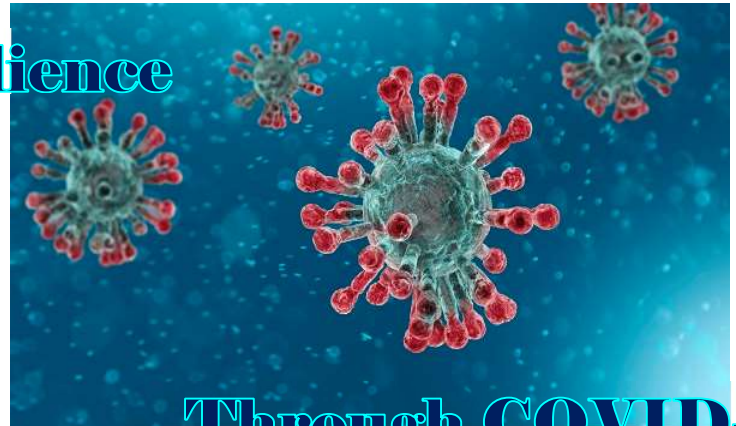


Power of I Wellness News

Newsletter 6 Δ June 01, 2020

Wellness Word — Resilience

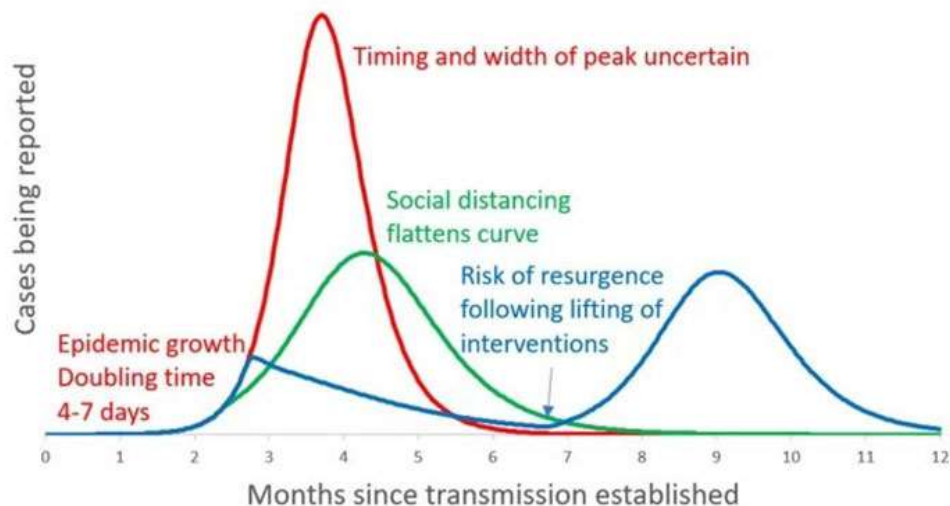
As we begin to reopen communities and our country at large after the 3-month interim in lockdown, we are able to mark real clinical progress: We have established an initial characterization of the virus and its mechanisms of transmission and routes of



Through COVID-19

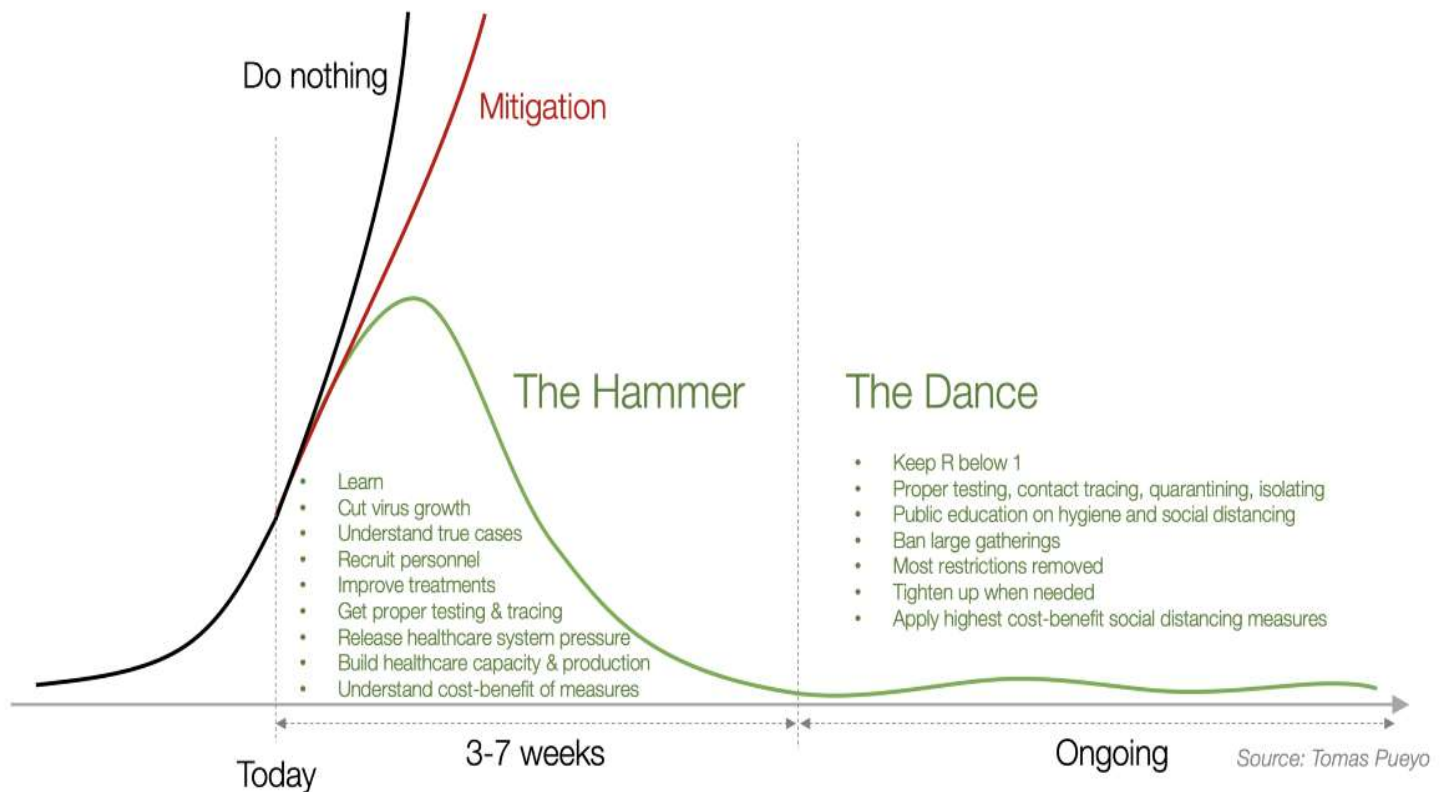
propagation within the body, albeit adding to and refining our pool of data case by case. We have determined that placing restrictions upon our activities, our distance and the means of transmission has resulted in stymied spread of the virus. International development of vaccines and treatments to alleviate viral symptoms, subsequent organ damage and lowering mortality rates is currently ongoing.

