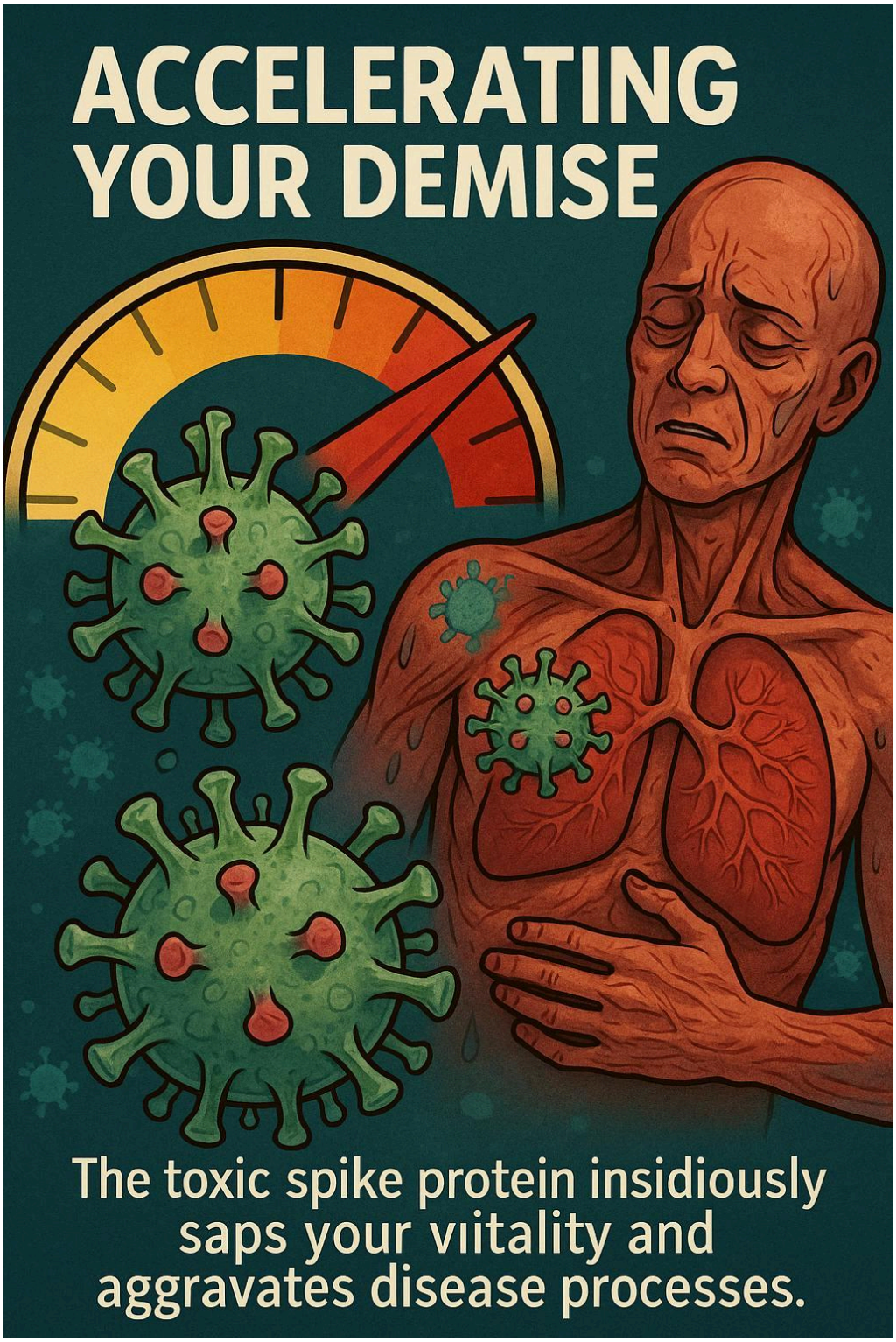
Seven Articles Everyone Needs to Read Regarding How the SARS-CoV-2 Spike Protein Was Weaponized With Vast Similarities to the Toxic Proteins Found in Snake Venom

Article #1: Accelerating Your Demise



By John Leake

The toxic spike protein insidiously saps your vitality and aggravates disease processes.

When I was a kid growing up in Texas I was fascinated by rattlesnakes, whose venom is absolutely devastating to every kind of tissue in the body. Once a friend shot a rattlesnake. While holding up the dead serpent to measure its length in comparison to his height, he accidentally dropped it and one of the fangs brushed his forearm. It didn't appear to break the skin, so we were all surprised a few minutes later when his forearm began to swell up like a balloon.

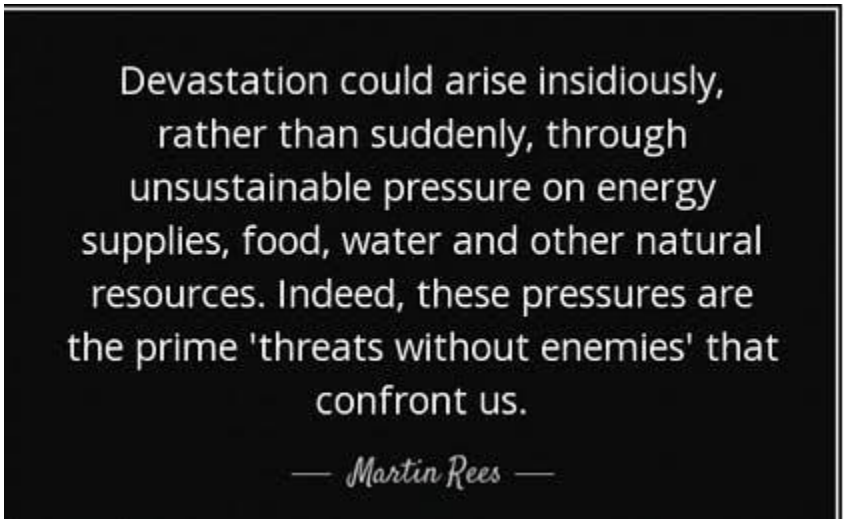
I once asked a chemist, "Why does the rattlesnake venom do so much damage to the skin, muscle, and blood?"

"Because it contains dozens of toxic proteins," he replied.

Toxic proteins. At the time I found this puzzling, as I'd always thought that proteins are the building blocks of life. What does it mean that some proteins can demolish the building blocks of life?

Some toxic proteins—like the cocktail contained in rattlesnake venom—are fast acting. Others—like the genetically engineered spike protein in SARS-CoV-2 and the mRNA vaccines derived from it—seem to work slowly and insidiously. The protein kills you not with a dramatic blow, but by aggravating and accelerating the inflammation and degeneration of your cells and organs. We are all going to die. The only questions are when and how much will the quality of our lives be impaired by sickness before we die.

Spike protein poisoning partly resembles the insidious malady of **syphilis**, which 19th century physicians called the "Great Imitator" due to its wide range of variable clinical symptoms.



The insidiousness of spike protein toxicity is what has enabled the villains who created it to obscure the fruits of their infernal work. In my own social circle, I know dozens of people between the ages of 50-75 who were in excellent health until 2022. About a year after they received the COVID-19 mRNA vaccines, they precipitously declined with diseases that we associate with senescence—that is, strokes, heart trouble, rapidly advancing cancers, and syndromes that strongly resemble Parkinson's

and Alzheimer's. While these disease processes may have already been underway, it appears that the spike protein aggravates and accelerates them.

