Glutamate and Heart Disease

Mycoplasmas
80,000 x magnification

By Linda Emmanuel
Today, heart disease is the #1 killer of Americans, even more than cancer. There are two ways to get heart disease, but they both have the same common denominator—glutamate.

MSG was discovered and manufactured in 1909. The following is an explanation as to how MSG (monosodium glutamate) introduced into the bloodstream by the consumption of food, leads to heart disease:

**MSG and Heart Disease**

Glutamate is an "excitotoxin" and causes nerve cells to "fire." When glutamate is outside the cell (in the bloodstream), this triggers a chemical reaction.

According to mycoplasma researchers, the body cannot tolerate "free roaming" glutamate and turns glutamate into glutamine. This is "protein modification," (the protein glutamate modified into glutamine). According to Nobel-prize winning researchers, protein modification in the body causes the production of ADMA. According to Wikipedia, the following is a description of Asymmetric dimethylarginine (ADMA):

"ADMA is a naturally occurring chemical found in the blood plasma. It is a metabolic product of continual protein modification processes in the cytoplasm of all human cells."

According to a Lindsey Waggoner documentary, scientists do not understand what is causing "protein modification" in the body. However, they do know that ADMA inhibits the endothelial cells from turning arginine into nitric oxide, but they cannot explain why. (ADMA is a marker in the blood for heart disease.). The following is an explanation as to how nitric oxide is used in the body:

Nitric oxide (NO) is a gas molecule that has a life of just a few seconds. Nitric oxide is manufactured in the endothelium, which is the inner most lining of blood vessels. Endothelium is only one cell thick; therefore, it cannot be seen by the naked eye.

When the smooth muscles that surround the blood vessels constrict, nerve stimulation prompts the endothelium to synthesize (manufacture) and release small amounts of nitric oxide.
When the endothelium is healthy and producing nitric oxide, the endothelium is as smooth as Teflon. When it is not producing and releasing nitric oxide, the endothelial cells become sticky and become vulnerable to bacterial plaque formation.

Nitric oxide:

- Eliminates inflammation in the blood vessels associated with plaque
- Inhibits the formation of plaque in the blood vessels
- Most importantly, dilates and relaxes blood vessels, increasing blood flow
- Now here is the ADMA blocking sequence which over time leads to heart disease:

The following is the sequence:

Glutamate released into the bloodstream → Protein modification (glutamate to glutamine) → ADMA is produced → ADMA inhibits the production of nitric oxide (arginine to nitric oxide) → Dysfunction of endothelial cells (unable to dilate and relax blood vessels) → Cardiovascular Disease (high blood pressure, "sticky" blood vessels, hardening of the arteries, plaque buildup) → Heart attack/stroke → Death.

NOTE: ADMA also inhibits immune system macrophages from producing nitric oxide to fight pathogens.

The following refers to mycoplasma, cell death (the release of glutamate) and the buildup of plaque, leading to heart disease:

**Mycoplasma and Heart Disease**

Setting: The host has a mycoplasma infection. The mycoplasmas penetrate the cell. The cell is defenseless on the inside. (The immune system is on the outside.) Inside the cell, the mycoplasma is parasite, using the cell's internal cell components to replicate.

The damaged cell eventually dies and ruptures, spilling out the newly replicated mycoplasmas, along with the contents of the cell. According to mycoplasma researchers, one of the proteins inside the cell is the amino acid glutamate.

Glutamate is an "excitotoxin" and causes nerve cells to "fire." When glutamate is outside the cell (in the bloodstream), this triggers a chemical reaction. The body turns
glutamate into glutamine (they are in the same family of proteins). The process to turn glutamate into glutamine is as follows:

The body uses/takes an ammonia ion from urea in the bloodstream, which causes the release of a cyanide ion. The cyanide ion then enters a neighboring cell where it focuses on the battery of the cell (mitochondria). At the fourth complex of the mitochondria Kreb’s cycle, the cyanide uptakes the energy generated. This deprives the cell of its energy and results in the (temporary) shutdown of that cell, resulting in chronic fatigue.

The cyanide does not kill the cell. Eventually, the red blood cells carry the cyanide away, and the cyanide is released from the body.

Once the endothelial cells become “sticky”, mycoplasma can adhere to the interior artery walls and colonize. The following are the four stages of a mycoplasma infection and how the mycoplasma infection leads to heart disease and eventually death:

**Stage 1 - Infection**

Mycoplasmas invade and penetrate through the invisible endothelium cells (innermost lining of the blood vessels) Mycoplasmas set up a colony by adhering to the host's tissues and to each other (using protein), then begin forming biofilm (plaque).