However, in the exceedingly short time that lockdowns have been partially lifted, we have already begun to record conflicts with those unwilling to social distance or wear protective masks and massive gatherings flaunting every protective guidance that has been issued; unsurprisingly, subsequent surges (in hundreds of persons) through health care systems are already being reported. Throughout the summer as restrictions ease, it is thus imperative that we do our part to remain vigilant, act responsibly and continue to practice now-proven methods of dampening transmission so as not to reverse the progress we have made thus far. To understand that the virus is not gone, should not be forgotten and to hold ourselves accountable so that we may all live as fully as possible but as safely as possible within the frame of our understanding.



According to the 5 stages of crisis management (Professor Juliette Kayem), we are currently transitioning from a response stage (stage 3) to an “adaptive recovery,” (stage 4) where we adapt to live with and manage the virus in the “new/now normal” until targeted treatments and effective vaccine distribution occur . This phase will be a more extended one than the initial response, characterized by i) reliable testing that allows for identification to isolation to containment; ii) limited community spread; iii) availability of treatment and ICU resources; and iv) reconstitution of operations.

Shown graphically (courtesy of Jigar Shah via LinkedIn) in another format, we are transitioning from The Hammer (or our initial responses) and The Dance (living with the virus in the safest non-isolated way possible).







Medical experts believe that a lack of social distancing, refusal to wear face masks and flouting of other precautions amid reopening could erase any progress in a matter of months. During the summer months “hot spots” are expected throughout the country, exacerbated by “super spreader” events, such as large parties and other gatherings that are especially high risk for transmission of the virus. Many experts, including National Institute of Allergy and Infectious Diseases director Anthony Fauci, MD, say that the chance of a second wave of the coronavirus could come as soon as September and persist through fall and winter.



Again, the presence and extent of the next expected peak is entirely our choice, up to us, as citizens of this country. It is responsible behaviors of ourselves, our families, our communities that will contribute to the successes that we may all share this summer. Hopefully, the coming months will provide opportunities for enjoyable time outdoors, small gatherings to catch up with family and friends and gratitude for all our additional activities and reconnections.



Epidemiological Comparison of Respiratory Viral Infections

Disease	Flu	COVID-19	SARS	MERS
Disease Causing Pathogen	 Influenza virus	 SARS-CoV-2	 SARS-CoV	 MERS-CoV
R_0 Basic Reproductive Number	1.3	2.0 - 2.5 *	3	0.3 - 0.8
CFR Case Fatality Rate	0.05 - 0.1%	~3.4% *	9.6 - 11%	34.4%
Incubation Time	1 - 4 days	4 - 14 days *	2 - 7 days	6 days
Hospitalization Rate	2%	~19% *	Most cases	Most cases
Community Attack Rate	10 - 20%	30 - 40% *	10 - 60%	4 - 13%
Annual Infected (global)	~ 1 billion	N/A (ongoing)	8098 (in 2003)	420
Annual Infected (US)	10 - 45 million	N/A (ongoing)	8 (in 2003)	2 (in 2014)
Annual Deaths (US)	10,000 - 61,000	N/A (ongoing)	None (since 2003)	None (since 2014)

* COVID-19 data as of March 2020.

Created in BioRender.com 

The following is a post from a cardiologist at UW, Dr James Stein, who is providing a COVID-19 update as we start to leave our cocoons.

"The purpose of this post is to provide a perspective on the intense but expected anxiety so many people are experiencing as they prepare to leave the shelter of their homes. My opinions are not those of my employers and are not meant to invalidate anyone else's - they simply are my perspective on managing risk.

In March, we did not know much about COVID-19 other than the incredibly scary news reports from overrun hospitals in China, Italy, and other parts of Europe. The media was filled with scary pictures of chest CT scans, personal stories of people who decompensated quickly with shortness of breath, overwhelmed health care systems, and deaths. We heard confusing and widely varying estimates for risk of getting infected and of dying - some estimates were quite high.

Key point #1: The COVID-19 we are facing now is the same disease it was 2 months ago. The "shelter at home" orders were the right step from a public health standpoint to make sure we flattened the curve and didn't overrun the health care system which would have led to excess preventable deaths. It also bought us time to learn about the disease's dynamics, preventive measures, and best treatment strategies - and we did. For hospitalized patients, we have learned to avoid early intubation, to use prone ventilation, and that remdesivir probably reduces time to recovery. We have learned how to best use and preserve PPE. We also know that several therapies suggested early on probably don't do much and may even cause harm (ie, azithromycin, chloroquine, hydroxychloroquine, lopinavir/ritonavir). But all of our social distancing did not change the disease.

Take home: We flattened the curve and with it our economy and psyches but the disease itself is still here.

Key point #2: COVID-19 is more deadly than seasonal influenza (about 5-10x so), but not nearly as deadly as Ebola, Rabies, or Marburg Hemorrhagic Fever where 25-90% of people who get infected die. COVID-19's case fatality rate is about 0.8-1.5% overall but much higher if you are 60-69 years old (3-4%), 70-79 years old (7-9%), and especially so if you are over 80 years old (CFR 13-17%). It is much lower if you are under 50 years old (<0.6%). The infection fatality rate is about half of these numbers.

Take home: COVID-19 is dangerous but the vast majority of people who get it survive it. About 15% of people get very ill and could stay ill for a long time. We are going to be dealing with it for a long time.

Key point #3: SARS-CoV-2 is very contagious, but not as contagious as Measles, mumps or even certain strains of pandemic influenza. It is spread by respiratory droplets and aerosols, not food and incidental contact.

Take home: Social distancing, not touching our faces and good hand hygiene are the key weapons to stop the spread. Masks could make a difference, too, especially in public places where people congregate. Incidental contact is not really an issue, nor is food.

What does this all mean as we return to work and public life? COVID-19 is not going away anytime soon. It may not go away for a year or two and may not be eradicated for many years, so we have to learn to live with it and do what we can to mitigate (reduce) risk. That means being willing to accept *some* level of risk to live our lives as we desire. I can't decide that level of risk for you - only you can make that decision. There are few certainties in pandemic risk management other than that fact that some people will die, some people in low risk groups will die, and some people in high risk groups will survive. It's about probability.

Here is some guidance - my point of view, not judging yours:

1. People over 60 years old are at higher risk of severe disease - people over 70 years old, even more so. They should be willing to tolerate less risk than people under 50 years old and should be extra careful. Some chronic diseases like heart disease and COPD increase risk, but it is not clear if other diseases like obesity, asthma, immune disorders, etc. increase risk appreciably. It looks like asthma and inflammatory bowel disease might not be as high risk as we thought, but we are not sure - their risks might be too small to pick up, or they might be associated with things that put them at higher risk. People over 60-70 years old probably should continue to be very vigilant about limiting exposures if they can.

However, not seeing family - especially children and grandchildren - can take a serious emotional toll, so I encourage people to be creative and flexible. For example, in-person visits are not crazy - consider one, especially if you have been isolated and have no symptoms. They are especially safe in the early days after restrictions are lifted in places like Madison or parts of major cities where there is very little community transmission. Families can decide how much mingling they are comfortable with - if they want to hug and eat together, distance together with masks, or just stay apart and continue using video-conferencing and the telephone to stay in contact. If you choose to intermingle, remember to practice good hand hygiene, don't share plates/forks/spoons/cups, don't share towels, and don't sleep together.