Thus, a man who would have died of a heart attack at 70 instead died at 54 after receiving his third shot because inflammation and atherosclerotic plaques in his coronary arteries were amplified by the shots.

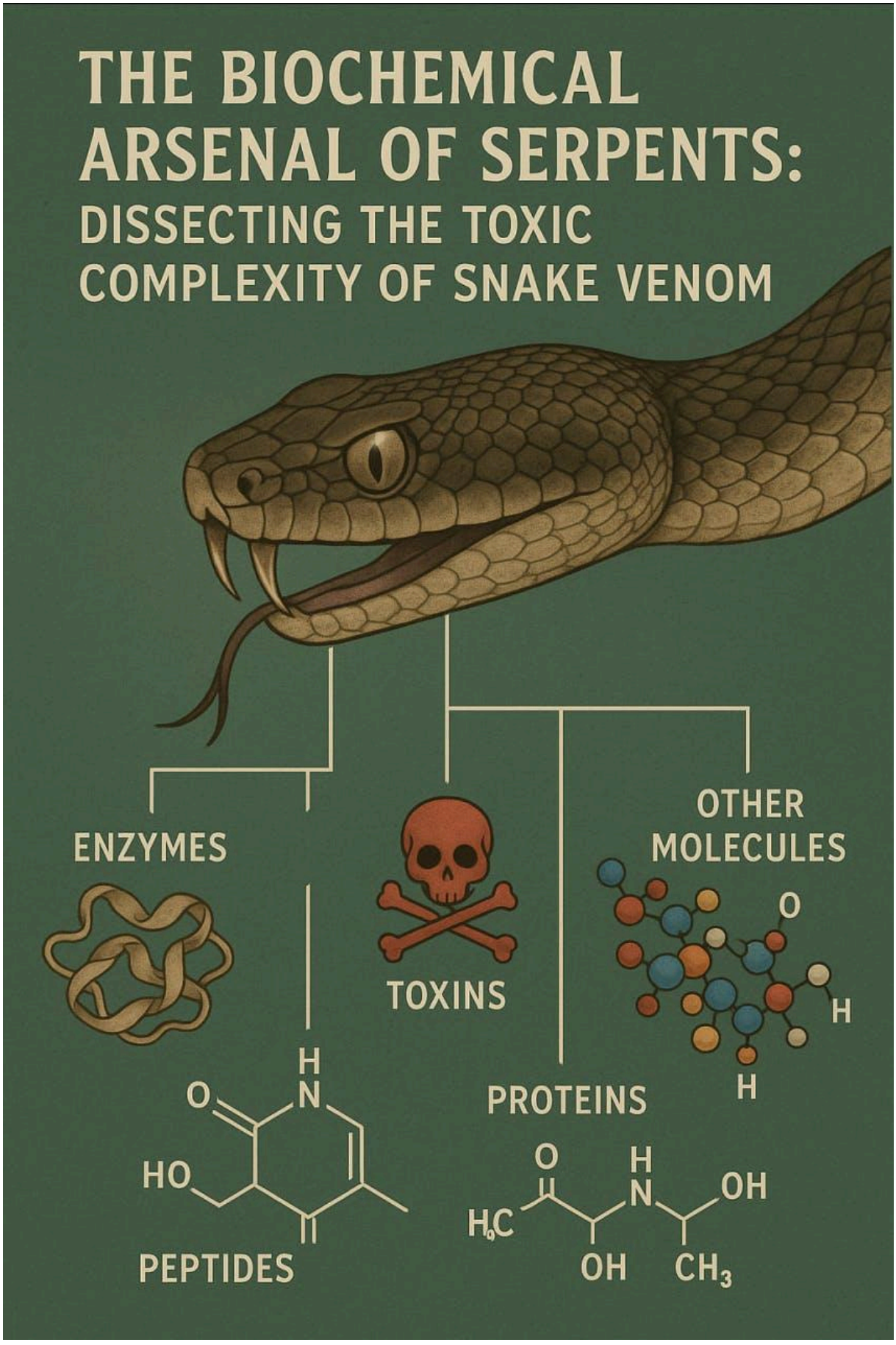
When suspicion was raised about the vaccines he received in 2021 and early 2022, the typical response is that some unfortunate men die of heart attacks in their fifties, which isn't *that* young.

The only way this obfuscation will end will be with the development and widespread availability of rapid testing for the presence of spike protein in the blood and tissues. The presence and quantity of the protein can be analyzed in conjunction with analyzing associated disease processes. Ending this obfuscation is the first step to finding a cure to the problem.

Note the stunning preposterousness that only a handful of undercapitalized, independent researchers like Dr. Peter McCullough and his colleagues are working on solving this problem.

Like autism, the problem of insidious spike protein poisoning now poses a major threat to national security. The American people cannot afford the increasing disease burden that is already upon us.

Article #2: The Biochemical Arsenal of Serpents: Dissecting the Toxic Complexity of Snake Venom



Snake venom is among the most intricately evolved biochemical weapons in the natural world. Composed of a diverse array of toxic proteins, peptides, and enzymes, it is designed not merely to immobilize prey, but to begin digesting it from within and defend the serpent from threats. The complexity of venom reflects millions of years of evolutionary arms races, with different snake families such as Elapidae (e.g., cobras and kraits) and Viperidae (e.g., rattlesnakes and vipers) tailoring their venom to specific prey, environments, and strike strategies. These venoms are not monolithic in their effects. Instead, they deploy a multifaceted assault on the body through mechanisms that affect the nervous system, cardiovascular system, blood clotting pathways, and cellular integrity. Venom's biochemical sophistication allows for multiple modes of toxicity—neurotoxic, hemotoxic, myotoxic, cytotoxic, nephrotoxic, and cardiotoxic—often in overlapping, synergistic fashion. Understanding these components not only helps in clinical management of envenomation but also opens the door to therapeutic breakthroughs in medicine.

One of the major protein families in venom are Phospholipases A_2 (PLA_2), enzymes that destroy phospholipid membranes of cells, resulting in tissue destruction, inflammation, and muscle breakdown. These enzymes have subtypes like Asp49 and Lys49, which exhibit varying degrees of catalytic and membrane-disruptive functions. PLA_2s are notorious for inducing myonecrosis, hemolysis, edema, and even triggering systemic neurotoxic effects when combined with other venom components. Snake Venom Metalloproteinases (SVMPs), another prominent family, act like molecular scalpels that slice through the extracellular matrix and degrade vascular endothelium, causing hemorrhage and tissue liquefaction. These come in several forms, from P-I (simplest) to P-III (most complex), each equipped with domains that increase their lethality. Serine Proteinases (SVSPs), on the other hand, mimic human enzymes like thrombin but sabotage normal coagulation by either consuming clotting factors or initiating uncontrolled clot formation, leading to either dangerous bleeding or thrombosis. Together, these enzymes orchestrate a molecular catastrophe in the host, paving the way for paralysis, shock, or death.

