2023-12-09 Brief | Lauri Harris

Cell Production of Wrong Proteins Cause Variety of Diseases

Purpose: Demonstrate evidence of Covid19 injections producing the wrong proteins which then induce a wide variety of diseases. The exact mechanism a large portion of the world accepted as “treating” covid was well known to cause harm to cells and the human body.

The disruption of protein production from life sustaining work is a mechanism of harm within the shots. Some will want to say that the adverse events and deaths are from multiple causes and can therefore not all be from the shots, but the shots carry mRNA to cells throughout the body. Depending on which cells uptake the mRNA, that cell (liver cell, heart cell, pancreas cell, blood vessel, cell, brain cell,….) will stop making life sustaining proteins and begin to make a toxic one(s). But since each cell is its own imperfect protein factory any of the cells could make mistakes and make other toxic or mis-folded proteins.

The lack of the correct protein or correctly folded protein being generated causes a wide variety of diseases. The production of the wrong protein or mis-folded protein causes a wide variety of diseases. Useless wrong proteins building up within cells cause disease. The wrong protein can cause birth-defects. The wrong protein production can cause cancers. Misfolded proteins can cause neurodegenerative diseases.

Proteins made by cells provide for every process such as proper processing of glucose or even transport of oxygen to the cells. Fetuses create organs and limbs based on the correct protein being present at the proper point during gestation. The Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) protein disfunction causes thick, sticky mucus to be produced in every organ of the body that makes mucus causing blockages and trapping germs, leading to infections. Some people with Marfan syndrome make too little fibrillin-1 protein.

Protein mis-folding is believed to be the primary cause of Alzheimer's disease, Parkinson's disease, Huntington's disease, Creutzfeldt-Jakob disease, cystic fibrosis, Gaucher's disease and many other degenerative and neurodegenerative disorders.

Amyloidosis is a rare disease that occurs when a protein called amyloid builds up in organs. This amyloid buildup can make the organs not work properly. Organs that may be affected include the heart, kidneys, liver, spleen, nervous system and digestive tract. Protein mis-folding resulting in intracellular pre-amyloid oligomer (PAO) accumulation is sufficient to cause cardiomyocyte death and heart failure.

It has taken 60 years for “science” to understand the mechanism of harm engaged by Thalidomide. The mechanism of harm was altering production of proteins inside human cells which lead to the malformations suffered by babies impacted with Thalidomide. The harms of altering protein production within cells was well known. Covid19 shots alter protein production in cells. So.

References with short synopsis of each:

Proteins are critical for healthy bodily functions both within cells and throughout the entire body. All chemical processes in living organisms need enzymes, and all enzymes are proteins. Proteins are polymers made up of amino acids. Proteins are involved in almost all the processes taking place in our body. A summary of the functions performed by proteins is as follows:

- As enzymes, proteins are required for all chemical processes in living organisms.
- As hormones and cellular receptors, proteins are needed for cellular signaling and coordination.
- As transport channels, proteins are needed for the entry of ions and larger-sized particles into the cells.
- Proteins, as components of cytoskeleton, maintain the shape of cells.
- Spindle fibers are protein fibers that are needed for cell division.
- Hemoglobin and myoglobin are the proteins required for oxygen transport.
- Albumin and other plasma proteins are needed for the transport of lipids, medications, and other substances in the blood.
- Contractile proteins are needed for muscle contraction.
- Antibodies are the proteins that protect our bodies from harmful disease(s).
- Plasma proteins maintain fluid balance in our body.
- Proteins regulate gene expression.

https://alevelbiology.co.uk/notes/functions-of-proteins/

One example of a complex set of proteins which govern the use of insulin within the body:

https://youtu.be/VbwRYFMPZS4?si=zx8UKeIl7Rm6edUc

This document describes how the cell converts the information carried in an mRNA molecule into a protein molecule. Most genes in a cell produce mRNA molecules that serve as intermediaries on the pathway to proteins.

https://www.ncbi.nlm.nih.gov/books/NBK26829/

The consequences of erroneous protein synthesis can include death to cells and the human. Errors in protein synthesis disrupt cellular fitness, cause disease, and shape gene and genome evolution. Misfolded proteins underlie a broad array of neurodegenerative diseases, and misincorporation of amino acids during translation may be a causative factor in the pathology of multiple sclerosis and ALS. Besides amino-acid misincorporations, sources of errors are transcription errors, aberrant splicing, premature termination, faulty post-translational modifications, and kinetic missteps during folding. Protein synthesis errors may also produce polypeptides displaying a *gain of toxic function*. The error may confer an alternate or pathological function on an otherwise normal, folded protein. More often, errors disrupt folding, and the misfolded molecule may be toxic. In this context, "toxic" simply means harmful and does not specify the modality or severity of the harm. Misfolded proteins may destabilize membranes, steal quality-control bandwidth from essential proteins, and induce chronic stress. Misfolded protein cytotoxicity has been studied extensively as a contributor to neurodegenerative disease. It has become increasingly clear that at the molecular level, misfolding-associated disease often reflect gains of toxic function rather than losses of function.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2764353/#:~:text=Errors%20in%20protein%20synthesis%20disrupt,shape%20gene%20and%20genome%20evolution.