2. Social distancing, not touching your face, and washing/sanitizing your hands are the key prevention interventions. They are vastly more important than anything else you do. Wearing a fabric mask is a good idea in crowded public place like a grocery store or public transportation, but you absolutely must distance, practice good hand hygiene, and don't touch your face. Wearing gloves is not helpful (the virus does not get in through the skin) and may increase your risk because you likely won't wash or sanitize your hands when they are on, you will drop things, and touch your face.

3. Be a good citizen. If you think you might be sick, then stay home. If you are going to cough or sneeze, turn away from people, block it and sanitize your hands immediately after.

4. Use common sense. Dial down the anxiety. If you are out taking a walk and someone walks past you, that brief (near) contact is so low risk that it doesn't make sense to get scared. Smile at them as they approach, turn your head away as they pass, move on. The smile will be more therapeutic than the passing is dangerous. Similarly, if someone bumps into you at the grocery store or reaches past you for a loaf of bread, don't stress - it is a very low risk encounter, also - as long as they didn't cough or sneeze in your face (one reason we wear cloth masks in public!).

5. Use common sense, part II. Dial down the obsessiveness. There really is no reason to go crazy sanitizing items that come into your house from outside, like groceries and packages. For it to be a risk, the delivery person would need to be infectious, cough or sneeze some droplets on your package, you touch the droplet, then touch your face, and then it invades your respiratory epithelium. There would need to be enough viral load and the virions would need to survive long enough for you to get infected. It could happen but it's pretty unlikely. If you want to have a staging station for 1-2 days before you put things away, sure, no problem. You also can simply wipe things off before they come into your house - that is fine, too.

For an isolated family, it makes no sense to obsessively wipe down every surface every day (or several times a day). Door knobs, toilet handles, commonly trafficked light switches could get a wipe off each day, but it takes a lot of time and emotional energy to do all those things and they have marginal benefits. We don't need to create a sterile operating room-like living space. Compared to keeping your hands out of your mouth, good hand hygiene, and cleaning food before serving it, these behaviors might be more maladaptive than protective.

6. There are few absolutes, so please get comfortable accepting some calculated risks, otherwise you might be isolating yourself for a really, really long time. Figure out how you can be in public and interact with people without fear. We are social creatures. We need each other. We will survive with and because of each other. Social distancing just means that we connect differently. Being afraid makes us contract and shut each other out. I hope we can fill that space created by fear and contraction with meaningful connections and learn to be less afraid of each other.



LOWEST RISK



HOME ALONE OR WITH HOUSEMATES

- Stay home as much as possible.
- Try to allow only people you live with into your home.
- Wash your hands.
- If you're sick, stay home and isolate from housemates.

MODERATE RISK



OUTDOOR ACTIVITIES

- Wash your hands and don't touch your face.
- Stay at least 6 feet from people you don't live with.
- Wear a mask.
- Avoid shared surfaces, like swings or benches.

HIGHER RISK



OUTDOOR GATHERINGS

- Wash your hands and don't touch your face.
- Stay at least 6 feet from people you don't live with.
- Wear a mask.
- Don't share food, toys, and other items, and avoid shared surfaces.
- Participate in events like these infrequently.

HIGHEST RISK



INDOOR GATHERINGS

- Wash your hands and don't touch your face.
- Stay at least 6 feet from people you don't live with.
- Wear a mask.
- Don't share food, toys, and other items, and avoid shared surfaces.
- Open windows for better ventilation.
- Try to avoid gathering indoors as much as possible.



VENTILATION: Indoor air circulation thru AC/heat ducts aids in transmission by swirling air currents which keeps the live virus afloat in a room. Open windows & doors to ventilate interiors and hold gatherings outdoors when possible to aid in dispersion of aerosolized virus droplets.

VIRAL LOAD: Viral concentration or the AMOUNT of virus exposure matters in how sick we get and how well our bodies are able to defend against the virus. Limit exposure and keep guests to 10 people or less. If meeting inside, minimize viral conduction by adequate social distancing, wearing face masks and open doors and windows for proper ventilation. If outside on trails, at beaches or in pools, etc. try to avoid large groupings of people. Consider putting off trips to theme parks, fairs or other vacations that involve crowds and tightly packed areas.

WATER FUN: According to the CDC, "There is no evidence that the virus that causes COVID-19 can be spread to people through the water in pools, hot tubs, spaces or water play areas." The agency notes that "proper operation and maintenance (include disinfection with chlorine and bromine) of these facilities should inactivate the virus in the water."

INDIVIDUALIZE: Avoid areas of easy cross-contamination. Avoid buffet-style or self-serving food stations that sit out for extended periods of time or that crowds of people come in contact with. Instead, consider picnic-style gatherings, bringing family food, individually packaged drinks. Use disposable plates, cups, utensils. Avoid communal coffee pots/stations and invest in individualized cups, i.e., Keurig-style brand.

CLEAN: Try to clean personal effects touched multiple times a day typically exposed to public venues—keys, phones, cards, sunglasses, steering wheels, door knobs. Avoid/limit using public restrooms/rest stops; remember transmission occurs through feces easily spread by flushing (releases a flume of aerosolized droplets into the air) or touching (unwashed hands on stall doors, restroom doors).

LIMIT SURFACE AREA CARRIED: Limiting what we wear/carry when leaving our homes limits what the virus can land/live on when we re-enter our homes. Consider dispensing with purses/bags, jewelry, etc. and just carrying essential items (cards/keys/phones) in a pants pocket. Wipe essential items carried, wash hands well and change clothing after re-entry before relaxing, sitting on couches, chairs, beds, etc.

Wellness Wizard—

Coronavirus May Be a Blood Vessel Disease, Which Explains Everything

Many of the infection's bizarre symptoms have one thing in common

Elemental: <https://elemental.medium.com/coronavirus-may-be-a-blood-vessel-disease-which-explains-everything-2c4032481ab2>