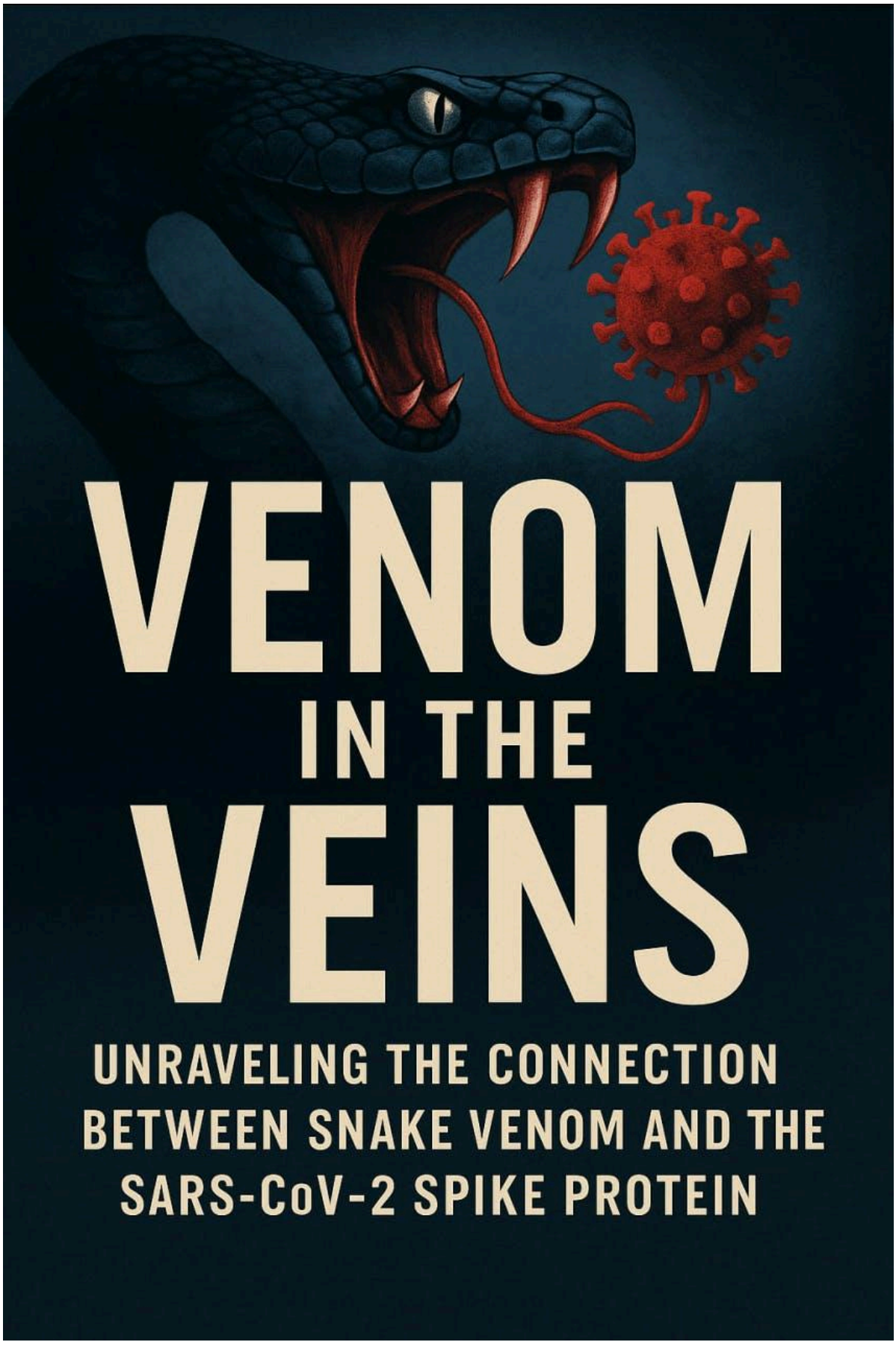
The neurotoxic branch of venom composition is dominated by Three-Finger Toxins (3FTx), small but potent peptides particularly abundant in elapid snakes. Their structural motif—three looped extensions from a central core—makes them versatile weapons. These include α-neurotoxins, which block neuromuscular transmission by targeting nicotinic acetylcholine receptors, leading to rapid paralysis and respiratory arrest. Other 3FTx variants include cardiotoxins that disrupt cardiac cells and cytotoxins that perforate cell membranes. Adding to this are C-Type Lectins (CTLs) and Disintegrins, both of which hijack platelet function and integrin interactions, altering clotting and promoting either thrombosis or hemorrhage. The L-Amino Acid Oxidases (LAAOs) generate reactive oxygen species like hydrogen peroxide, leading to oxidative damage and triggering cell death. Though often overshadowed, Cysteine-Rich Secretory Proteins (CRISPs) modulate ion channels and smooth muscle activity, contributing to prey immobilization by subtly disrupting neuromuscular control.

Other unique components provide additional layers of venom complexity. Bradykinin-Potentiating Peptides (BPPs) interfere with the body’s regulation of blood pressure by inhibiting angiotensin-converting enzyme (ACE), resulting in profound hypotension. Some snake venoms also contain Kunitz-Type Protease Inhibitors, which can block potassium channels or neutralize serine proteases, leading to neurotoxicity or impaired clotting. Natriuretic Peptides contribute to venom-induced cardiovascular collapse by inducing vasodilation and promoting sodium excretion, while Sarafotoxins, found in burrowing asps, mimic potent vasoconstrictive molecules called endothelins, causing lethal blood pressure fluctuations. Additional venom ingredients like

Acetylcholinesterase (AChE) degrade neurotransmitters at synapses, Hyaluronidase acts as a spreading factor to accelerate venom diffusion, and Nucleotidases/Nucleases break down ATP and nucleic acids, weakening cellular defenses and promoting systemic failure. These ingredients don't act in isolation. In fact, venom potency lies in its synergy—where PLA_2s compromise cell membranes, SVMPs destroy blood vessels, and SVSPs hijack clotting—all acting in concert to overwhelm the body's systems.

Ultimately, snake venom represents nature's most refined form of biochemical warfare. It is not merely a tool for hunting—it is an ecosystem of molecular sabotage designed for rapid subjugation of prey or defense against predators. Its impact on human victims is severe and multifaceted: neurotoxins paralyze, hemotoxins disrupt clotting and induce hemorrhage, cytotoxins melt tissue, myotoxins dissolve muscle, and cardiotoxins destabilize the heart. In some species, nephrotoxicity leads to acute kidney injury due to systemic myolysis and toxin load. The complexity and specificity of venom constituents also make them valuable in biomedical research. Several venom-derived compounds have inspired drugs for hypertension, anticoagulation, and even cancer therapy. Still, the brutal efficiency of snake venom reminds us that evolution is an unforgiving chemist—its formulas perfected for death, but occasionally, paradoxically, yielding cures in the hands of human science.

Article #3: Venom in the Veins: Unraveling the Connection Between Snake Venom and the SARS-CoV-2 Spike Protein



The emergence of SARS-CoV-2 and the resultant focus on its spike protein has stirred not only global health concern but also curiosity and controversy. Amid these conversations, some scientists and theorists have drawn biochemical and symbolic comparisons between the virus's spike protein and snake venom toxins. While mainstream medical institutions have dismissed many of these links as fringe, a growing body of literature and observational overlap has raised valid scientific questions. The biochemical behaviors of toxic venom proteins and the SARS-CoV-2 spike protein show startling similarities in how they target cellular receptors, disrupt physiological systems, and instigate inflammatory cascades. Both entities share a remarkable affinity for the ACE2 receptor, which is critical in regulating cardiovascular and neurological functions. The spike protein, like certain venom peptides, can mimic enzymatic actions that enable cellular entry, alter vascular permeability, and induce abnormal immune responses—all hallmarks of envenomation.