Subclinical protein shortage can result in a number of illnesses that are not often linked with protein deficiencies. For example, subclinical protein shortages have been reported to be responsible for neurological problems such as depression and anxiety, impaired memory, confusion, and irritability.

https://staminacomfort.com/what-diseases-can-misfolded-proteins-cause#toc-heading-5

There are 20,000 to over 100,000 unique types of proteins within a typical human cell. Why so many? Proteins are the workhorses of the cell. Each expertly performs a specific task. Some are structural, lending stiffness and rigidity to muscle cells or long thin neurons, for example. Others bind to specific molecules and shuttle them to new locations, and still others catalyze reactions that allow cells to divide and grow. This wealth of diversity and specificity in function is made possible by a seemingly simple property of proteins: they fold. A protein's function depends on its shape, and when protein formation goes awry, the resulting misshapen proteins cause problems that range from bad, when proteins neglect their important work, to ugly, when they form a sticky, clumpy mess inside of cells. Current research suggests that the world of proteins is far from pristine. Protein formation is an error-prone process, and mistakes along the way have been linked to a number of human diseases.

https://sitn.hms.harvard.edu/flash/2010/issue65/

Protein misfolding is believed to be the primary cause of Alzheimer's disease, Parkinson's disease, Huntington's disease, Creutzfeldt-Jakob disease, cystic fibrosis, Gaucher's disease and many other degenerative and neurodegenerative disorders.

The accumulation of misfolded proteins (e.g. mutant or damaged proteins) triggers cellular stress responses that protect cells against the toxic buildup of such proteins. However, prolonged stress due to the buildup of these toxic proteins induces specific death pathways.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3970707/#:~:text=Accumulation%20of%20these%20proteins%20in,death%20%5B1–10%5D

If a cell has damaged DNA, the likelihood of producing faulty proteins is higher. The daughter cells of such a damaged parent cell would also produce faulty proteins that might eventually become cancerous.

https://courses.lumenlearning.com/suny-biology1/chapter/cancer-and-the-cell-cycle/#:~:text=If%20a%20cell%20has%20damaged,that%20might%20eventually%20become%20cancerous.

At molecular level, misfolded proteins can propagate changes into the native proteins, modifying the function of the proteins inducing cellular stress and damage. Protein misfolding can be transmitted from one cell to another which may propagate the pathology throughout the affected tissue.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3175247/#:~:text=At%20molecular%20level%2C%20misfolded%20proteins,pathology%20throughout%20the%20affected%20tissue.

By changing a gene’s instructions for making a protein, a variant can cause a protein to malfunction or to not be produced at all. When a variant alters a protein that plays a critical role in the body, it can disrupt normal development or cause a health condition.

https://medlineplus.gov/genetics/understanding/mutationsanddisorders/mutationscausedisease/#:~:text=By%20changing%20a%20gene's%20instructions,or%20cause%20a%20health%20condition.

The way proteins misfold and aggregate is closely associated with the accumulation of toxic proteins that cause many neurodegenerative diseases. Alzheimer’s involves the presence of two misfolded proteins in the brain: beta-amyloid protein and tau protein. Parkinson’s disease is typically characterized by the accumulation of the alpha-synuclein protein in the brain.

Huntington's disease is caused by an abnormal form of the huntingtin protein with an extended glutamine tract. Misfolded huntingtin protein forms amyloid aggregates that build up in neurons which in turn leads to neuronal dysfunction and cell death.

https://www.bmglabtech.com/en/blog/misfolded-proteins-and-neurodegenerative-diseases/#:~:text=It%20is%20therefore%20not%20surprising,or%20impaired%20function%20and%20disease.

Protein misfolding is thought to be the root cause of Alzheimer's disease, Parkinson's disease, Huntington's disease, Creutzfeldt-Jakob disease, cystic fibrosis, Gaucher's disease, and many other neurodegenerative illnesses. Protein misfolding may also play a role in diabetes, heart disease, and cancer.