Dana Smith, Senior Writer at Elemental at Medium

May 29, 2020

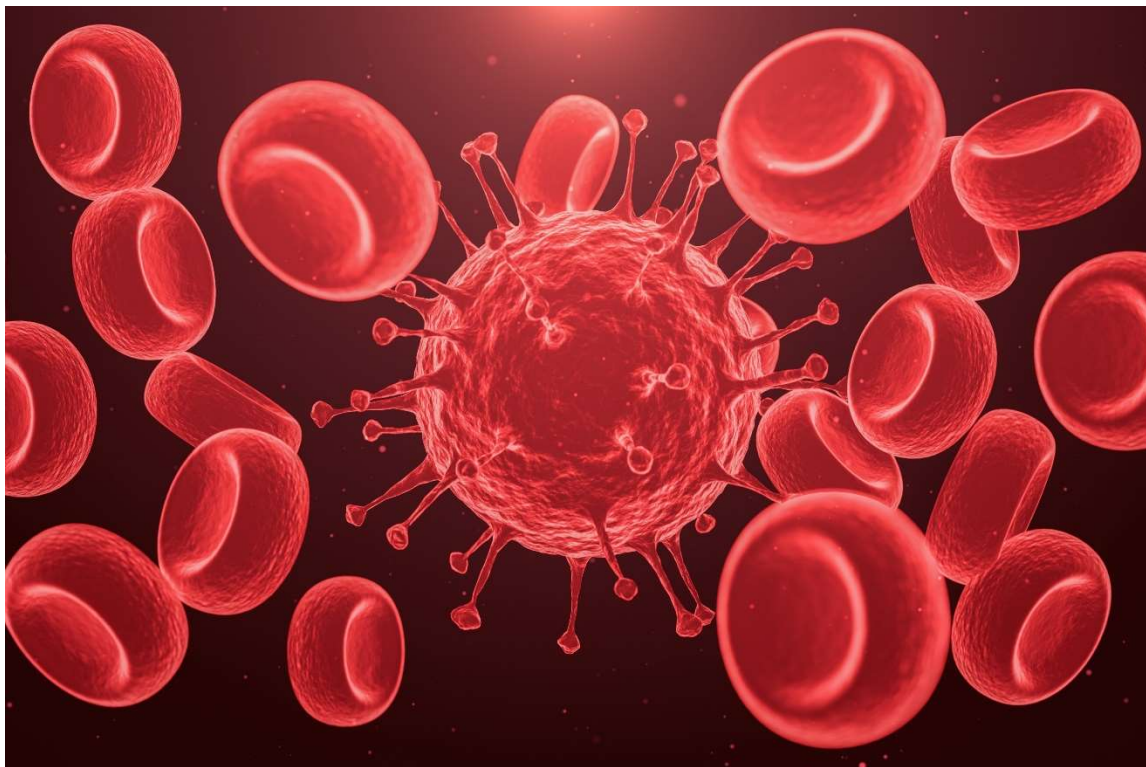


Image: MR.Cole_Photographer/Getty Images

In April, blood clots emerged as one of the many mysterious symptoms attributed to Covid-19, a disease that had initially been thought to largely affect the lungs in the form of pneumonia. Quickly after came reports of young people dying due to coronavirus-related strokes. Next it was Covid toes — painful red or purple digits.

What do all of these symptoms have in common? An impairment in blood circulation. Add in the fact that 40% of deaths from Covid-19 are related to cardiovascular complications, and the disease starts to look like a vascular infection instead of a purely respiratory one.

Months into the pandemic, there is now a growing body of evidence to support the theory that the novel coronavirus can infect blood vessels, which could explain not only the high prevalence of blood clots, strokes, and heart attacks, but also provide an answer for the diverse set of head-to-toe symptoms that have emerged.

“All these COVID-associated complications were a mystery. We see blood clotting, we see kidney damage, we see inflammation of the heart, we see stroke, we see encephalitis [swelling of the brain],” says William Li, MD, president of the Angiogenesis Foundation. “A whole myriad of seemingly unconnected phenomena that you do not normally see with SARS or H1N1 or, frankly, most infectious diseases.”

“If you start to put all of the data together that’s emerging, it turns out that this virus is probably a vasculotropic virus, meaning that it affects the [blood vessels],” says Mandeep Mehra, MD, medical director of the Brigham and Women’s Hospital Heart and Vascular Center.

In a paper published in April in the scientific journal *The Lancet*, Mehra and a team of scientists discovered that the SARS-CoV-2 virus can infect the endothelial cells that line the inside of blood vessels. Endothelial cells protect the cardiovascular system, and they release proteins that influence everything from blood clotting to the immune response. In the paper, the scientists showed damage to endothelial cells in the lungs, heart, kidneys, liver, and intestines in people with Covid-19.

“The concept that’s emerging is that this is not a respiratory illness alone, this is a respiratory illness to start with, but it is actually a vascular illness that kills people through its involvement of the vasculature,” says Mehra.

A respiratory virus infecting blood cells and circulating through the body is virtually unheard of.

A one-of-a-kind respiratory virus

SARS-CoV-2 is thought to enter the body through ACE2 receptors present on the surface of cells that line the respiratory tract in the nose and throat. Once in the lungs, the virus appears to move from the alveoli, the air sacs in the lung, into the blood vessels, which are also rich in ACE2 receptors.

“[The virus] enters the lung, it destroys the lung tissue, and people start coughing. The destruction of the lung tissue breaks open some blood vessels,” Mehra explains. “Then it starts to infect endothelial cell after endothelial cell, creates a local immune response, and inflames the endothelium.”

A respiratory virus infecting blood cells and circulating through the body is virtually unheard of. Influenza viruses like H1N1 are not known to do this, and the original SARS virus, a sister coronavirus to the current infection, did not spread past the lung. Other types of viruses, such as Ebola or Dengue, can damage endothelial cells, but they are very different from viruses that typically infect the lungs.

Benhur Lee, MD, a professor of microbiology at the Icahn School of Medicine at Mount Sinai, says the difference between SARS and SARS-CoV-2 likely stems from an extra protein each of the viruses requires to activate and spread. Although both viruses dock onto cells through ACE2 receptors, another protein is needed to crack open the virus so its genetic material can get into the infected cell. The additional protein the original SARS virus requires is only present in lung tissue, but the protein for SARS-CoV-2 to activate is present in all cells, especially endothelial cells.

“In SARS1, the protein that’s required to cleave it is likely present only in the lung environment, so that’s where it can replicate. To my knowledge, it doesn’t really go systemic,” Lee says. “[SARS-CoV-2] is cleaved by a protein called furin, and that’s a big danger because furin is present in all our cells, it’s ubiquitous.”

Endothelial damage could explain the virus' weird symptoms

An infection of the blood vessels would explain many of the weird tendencies of the novel coronavirus, like the high rates of blood clots. Endothelial cells help regulate clot formation by sending out proteins that turn the coagulation system on or off. The cells also help ensure that blood flows smoothly and doesn't get caught on any rough edges on the blood vessel walls.

“The endothelial cell layer is in part responsible for [clot] regulation, it inhibits clot formation through a variety of ways,” says Sanjum Sethi, MD, MPH, an interventional cardiologist at Columbia University Irving Medical Center. “If that's disrupted, you could see why that may potentially promote clot formation.”

Endothelial damage might account for the high rates of cardiovascular damage and seemingly spontaneous heart attacks in people with Covid-19, too. Damage to endothelial cells causes inflammation in the blood vessels, and that can cause any plaque that's accumulated to rupture, causing a heart attack. This means anyone who has plaque in their blood vessels that might normally have remained stable or been controlled with medication is suddenly at a much higher risk for a heart attack.

“Inflammation and endothelial dysfunction promote plaque rupture,” Sethi says. “Endothelial dysfunction is linked towards worse heart outcomes, in particular myocardial infarction or heart attack.”