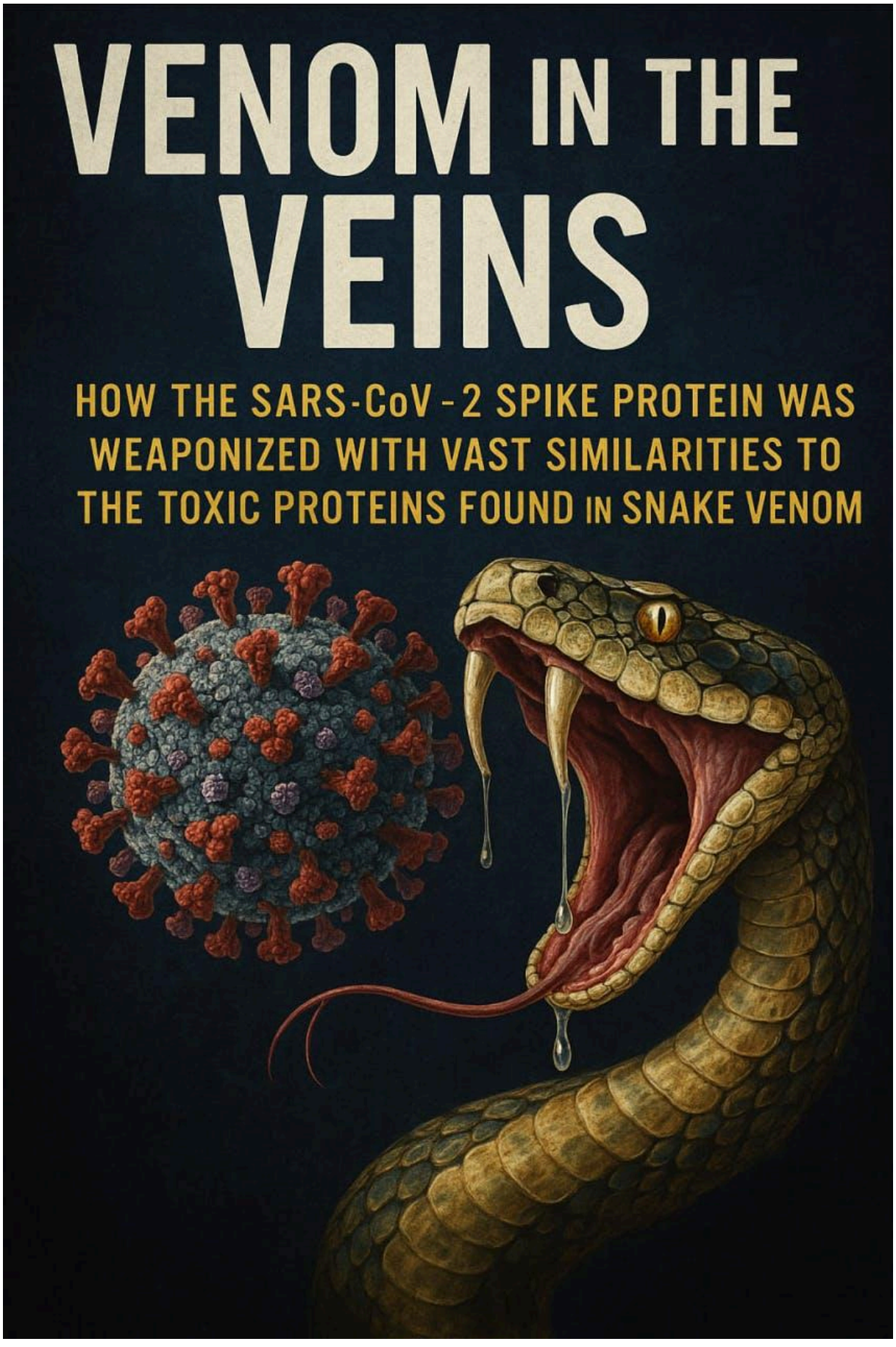
Biochemically, snake venom is a rich blend of toxic protiens and potent molecules such as phospholipase A2 (PLA_2), snake venom metalloproteinases (SVMPs), and serine proteases, which target specific cell structures to disable prey. These enzymes disrupt cellular membranes, trigger hemorrhage, and interfere with nerve transmission. Similarly, the SARS-CoV-2 spike protein, while not enzymatic in the same way, attaches to the ACE2 receptor to facilitate viral entry. This receptor-binding event leads to downstream inflammation, immune dysregulation, and clot formation, not unlike the vascular and neurological damage seen in venomous bites. Studies have noted that the spike protein itself—independent of viral replication—can induce endothelial injury, inflammation, and clotting disorders such as elevated D-dimer levels and microthromboses, especially evident in long COVID or adverse vaccine responses. The shared pathways through which both the spike and venom components act—particularly involving ACE2, blood-brain barrier permeability, and neuroinflammation—highlight a curious convergence of biology between nature's ancient weapons and modern bioengineering.

Theoretical and controversial claims further stretch this connection, suggesting deliberate or accidental biomimicry. A 2022 hypothesis posited that certain peptide sequences in the spike protein closely resemble those found in α-bungarotoxin (a neurotoxin from the banded krait) and conotoxins from marine cone snails. These peptides are known for their ability to block neurotransmitter receptors and paralyze muscles. If embedded within the spike protein, even in fragmentary form, such motifs could interact with nervous system pathways and immune receptors in unpredictable ways. Adding fuel to the fire, the "Watch the Water" theory—dismissed by many yet compelling to others—proposes that synthetic or natural venom components were purposefully introduced via vaccines or environmental vectors. Although lacking definitive empirical support, the theory draws attention to overlapping symptom profiles: respiratory failure, neurological impairment, coagulation anomalies, and lingering chronic effects—all documented both in severe COVID cases and venom exposures. Peer-reviewed studies from journals like Toxins and Circulation Research have explored the structural resemblance and functional mimicry between the spike and known venom peptides, lending limited but intriguing support to these claims.

Beyond the cellular and molecular details lies a powerful metaphorical resonance that has captivated many observers. Venom, in mythology and medicine, has always been a double-edged sword—symbolizing both destruction and healing. Similarly, the spike protein, hailed initially as the keystone of vaccine salvation, has revealed a darker profile in its adverse outcomes. Both snake venom and the spike protein can paralyze, destabilize, and silently undermine health before symptoms fully manifest. The fear of an unseen enemy, the sense of betrayal by a biological agent once thought to be beneficial (as in the case of vaccines), and the psychological toll of prolonged uncertainty—mirror the ancient dread evoked by venomous creatures. In this light, the “venom-spike” connection transcends molecular mimicry and taps into a deeper archetypal narrative: one of manipulation, hidden danger, and the battle for life at the invisible level of cells and codes.