Point-nonsense mutations have been linked to a variety of illnesses, including:

- Cystic fibrosis caused by the G542X mutation in the cystic fibrosis transmembrane conductance regulator (CFTR)
- Beta thalassaemia (β-globin)
- Hurler syndrome.
- Dravet Syndrome

Each protein has its own unique shape. If the temperature or pH of a protein's environment is changed, or if it is exposed to chemicals, these interactions may be disrupted, causing the protein to lose its three-dimensional structure and turn back into an unstructured string of amino acids. When a protein loses its higher-order structure, but not its primary sequence, it is said to be denatured. Denatured proteins are usually non-functional.

https://www.khanacademy.org/science/biology/macromolecules/proteins-and-amino-acids/a/orders-of-protein-structure#:~:text=Denaturation%20and%20protein%20folding&text=If%20the%20temperature%20or%20pH,unstructured%20string%20of%20amino%20acids.

Amyloidosis (am-uh-loi-DO-sis) is a rare disease that occurs when a protein called amyloid builds up in organs. This amyloid buildup can make the organs not work properly. Organs that may be affected include the heart, kidneys, liver, spleen, nervous system and digestive tract.

https://www.mayoclinic.org/diseases-conditions/amyloidosis/symptoms-causes/syc-20353178#:~:text=Amyloidosis%20(am%2Duh%2Dloi,nervous%20system%20and%20digestive%20tract.

Patients with CF suffer from a cystic fibrosis transmembrane conductance regulator (CFTR) gene mutation, which causes the CFTR protein to malfunction. The CFTR protein is located in every organ of the body that makes mucus; including the lungs, liver, pancreas, intestines and sweat

glands. It is also present in many other cells in the body. When the CFTR protein isn't working correctly, thick, sticky mucus is produced that causes blockages and traps germs, leading to infections.

https://www.lung.org/lung-health-diseases/lung-disease-lookup/cystic-fibrosis/learn-about-cystic-fibrosis

Alpha-1 is a genetic disorder that affects the lungs and sometimes the liver. Even though it is one of the most common genetic disorders, Alpha-1 can be hard to diagnose. One challenge is that most people with Alpha-1 are healthy for at least the first few decades of their lives. For many, symptoms do not appear until middle adulthood. Another challenge is that the effects of the disorder look a lot like other conditions. Lung symptoms can mimic asthma, bronchitis, or smoking-induced emphysema. Liver symptoms can mimic cirrhosis. This often leads to misdiagnosis and a delay in treatment. In the United States, more than 90% of people with Alpha-1 never learn that they have it.

The affected gene in Alpha-1 is SERPINA1, on chromosome 14. This gene codes for a protein called alpha-1 antitrypsin (AAT). People with the disorder have two non-working copies (alleles) of the gene; they make little or no working AAT protein.

AAT protein is normally made in the liver and released into the blood stream. From there, it can travel throughout the body—most importantly to the lungs. When we breathe in irritants like viruses or smoke, AAT protects the lungs from damage.

In people with Alpha-1, very little or no AAT protein makes it to the lungs. The lungs are left unprotected. Some people who have Alpha-1 make a sticky version of AAT protein that builds up in the liver. Not only do their lungs become damaged, but the sticky AAT protein can also harm the liver.

https://learn.genetics.utah.edu/content/genetics/alpha1

The data confirm that protein misfolding resulting in intracellular PAO accumulation is sufficient to cause cardiomyocyte death and heart failure.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2559970/

Protein disorder occurs along a continuum. At one end of the spectrum lie proteins like p21, which fold on contact with other proteins. At the other end are ones that remain limp and floppy, like wet noodle strands, never taking on a shape. Researchers still don't know how this range corresponds to their versatile functions, but being more like a string than like a lump with keyholes means that a protein can make many contacts with other molecules to regulate the

network of signals that drives the cell. “You have all these on-off switches for all kinds of functions,” said Dunker.

But even though IDPs in multicelled organisms make up 30 to 50 percent — depending on the organism — of the proteins that genes are able to make, it turns out that at any given moment, they exist in the cell in only tiny amounts. Babu made this discovery in 2008, after a researcher in his lab raised a niggling question: If these unfolded proteins were in fact so common, and if many of them floated around the cell like limp spaghetti, why weren’t they getting all tangled up, or causing trouble in the cell by tangling up other molecules? When they examined a database of around 5,000 human proteins, they found that most unstructured proteins were expressed in small quantities and quickly destroyed after they had done their job.