Blood vessel damage could also explain why people with pre-existing conditions like high blood pressure, high cholesterol, diabetes, and heart disease are at a higher risk for severe complications from a virus that's supposed to just infect the lungs. All of those diseases cause endothelial cell dysfunction, and the additional damage and inflammation in the blood vessels caused by the infection could push them over the edge and cause serious problems.

The theory could even solve the mystery of why ventilation often isn't enough to help many Covid-19 patients breathe better. Moving air into the lungs, which ventilators help with, is only one part of the equation. The exchange of oxygen and carbon dioxide in the blood is just as important to provide the rest of the body with oxygen, and that process relies on functioning blood vessels in the lungs.

“If you have blood clots within the blood vessels that are required for complete oxygen exchange, even if you're moving air in and out of the airways, [if] the circulation is blocked, the full benefits of mechanical ventilatory support are somewhat thwarted,” says Li.

A new paper published last week in the *New England Journal of Medicine*, on which Li is a co-author, found widespread evidence of blood clots and infection in the endothelial cells in the lungs of people who died from Covid-19. This was in stark contrast to people who died from H1N1, who had nine times fewer blood clots in the lungs. Even the structure of the blood vessels was different in the Covid-19 lungs, with many more new branches that likely formed after the original blood vessels were damaged.

“We saw blood clots everywhere,” Li says. “We were observing virus particles filling up the endothelial cell like filling up a gumball machine. The endothelial cell swells and the cell membrane starts to break down, and now you have a layer of injured endothelium.”

Finally, infection of the blood vessels may be how the virus travels through the body and infects other organs — something that's atypical of respiratory infections.

“Endothelial cells connect the entire circulation [system], 60,000 miles worth of blood vessels throughout our body,” says Li. “Is this one way that Covid-19 can impact the brain, the heart, the Covid toe? Does SARS-CoV-2 traffic itself through the endothelial cells or get into the bloodstream this way? We don't know the answer to that.”

In another paper that looked at nearly 9,000 people with Covid-19, Mehra showed that the use of statins and ACE inhibitors were linked to higher rates of survival.

If COVID-19 is a vascular disease, the best antiviral therapy might not be antiviral therapy

An alternative theory is that the blood clotting and symptoms in other organs are caused by inflammation in the body due to an over-reactive immune response — the so-called cytokine storm. This inflammatory reaction can occur in other respiratory illnesses and severe cases of pneumonia, which is why the initial reports of blood clots, heart complications, and neurological symptoms didn't sound the alarm bells. However, the magnitude of the problems seen with Covid-19 appear to go beyond the inflammation experienced in other respiratory infections.

“There is some increased propensity, we think, of clotting happening with these [other] viruses. I think inflammation in general promotes that,” Sethi says. “Is this over and above or unique for SARS-CoV-2, or is that just because [the infection] is just that much more severe? I think those are all really good questions that unfortunately we don't have the answer to yet.”

Anecdotally, Sethi says the number of requests he received as the director of the pulmonary embolism response team, which deals with blood clots in the lungs, in April 2020 was two to three times the number in April 2019. The question he's now trying to answer is whether that's because there were simply more patients at the hospital during that month, the peak of the pandemic, or if Covid-19 patients really do have a higher risk for blood clots.

“I suspect from what we see and what our preliminary data show is that this virus has an additional risk factor for blood clots, but I can't prove that yet,” Sethi says.

The good news is that if Covid-19 is a vascular disease, there are existing drugs that can help protect against endothelial cell damage. In another *New England Journal of Medicine* paper that looked at nearly 9,000 people with Covid-19, Mehra showed that the use of statins and ACE inhibitors were linked to higher rates of survival. Statins reduce the risk of heart attacks not only by lowering cholesterol or preventing plaque, they also stabilize existing plaque, meaning they're less likely to rupture if someone is on the drugs.

“It turns out that both statins and ACE inhibitors are extremely protective on vascular dysfunction,” Mehra says. “Most of their benefit in the continuum of cardiovascular illness — be it high blood pressure, be it stroke, be it heart attack, be it arrhythmia, be it heart failure — in any situation the mechanism by which they protect the cardiovascular system starts with their ability to stabilize the endothelial cells.”


Mehra continues, “What we're saying is that maybe the best antiviral therapy is not actually an antiviral therapy. The best therapy might actually be a drug that stabilizes the vascular endothelial. We're building a drastically different concept.”

What's New?

△ NOTE: ALL WORKSHOPS, SEMINARS & PRIVATE SESSIONS HAVE BEEN CANCELLED UNTIL FURTHER NOTICE.

△ WELLNESS FOCUS: INFLAMMATORY NATION. Our summer series will explore the concept of inflammation, the roles it plays at the cellular and systemic levels and its importance in the development, maintenance and progression of wound recovery, illness and the chronic diseases that determine our quality and length of life. We will detail its principle drivers as well as strategies to combat and cool these internal fires.

Wellness Bites: GARLIC

 Garlic is a plant in the Allium (onion) family, closely related to onions, shallots and leeks. Its use has been well documented by many major civilizations, including the Egyptians, Babylonians, Greeks, Romans and Chinese. It is thought that garlic is native to Siberia but spread to other parts of the world over 5000 years ago. Although a popular ingredient in cooking due to its strong smell and delicious taste, throughout ancient history the main use of garlic was for its health and medicinal properties.



Manganese, vitamin B6, vitamin C, selenium, fiber, calcium, copper, potassium, phosphorus, iron and vitamin B1, antioxidants.



Scientists now know that most of garlic's benefits are derived from sulfur compounds formed when a fresh clove is chopped, crushed or chewed. Sulfur compounds enter the body from the digestive tract and travel all over the body, where it exerts potent biological effects, improving blood pressure, reducing total/LDL cholesterol levels, preventing blood clots & lowering risk of heart disease. Garlic contains antioxidants that protect against cell damage and aging which may reduce the risk of Alzheimer's disease and dementia & Garlic has been shown to protect against organ damage from heavy metal toxicity (i.e., lead) and related symptoms.

Allicin, the major biologically active component of garlic, in its pure form has been found to exhibit i) antibacterial activity against a wide range of Gram-negative & Gram-positive bacteria (including some multidrug-resistant strains); ii) antifungal activity, particularly against *Candida albicans*; iii) antiparasitic activity, including some major human intestinal protozoan parasites; and iv) antiviral activity.



Garlic consumption can result in bad breath, body odor, stomach issues (heartburn, upset stomach). Caution has been advised for those with bleeding disorders or taking blood-thinning medications.



Garlic burns super easy & chopping into little pieces will cook/burn it even quicker. Crucial rule of thumb: Add garlic halfway through the cooking process, never first!! Try roasting garlic by placing whole head on foil after trimming top 1/4 off to expose tops of cloves. Drizzle 1-2 tablespoons of olive oil over head and wrap with foil. Bake at 400° for ~45 minutes. Continue roasting until deeply golden for a more caramelized flavor. Cool & remove clove from its paper by lightly pushing bottom of clove. Enjoy the heady aroma and easily spreadable, mashable, mixable garlic form.



INEXPENSIVE! NUTRIENT-DENSE! Garlic should be stored at room temperature, preferably in a dry, dark space with plenty of air circulation, never in fridge or freezer. Peel garlic by placing clove on cutting board and smashing downwards with the flat of a knife, which will loosen the skin & allow for easy removal.