In conclusion, while snake venom and the SARS-CoV-2 spike protein originate from vastly different biological systems—reptilian glands versus viral genomes—their functional intersections deserve rigorous scrutiny. Both possess the capability to hijack critical physiological pathways, cause systemic harm, and evoke neurological, vascular, and immunological distress. The spike protein’s sequence contains motifs that partially mimic known toxins, which could help explain some of its more severe and puzzling effects. These overlaps warrant more transparent investigation rather than dismissal, especially as vaccine-injured individuals and long COVID patients seek answers. Whether the similarities are coincidental, convergently evolved, or artificially engineered remains a topic of intense debate. But in both science and symbolism, the serpent still strikes—reminding us that nature’s most ancient poisons may have more in common with our most modern threats than we dare to imagine.

Article #4: Venom in the Veins: How the SARS-CoV-2 Spike Protein Was Weaponized with Vast Similarities to the Toxic Proteins Found in Snake Venom



In the early days of the COVID-19 pandemic, the world focused on the virus's origin, transmission, and potential vaccines. But beneath mainstream narratives, some researchers and scientists began uncovering chilling biochemical similarities between the SARS-CoV-2 spike protein and certain peptides found in snake venom. While the official narrative held firm to the natural zoonotic origin theory, an undercurrent of evidence emerged suggesting that the spike protein, far from being a natural evolutionary product, was synthetically engineered with deliberate intent—echoing the toxic profile of venom proteins known for their lethal precision and ability to hijack biological systems. The alarming overlap in structural motifs and biological pathways between the spike protein and venom-derived toxins points to a biotechnological design that is as sinister as it is sophisticated.

Snake venom is a biochemical weapon evolved over millennia, comprising enzymes and peptides that disrupt physiological functions with ruthless efficiency. Among the most notorious components are phospholipase A_2 enzymes, serine proteases, bradykinin-potentiating peptides, and metalloproteinases, which collectively target blood clotting, neural transmission, and cell integrity. The SARS-CoV-2 spike protein, which facilitates viral entry into human cells via the ACE2 receptor, appears to mimic several of these toxic mechanisms. Research shows that the spike protein also modulates bradykinin pathways—inducing vascular permeability, hypotension, and inflammation—an effect strikingly similar to the way snake venom disrupts homeostasis. This 'bradykinin storm' contributes to many of COVID-19's most severe symptoms, including pulmonary edema, cytokine overdrive, and multi-organ failure. The spike protein's unusual furin cleavage site, a feature absent in related coronaviruses, further supports the hypothesis of gain-of-function manipulation to enhance its pathogenicity and mimic the multifaceted effects of envenomation.

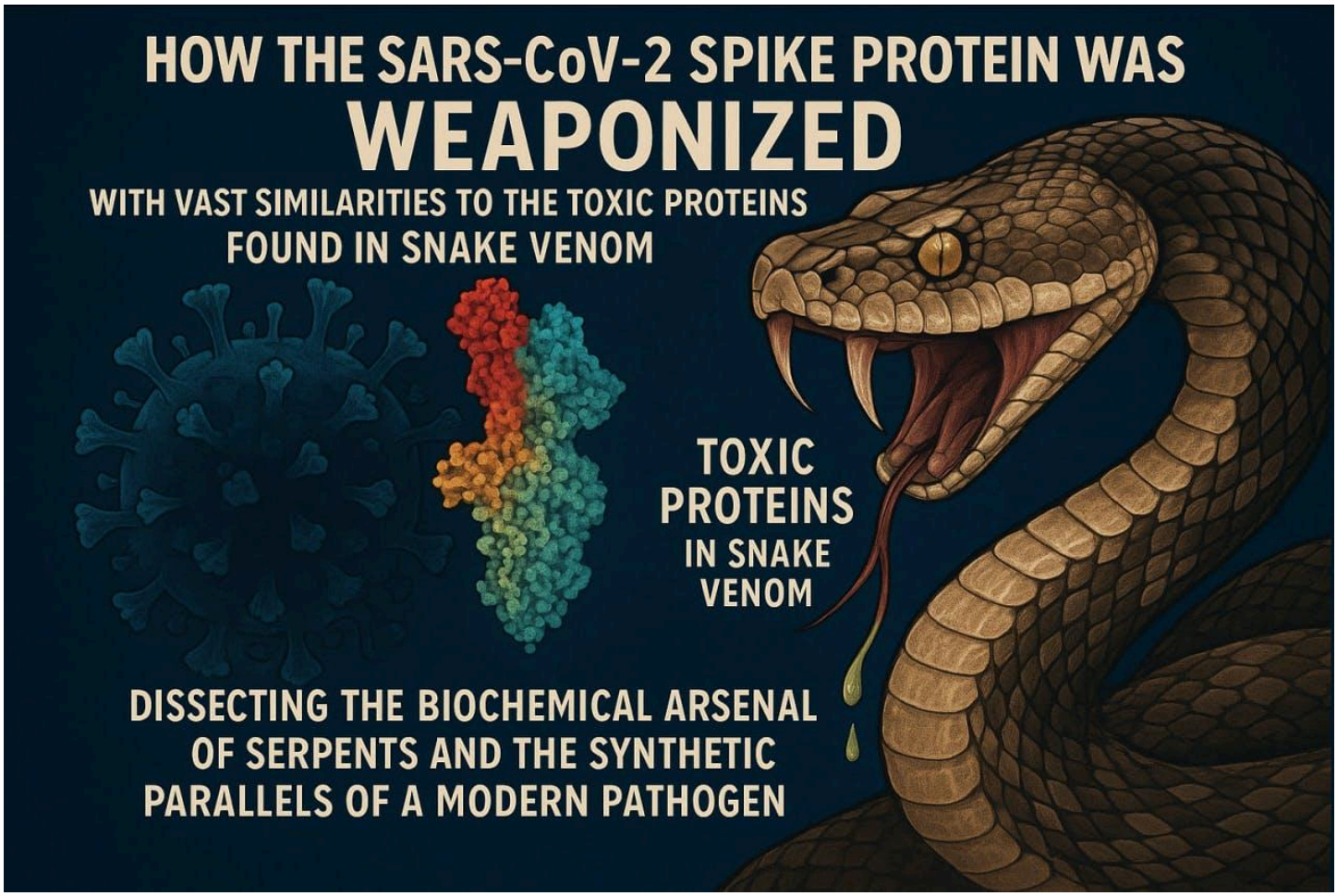
One of the most compelling arguments for weaponization lies in the spike protein's impact on blood and neurological functions. Similar to the coagulopathic effects of viper venom, the spike protein has been linked to thrombotic events, myocarditis, and cerebral vascular complications. These effects are not only observed in COVID-19 infections but have also manifested post-vaccination in some individuals, raising concerns about the persistent expression of spike protein in the body. The venom-like action of the spike protein on platelets and endothelial cells mimics snake venom metalloproteinases (SVMPs), which degrade extracellular matrix proteins and promote hemorrhage or clotting depending on the dosage and context. Furthermore, emerging studies have shown that the spike protein can cross the blood-brain barrier and bind to neural tissues, much like neurotoxic components in cobra venom that interfere with synaptic communication. These parallels raise haunting questions: Was this convergence accidental, or was it a blueprint borrowed from nature's deadliest toolkit?