The reason cells regulate their production so tightly and make sure they turn over so quickly is that IDPs pack a huge punch, Babu said. Having too many would be like having a glut of upper management — with too many people shouting commands, productivity grinds to a halt. Extend that logic to a cell, though, and things can get ugly: Because IDPs regulate how different components of the cell communicate with one another, having extra copies floating around could leave them sending signals that shouldn’t get sent. “These proteins are so dangerous that you can’t afford not to regulate them,” Babu said.

https://www.quantamagazine.org/how-disordered-proteins-are-upending-molecular-biology-20170118/

“The key now is that we need to understand how these proteins are functioning in biology,” said Peter Wright, a structural biologist at the Scripps Research Institute in La Jolla, California. In response to the recent revelations, an international group of researchers has launched a project called the Human Dark Proteome Initiative to study how disordered proteins cause disease. Scientists know that they still have much to learn about what these shape-shifters are up to. “It’s a re-envisioning of cell biology,” said Madan Babu, a molecular biologist at the University of Cambridge.

https://www.quantamagazine.org/how-disordered-proteins-are-upending-molecular-biology-20170118/

The human dark proteome consists of the approximately one third of proteins in the human proteome that are disordered and therefore “unseen” by traditional structural biology methods. Recent discoveries and technological developments create unprecedented opportunities to advance this important new field of science and will profoundly impact our understanding of and ability to combat devastating diseases such as cancer, diabetes, infectious disease, cardiovascular disease, and neurodegenerative disorders. The seven-point mission of the HDPI is to:

https://darkproteome.wordpress.com/about/mission/

Errors in protein synthesis disrupt cellular fitness, cause disease phenotypes and shape gene and genome evolution. Experimental and theoretical results on this topic have accumulated rapidly in disparate fields, such as neurobiology, protein biosynthesis and degradation and molecular evolution, but with limited communication among disciplines. Here, we review studies of error frequencies, the cellular and organismal consequences of errors and the attendant long-range evolutionary responses to errors. We emphasize major areas in which little is known, such as the failure rates of protein folding, in addition to areas in which technological innovations may enable imminent gains, such as the elucidation of translational missense error frequencies. Evolutionary responses to errors fall into two broad categories: adaptations that minimize errors and their attendant costs and adaptations that exploit errors for the organism's benefit.

https://www.nature.com/articles/nrg2662

The affected gene in Marfan syndrome is FBN1, on chromosome 15. It codes for a large protein called fibrillin-1. People with Marfan syndrome have one non-working copy (allele) of FBN1 and one healthy copy. They make a mix of healthy and non-working protein.

Cells that build connective tissue make fibrillin-1 protein and release it into the space around them. Here, many molecules of fibrillin-1 link together to make long, thread-like microfibrils. The main job of microfibrils is to make connective tissue strong and elastic. Their secondary job is to help control growth and development.

Some people with Marfan syndrome make too little fibrillin-1 protein, and they have too few microfibrils. Other people make a combination of healthy and non-working fibrillin-1 proteins. They form microfibrils that can't do their job very well.

Not everyone who inherits a non-working copy of the FBN1 gene has Marfan syndrome. Some versions cause closely-related connective tissue disorders.

https://learn.genetics.utah.edu/content/genetics/marfan

It has taken 60 years for “science” to understand the mechanism of harm engaged by Thalidomide. The mechanism of harm was altering production of proteins inside human cells which lead to the malformations suffered by babies impacted with Thalidomide. Babies were born with hands and feet growing from their torsos. Some babies were missing internal organs or even eyes.

https://www.dana-farber.org/newsroom/news-releases/2018/after-60-years--scientists-uncover-how-thalidomide-produced-birth-defects/

Dec 7, 2023 C&C “Nonsense” Substack article regarding “The ‘vaccine’ creates stochastic [completely random] proteins one third of the time. In one-third of cells, not people,…”

https://open.substack.com/pub/coffeeandcovid/p/nonsense-thursday-december-7-2023?r=w2wxs&utm_campaign=post&utm_medium=email

The researchers discovered that a necessary ingredient in the mRNA vaccines (1 methylpseudouridine) has an unfortunate side-effect: it messes up RNA translation *one-third of the time* by slipping a gear every so often. Instead of making the intended *spike protein*, these tiny mistranslational slip-ups create … other things. Other *kinds* of proteins. New ones. And there’s no way at all to predict what *kind* of protein it will create. It’s *stochastic* (completely random). The ‘vaccine’ creates stochastic proteins *one third of the time.* In one-third of *cells.*

https://www.nature.com/articles/s41586-023-06800-3?utm_source=substack&utm_medium=email

Yes, indeed. I saw just a spate of increase in birth defects. I saw a state of increased miscarriages, also known as spontaneous abortions, as you know. Not to confuse our audience, who we don’t have your knowledge – miscarriages, we call spontaneous abortions, which have nothing to do with an induced abortion. And then I saw a really dramatic increase in first- and second-trimester loss, fetal death after 20 weeks. I saw a really significant increase in really abnormal reactions, very unusual autoimmune diseases, significant increase in growth restriction, peculiar appearance of placentas, which seemed to follow a kind of an instant pattern recognition.

Ob/Gyn Warns of Possible Links Between mRNA Injections, Miscarriages and Malformations - DailyClout