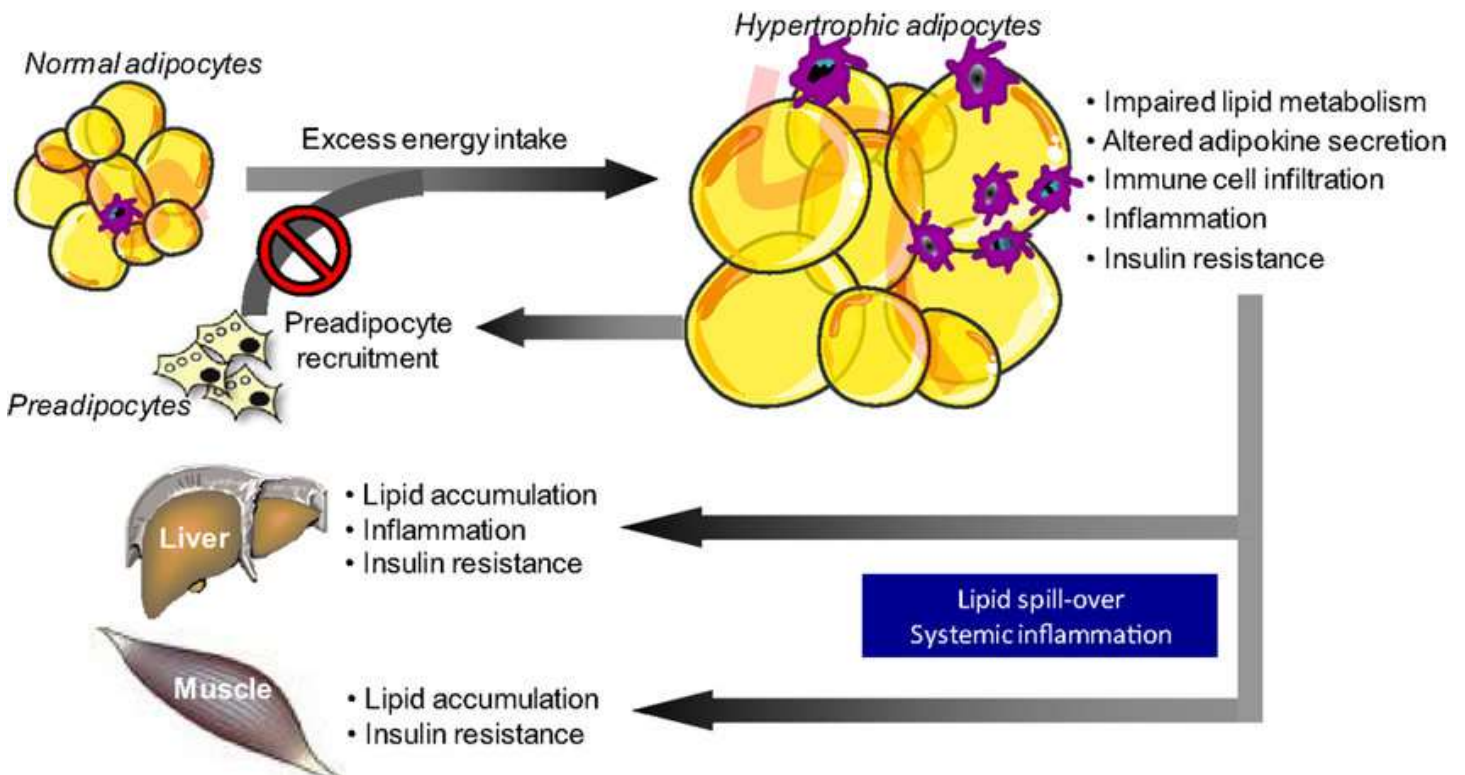
Wellness Focus

Inflammation Nation



Our summer series will introduce the concept of Inflammation as both a defensive mechanism and also an offensive driver of chronic disease. This duality between help and harm is seen over and over within our bodies as we move in and out of an equitable balance through processes such as our “fight or flight” stress response, our “shock” response to trauma and our immune response to compromised systemic protections.

To specifically illustrate this principle, we can pinpoint our last (May) newsletter as we concluded our discussion of the Metabolic Network where we learned about a myriad of harmful effects from the presence of TOO MUCH adipose tissue:



However, fat is essential to the human body; it is, in fact, a necessity in certain quantities for life itself. We all need adequate amounts of fat for energy, body insulation and protection of internal organs. “Essential fat” is needed for temperature regulation/metabolic sustenance; maintenance of integrity of skin and hair; as primary components in cell membranes, insulation for nerve fibers/conduction of nerve impulses, emulsifiers for digestion; and as a starting factor for production of bile (digestion), vitamin D and steroid hormones. Fat is also a starting material for prostaglandins, which help dilation of blood vessels (decreases bp), prevention of clot formation by aggregation of platelets, regulation of smooth muscle contractions and is a significant contributor in inflammatory responses of the body to injury, mediating increased blood flow, chemotaxis (chemical signals summoning WBCs to fight infection) & subsequent dysfunction of tissues and organs.

As we will see with many bodily functions, it is the Amount, not the Presence of these factors that make the difference in positive or negative cumulative effects over time. Over our summer 3-month series we will discover how this holds true for Inflammation.

What is Inflammation?

The word "inflammation" traces back to the Latin for "set afire" and is created by our immune systems to protect against infection, injury or disease. Inflammation is part of the complex biological response of vascular tissues to harmful stimuli, such as pathogens, damaged cells or irritants. It is a protective response involving host cells, blood vessels, proteins and other mediators (lymph, immune reactions). Through inflammation the body is able to 1) eliminate the initial cause of cell injury; 2) eliminate necrotic (dead) cells and tissues resulting from the original insult; and 3) initiate the process of repair.

There can be any number of causes which initiate the inflammatory process:

Infective agents: like bacteria, viruses and their toxins, fungi, parasites (infections)