Beyond biochemical mimicry, the political and scientific suppression of early treatment protocols, such as the use of anti-venom-related compounds (e.g., nicotine receptor modulators or anti-inflammatory agents targeting bradykinin pathways), suggests a broader agenda at play. Some researchers, notably Dr. Bryan Ardis, have speculated that the spike protein's effects are not coincidentally venom-like but rather a bioweapon inspired by venom. Ardis' hypothesis gained traction when unexplained neurological symptoms, clotting disorders, and organ failures were found to be consistent with systemic envenomation rather than viral infection alone. Furthermore, snake venom-based compounds are already used in pharmaceutical development—demonstrating that scientists are not only capable of isolating and synthesizing these toxins but also manipulating them for targeted delivery. This technological capability brings into question whether the spike protein was

crafted using venom-mimetic strategies to induce long-term immunological and systemic damage under the guise of a pandemic.

In the final analysis, the uncanny similarities between snake venom proteins and the SARS-CoV-2 spike protein cannot be dismissed as mere coincidence. From its interaction with ACE2 and nAChR receptors to its influence on coagulation, inflammation, and neuronal pathways, the spike protein behaves less like a typical viral antigen and more like a deliberately engineered biochemical agent. This reframes the entire pandemic—not as a natural disaster but as a coordinated global event centered around the release and dissemination of a toxin masquerading as a viral spike protein. This realization demands not only scientific inquiry but also ethical reckoning, legal accountability, and a reevaluation of how modern biowarfare might be waged not through bombs or bullets, but through microscopic molecular saboteurs that mimic the deadliest elements of nature itself.

Article #5: How the SARS-CoV-2 Spike Protein Was Weaponized with Vast Similarities to the Toxic Proteins Found in Snake Venom: Dissecting the Biochemical Arsenal of Serpents and the Synthetic Parallels of a Modern Pathogen



The SARS-CoV-2 spike protein, the defining feature of the virus responsible for the COVID-19 pandemic, has garnered intense scrutiny for its unique structural properties and pathological effects on the human body. Unlike traditional viral spike proteins, which primarily facilitate receptor binding and cell entry, the SARS-CoV-2 spike appears to play a far more complex and potentially sinister role. Researchers have noted the spike's uncanny ability to interact with a broad range of human cell receptors, dysregulate immune responses, and initiate a cascade of inflammatory and thrombotic events, mirroring the destructive capabilities of known biological toxins. Among these, snake venom proteins—particularly those derived from elapid species such as cobras and kraits—have been shown to possess strikingly similar biochemical characteristics. Phospholipase A2 enzymes, metalloproteinases, and neurotoxins found in venom are notorious for their capacity to paralyze, degrade tissue, and manipulate hemostasis, and eerily, many of these same pathways are disrupted in severe cases of COVID-19. This biochemical overlap has led a growing number of scientists to question whether the spike protein's function is natural evolution—or bioengineering.

Dissecting the molecular makeup of snake venom reveals a diverse arsenal of toxic components, each designed by nature for maximum lethality and biological disruption. Elapid venoms, for instance, are rich in three-finger toxins (3FTx), which bind to acetylcholine receptors and inhibit nerve signal transmission, leading to paralysis. Viperid venoms contain hemorrhagins—metalloproteinases that

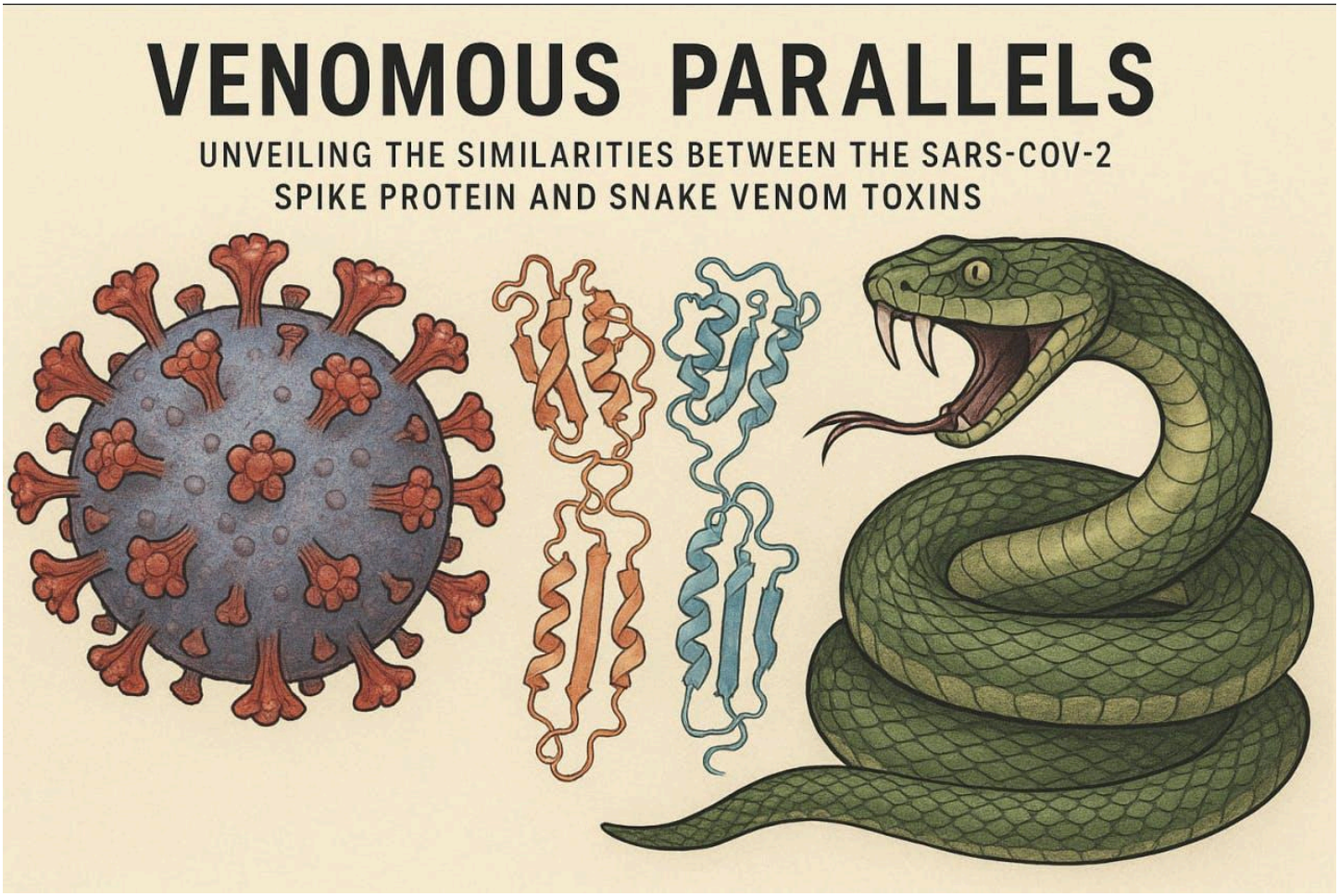
break down capillary walls, leading to internal bleeding. Some venoms contain serine proteases that mimic or block coagulation factors, tipping the balance toward thrombosis or uncontrolled bleeding. These toxins are highly specific, with evolved targeting mechanisms that latch onto cell membranes, disrupt mitochondrial function, and hijack enzymatic processes. Intriguingly, the SARS-CoV-2 spike protein, particularly in its modified mRNA-injected form, shows an ability to similarly bind to nicotinic acetylcholine receptors (nAChRs), ACE2, CD147, and neuropilin-1—receptor affinities that are uncommon for a standard respiratory virus. These interactions may induce symptoms ranging from blood clotting to neurological impairment, mimicking the pathophysiology of envenomation, raising profound questions about whether this is merely convergent biology or intentional molecular mimicry.