FYI: In the case of arterial plaque, biofilm is located between the endothelium lining (innermost layer of the blood vessel) and the smooth muscle (middle layer) of the artery wall. (Arteries have three layers of cells.)

(FACT: Mycoplasmas do not target veins, unless a surgically moved vein replaces an artery, as in bypass surgery. Then that vein, too, becomes vulnerable.)

**Stage 2 - Immune System Responds**

The immune system responds by sending white blood cells, specifically monophages (macrophages), to the site.

Wikipedia’s definition of atherosclerosis:

"Atherosclerosis is a specific form of arteriosclerosis in which an artery wall thickens, as a result of invasion and accumulation of white blood cells (termed fatty streaks early on because of appearance being similar to that of marbled
steak) and containing both living active white blood cells (called inflammation) and remnants of dead cells, including cholesterol and triglycerides, eventually calcium and other crystalized materials, within the outer most and older plaque."

Macrophages are the warriors and cleanup crew of the immune system. They gobble up pathogens, dead cells, etc. They respond to the pathogenic mycoplasmas, which are attacking the host.

Stage Three - Formation of Plaque and Fibrous Cap

As the colony grows, more and more white blood cells (WBC) appear at the site of infection, burrowing into the layers of plaque, gobbling up low density lipoproteins (LDL) in the process. The macrophages get so bloated, that they cannot handle the overload and die. (Scientists call these macrophages "foam cells"). They then become part of the biofilm.

As the colony grows (plaque builds), the inside of the blood vessel (lumen) narrows. The human body then responds by widening the blood vessel. (FYI: Over time, if the artery enlarges to 2 to 3 times the usual diameter, the walls become weak enough that just the stress of the pulse can lead to sudden hemorrhage, which leads to rapid death.)

Over time, a fibrous cap is formed on top of the plaque, which is made up of fibrous connective tissue. It contains macrophages, smooth muscle cells, foam cells, lymphocytes, collagen and elastin. According to Wikipedia:

".....increased macrophage activity, macrophage-enzymes erode away the fibrous membrane beneath the endothelium so that the cover separating the plaque from blood flow in the lumen becomes thin and fragile."

Stage Four - Heart Attack or Stroke

Over time (decades), the fibrous cap is now thin and fragile, which makes it even more susceptible to rupture.

The artery walls, too, have finally failed to keep up with the growth of the plaque, plus the continuous rupturing and clotting of the plaque. The lumen is now narrowing (stenosis), and the threat of total blockage is looming on the horizon. Stretched dangerously thin, the blood vessel walls can also rupture, causing internal bleeding.
(FYI: When a blood vessel is injured, the body uses platelets and fibrin to form a blood clot to prevent blood loss. A clot can break free and begin to travel around the body.)

FACT: If the fibrous cap ruptures, the body immediately sends platelets to close up the lesion. The rupture may release a shower of debris into the bloodstream. The body forms clots both on the particles (debris) and over the rupture.

According to Wikipedia:

"Mechanical stretching and contraction of the artery, with each heartbeat, i.e., the pulse, results in rupture of the thin covering membrane (fibrous cap), spewing clot-promoting plaque contents into the bloodstream."

If the clot is large enough over the lesion (rupture), it can block all blood flow in that blood vessel, resulting in either a heart attack or a stroke. The clot can also travel to smaller arteries and cause blockage, resulting in cell death (lack of oxygen).

Blood flow (oxygen) blocked to the brain will result in a stroke.

**SYMPTOMS OF MSG**

- Headache
- Fatigue
- Flushing
- Sweating
- Numbness and tingling or burning in the face, neck and other areas
- Rapid fluttering heartbeats
- Chest pain
- Nausea

What food manufacturers disguise MSG as on packaging (label):

1. Monosodium glutamate or sodium glutamate
2. Sodium 2-aminopentanedioate
3. Glutamic acid, monosodium salt, monohydrate
4. L-Glutamic acid, monosodium salt, monohydrate
5. L-Monosodium glutamate monohydrate
6. Monosodium L-glutamate monohydrate
7. MSG monohydrate
8. Sodium glutamate monohydrate
9. UNII-W81N5U6R6U
10. Flavour Enhancer

NOTE: Our government allows manufacturers to list MSG as “natural flavors” if the sodium ion is missing from MSG. However, glutamate is still dangerous without the salt.

FACT: The Pakistan government banned MSG from being used in food products in January 2018

CONCLUSION

Mycoplasma has been around forever. Before MSG was introduced (1909), people with severe tooth decay were susceptible to contracting a bacterial infection if the bacteria entered the bloodstream. The host would have died long before heart disease set in because of poor hygiene, contaminated water, poor nutrition, and the lack of effective treatments.