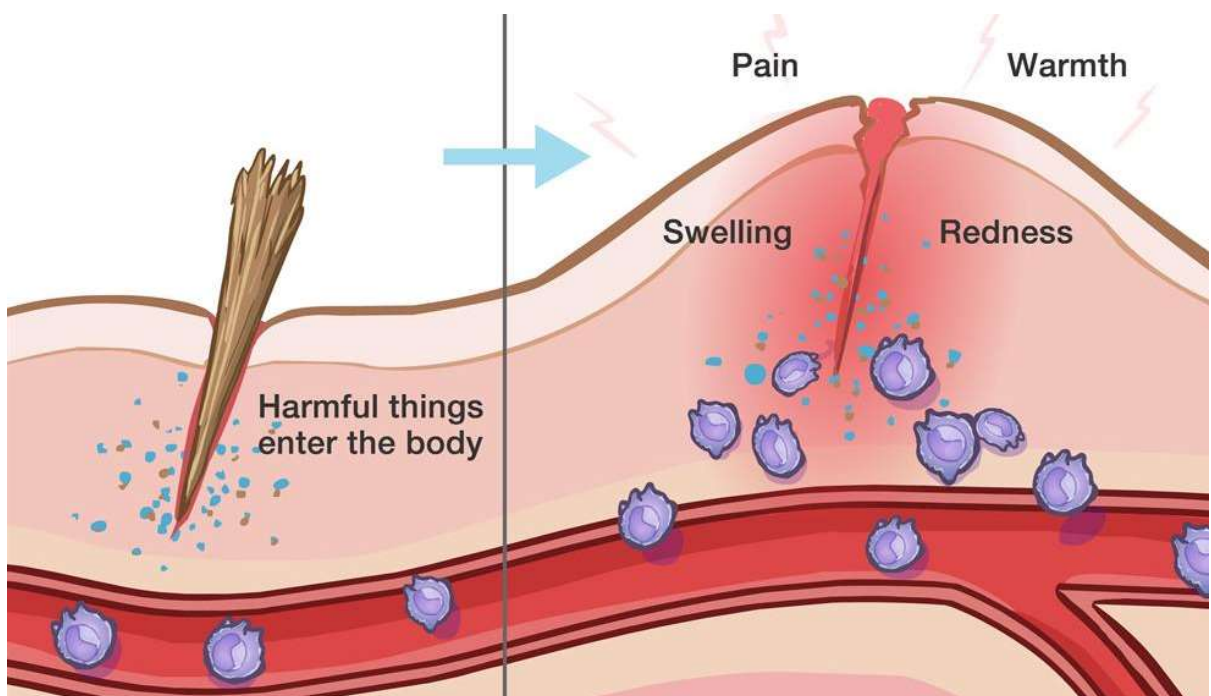
Immunological agents: like cell-mediated and antigen-antibody reactions (hypersensitivity reactions)

Physical agents: like heat, cold, radiation, mechanical trauma

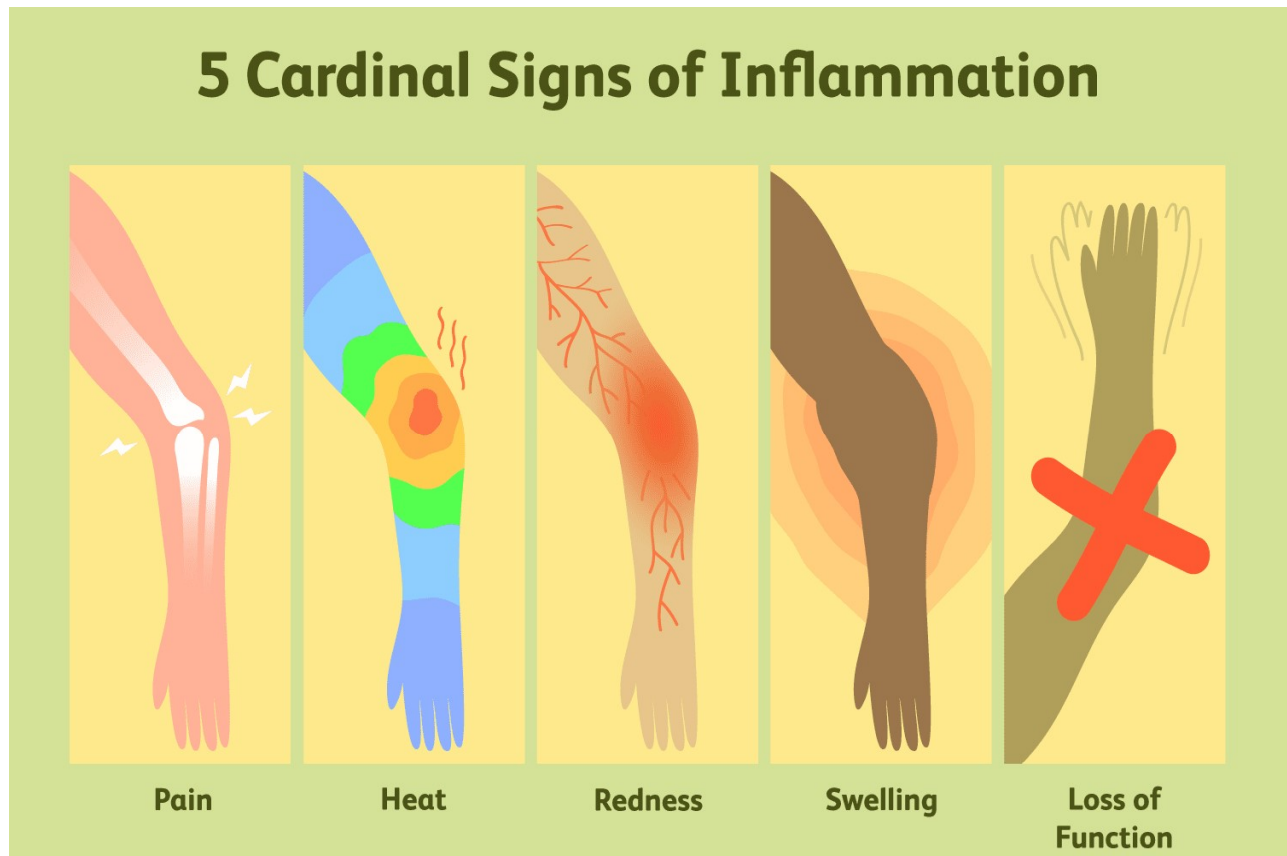
Chemical agents: like organic and inorganic poisons, corrosives, acids, alkalis, reducing agents

Inert materials: like foreign materials/bodies (inhaled, swallowed, penetration)

Tissue necrosis: like damaged or dead cells, tissue (lack of blood supply)



The classical signs of acute, or short-term, inflammation are pain (*dolor*), heat (*calor*), redness (*rubor*), swelling (*tumor*), and loss of function (*functio laesa*).



Pain: This may occur continuously or only when a person touches the affected area.

Heat: Increased blood flow may leave the affected area warm to the touch.