Several studies published in 2021 and 2022 deepened the mystery by revealing that the spike protein shares peptide sequences and structural motifs with known snake venom proteins. One preprint that caused a wave of controversy suggested that sequences within the spike were homologous to components found in krait and cobra venom, particularly PLA2 enzymes and 3FTx domains. These motifs are not just structurally similar—they appear to produce similar biochemical outcomes, such as suppression of interferon signaling, mitochondrial dysfunction, and inflammatory cytokine release. The spike protein's superantigen-like region, for example, has been linked to the same type of cytokine storms seen in venom-induced systemic inflammatory responses. If these similarities are more than coincidental, they hint at an unprecedented form of weaponization—where the spike protein becomes a synthetic hybrid of viral vector and venom toxin. This raises a chilling possibility: rather than emerging naturally from zoonotic recombination, SARS-CoV-2 may have been designed to use a venom-mimicking mechanism as a novel mode of biowarfare.

Moreover, the delivery mechanism of the spike protein—especially through mRNA and adenoviral vector platforms—introduces additional parallels to venom biology. In nature, venom is not just toxic—it is also a delivery system, rapidly distributing bioactive proteins into tissue and bloodstream to override cellular defenses. Similarly, the COVID-19 vaccines encode the spike protein and use lipid nanoparticles or viral vectors to introduce this genetic code into human cells. These cells, once transfected, become factories producing spike proteins that migrate into circulation. This method of delivery mirrors the principles of envenomation: target the host's cells, hijack their machinery, and flood the system with a toxin that overwhelms physiological control mechanisms. Cases of myocarditis, neurological damage, autoimmune flare-ups, and long COVID symptoms—many of which mirror systemic envenomation syndromes—add a disturbing layer of evidence that the spike protein behaves in a way far more aggressive than what is typical for respiratory viral antigens.

In conclusion, the deep biochemical resemblance between the SARS-CoV-2 spike protein and known venom toxins is not only scientifically provocative—it is geopolitically and ethically explosive. If the spike protein was engineered with the intent to mimic venom-like activity, it represents a monumental shift in the concept of biowarfare—from using classical pathogens to deploying synthetic hybrid molecules designed to manipulate the human body on a molecular level. The implications span medicine, military ethics, public health, and international law. Dissecting this issue demands not just scientific rigor, but moral courage, as researchers and citizens alike confront the possibility that a global crisis was precipitated not by nature's randomness, but by the deliberate use of molecular mimicry to unleash a pathogen with the hallmarks of a serpent's fang. As investigations continue, humanity stands at a crossroads, tasked with unmasking the true origin of this pathogen—and ensuring that such biochemical weaponization never darkens the world again.

Article #6: Venomous Parallels: Unveiling the Similarities Between the SARS-CoV-2 Spike Protein and Snake Venom Toxins



The emergence of SARS-CoV-2 and the subsequent global pandemic has led researchers down a labyrinthine path of investigation, revealing unsettling similarities between the virus's spike protein and certain toxic proteins found in snake venom. While initially dismissed by some as speculative or conspiratorial, these parallels are grounded in biochemical structure and function. Snake venom is a highly evolved biochemical weapon composed of peptides and proteins that target specific physiological systems, notably the nervous, cardiovascular, and coagulation systems. Interestingly, the spike protein of SARS-CoV-2 also demonstrates a multifaceted ability to interfere with similar systems in the human body. Both classes of proteins share features such as receptor binding domain mimicry, enzymatic activity modulation, and potential interference with hemostatic balance. This observation invites a deeper inquiry into whether the spike protein's pathogenicity is merely a result of natural evolution or something far more deliberate and engineered.

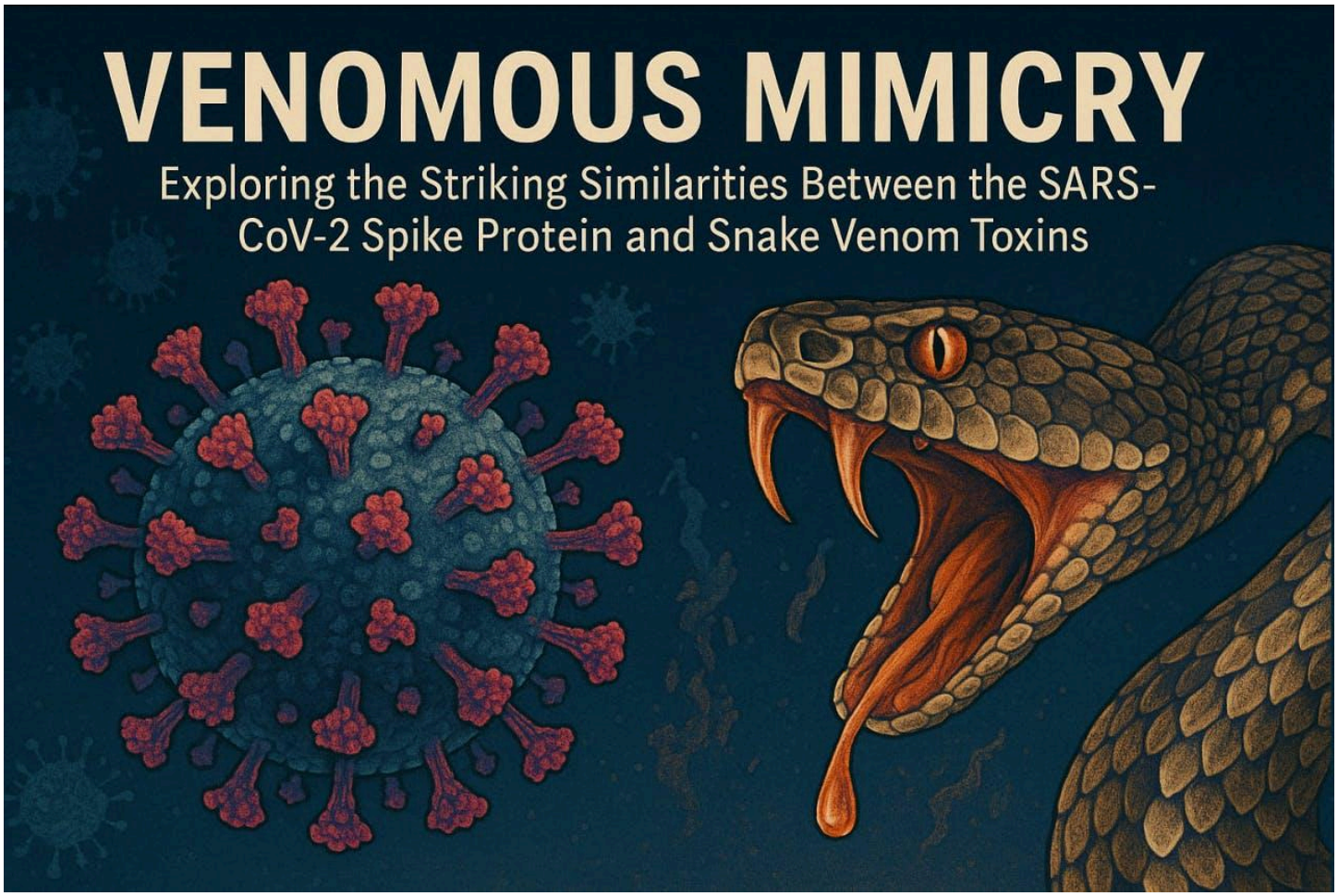
The spike protein of SARS-CoV-2, primarily known for facilitating viral entry through the ACE2 receptor, exhibits functional motifs and sequence homologies reminiscent of snake venom phospholipase A2 (PLA2) enzymes and neurotoxins. These snake venom components are known to disrupt cellular membranes, impair mitochondrial function, and modulate immune responses, which echoes the diverse pathological effects attributed to spike protein interaction with endothelial cells, neurons, and cardiomyocytes. Researchers have identified that certain amino acid sequences in the spike protein share homology with neurotoxic and cytotoxic domains found in Elapidae (cobra and krait) venoms, suggesting a potential for neurological implications beyond viral replication. The spike