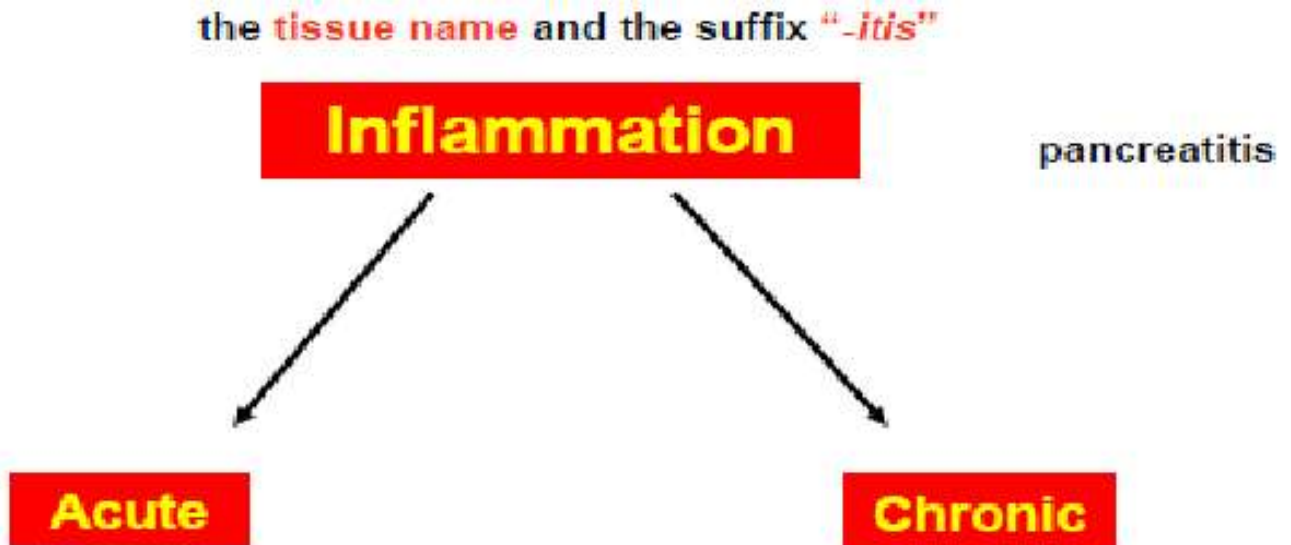
Redness: This happens because of an increase in the blood supply to the capillaries in the area.

Swelling: A condition called edema can develop if fluid builds up.

Loss of Function: There may be difficulty moving a joint, breathing, sensing smell and so on.

These signs are not always present. Sometimes inflammation is "silent," without symptoms. A person may also feel tired, generally unwell and have a fever. Signs of acute inflammation can appear within hours or days, depending on the cause. In some cases, they can rapidly become severe. How they develop and how long they last will depend on the cause, which part of the body they affect and other individual factors. Generally, the acute response is rapid in onset and only lasts for a few days.

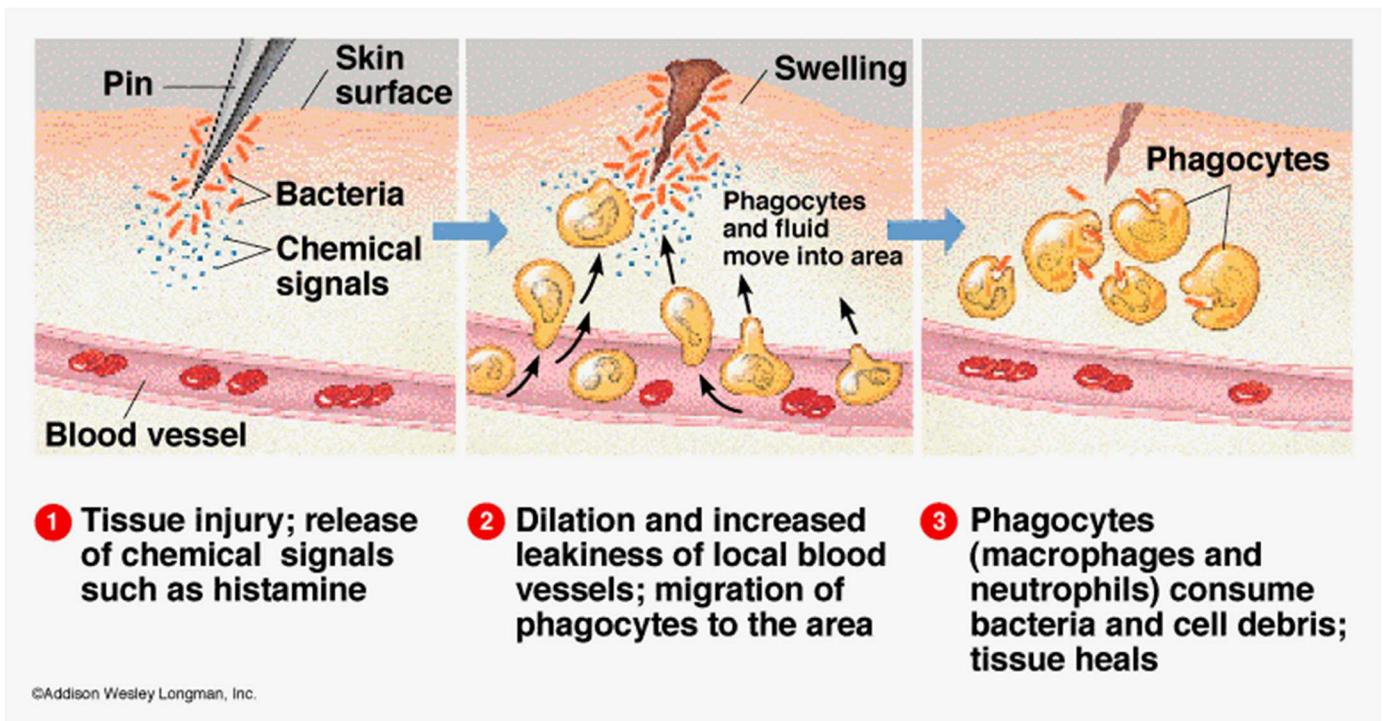
Inflammation is designated by adding the suffix "itis" to the English, Latin or Greek name of the organ affected, e.g., tonsillitis, appendicitis, gastritis....etc., and can be characterized by duration of its presence.



Acute versus chronic inflammation are distinguished by the duration and the type of infiltrating inflammatory cells

Stages of Acute Inflammation:

- 1) Recognition of injurious agent
- 2) Recruitment of leukocytes
- 3) Removal of agent
- 4) Regulation of response
- 5) Resolution (repair)



When the body detects damage or pathogens, the immune system is triggered by chemical signals which in turn catalyze several cascading reactions. Vascular changes occur, leading to 1) small blood vessels (capillaries) enlarging and become more permeable or "leaky" and 2) slowing of the blood in the bloodstream through dilation (enlarging) and fluid exudation (leaky vessels). These changes enable white blood cells (leukocytes) and plasma proteins to reach the injury site, collect and respond to the stimulus more easily. As affected tissues begin to accumulate plasma proteins, fluid begins to build up which results in swelling and subsequent discomfort/pain.

Cellular recruitment and activation also occur as the body releases neutrophils and macrophages, types of leukocyte, which move toward the affected area. Leukocytes contain molecules that help fight pathogens. These white blood cells move from blood vessels into soft tissue at the site of inflammation where they literally "eat" or engulf damaged cells and debris to remove the site of offensive agents. This process is called phagocytosis and includes the lysis or destruction, breakdown of the engulfed particles inside the neutrophils.

Infiltrating white blood cells also release cytokines and growth factors that stimulate tissue repair. Just as there is chemical signaling to activate the immune response, there is also regulation through active suppression of this inflammatory signaling which plays an essential role in repair. In tissues comprised of cells with substantial regenerative capacity, resolution of inflammation is followed by restoration of normal architecture of the tissue. In organs with limited regenerative capacity (such as the heart muscle), extensive necrotic injury heals thru the formation of scar tissue. In all phases of tissue repair, dynamic changes in composition of the healing matrix occur through cellular response and modulation of growth factor signals.