protein’s binding to nicotinic acetylcholine receptors (nAChRs), a pathway also targeted by α-bungarotoxin from snake venom, could explain the widespread neurological symptoms — including anosmia, fatigue, brain fog, and seizures — observed in both acute COVID-19 and long-haul syndrome patients.

Moreover, both snake venom toxins and the spike protein can induce a pro-thrombotic state. Venom-derived serine proteases, metalloproteinases, and disintegrins interact with the coagulation cascade and platelet receptors to cause hemorrhage or thrombosis. Similarly, the spike protein has been implicated in causing endothelial damage, platelet aggregation, and microclot formation, particularly through its effects on the vascular endothelium and glycocalyx. Autopsy findings in COVID-19 victims often show widespread endothelialitis, microvascular inflammation, and thrombi in capillary beds — strikingly reminiscent of envenomation pathology. This raises grave concerns about the spike protein’s persistence in the body following vaccination or natural infection, especially as circulating spike fragments have been detected months post-inoculation, potentially contributing to long-term cardiovascular complications. The question then arises: are we dealing with a pathogen that mimics venom, or a venom-like construct disguised as a pathogen?

Finally, the immunological implications of these similarities must not be underestimated. Snake venom toxins are known to dysregulate immune responses by overactivating or suppressing specific pathways. The spike protein similarly elicits a complex immune response, often characterized by hyperinflammation (cytokine storms), autoimmunity, and immune exhaustion. Furthermore, just as repeated exposure to venom can sensitize the immune system or induce autoimmune phenomena, prolonged spike protein expression — particularly via mRNA or adenoviral vector platforms — may pose similar risks. This resemblance is not only biochemical but potentially strategic, suggesting a model of immune manipulation via a bioengineered protein with venom-like traits. As we continue to study these correlations, it becomes increasingly clear that understanding the venom-like properties of the spike protein help us understand the similarities of the SARS-CoV-2 Spike Protein compared to snake venom and demand a re-evaluation of current treatment strategies. The convergence of virology, toxicology, and immunology in this context should not be dismissed but urgently further investigated.

Article #7: Venomous Mimicry: Exploring the Striking Similarities Between the SARS-CoV-2 Spike Protein and Snake Venom Toxins with Scientific Investigations & Studies



Over the past several years, a growing number of scientific investigations and studies have shed light on a provocative and biologically interesting phenomenon: the structural and functional similarities between the SARS-CoV-2 spike (S) protein and certain components of snake venom toxins. These studies have gone beyond superficial resemblances to identify specific protein motifs, disulfide bonding patterns, and functional domains within the spike protein that mimic those found in known venom-derived enzymes and peptides. For instance, researchers have pointed to conserved cysteine-rich motifs and looped structures in the receptor-binding domain (RBD) of the SARS-CoV-2 spike protein that resemble the three-finger toxin architecture seen in elapid snake venoms (e.g., cobras and kraits). These motifs are not only structurally significant but are associated with neurotoxic and cytotoxic activity in the animal kingdom—raising serious questions about whether similar biochemical pathways may be contributing to the systemic inflammation, neurological symptoms, and vascular damage seen in severe COVID-19 cases.

One particularly compelling area of comparison is the enzymatic behavior and receptor-binding characteristics shared between certain venom proteins—such as phospholipase A2 (PLA2), metalloproteinases, and neurotoxins—and the spike protein's affinity for binding to ACE2 receptors. Venom toxins often manipulate or hijack host receptors to gain entry into cells or disrupt vital physiological processes. Likewise, the SARS-CoV-2 spike protein engages ACE2 and related pathways to facilitate viral entry and replication, triggering a cascade of downstream effects including

endothelial dysfunction, coagulopathy, and immune hyperactivation. Several peer-reviewed papers, such as those published in Toxicon and Frontiers in Immunology, have hypothesized that the spike protein might exert venom-like effects by acting as a "superantigen" or by initiating proinflammatory pathways in a manner analogous to envenomation. Additionally, certain short amino acid sequences within the spike protein appear homologous to bradykinin-potentiating peptides found in venom, which may contribute to the infamous “bradykinin storm” theory proposed as a mechanism of COVID-19-induced inflammation and lung injury.

Beyond structural resemblance and receptor interaction, the bioactive consequences of spike protein exposure—either via infection or injection of synthetic spike protein through mRNA platforms—mirror the pathological outcomes of snake venom exposure in some very striking ways. Both can cause blood clotting disorders, hemolysis, cytokine storms, immune dysregulation, and organ damage. In some studies, researchers have found that monoclonal antibodies against venom-derived toxins show cross-reactivity with SARS-CoV-2 spike epitopes, suggesting an immunological overlap that warrants deeper investigation. These parallels have profound implications—not only for understanding the pathogenesis of COVID-19 but also for the broader field of bioengineering and virology, where synthetic biology may inadvertently or deliberately produce proteins that simulate toxic effects known in nature. As the body of evidence grows, the scientific and medical communities must confront the possibility that SARS-CoV-2 and its spike protein are not just biologically novel, but potentially represent a form of pathogenic biomimicry unlike any seen before in human disease. This emerging understanding could open new therapeutic avenues—such as the use of venom-neutralizing agents or ACE2 decoys—as well as demand a re-evaluation of how we assess the safety of viral vector and protein-based technologies moving forward.

Here are some notable findings:

1. Toxin-like Peptides in COVID-19 Patients

A study published in *Frontiers in Immunology* identified the presence of toxin-like peptides in plasma and urine samples from COVID-19 patients. These peptides closely resemble toxic components found in animal venoms, such as conotoxins, phospholipases, phosphodiesterases, zinc metalloproteinases, and bradykinins. Their presence suggests a potential association between SARS-CoV-2 infection and the release of these venom-like peptides, which may contribute to various clinical manifestations, including neurological symptoms.

https://pmc.ncbi.nlm.nih.gov/articles/PMC8772524/

2. Interaction with Nicotinic Acetylcholine Receptors (nAChRs)

Research has proposed that the SARS-CoV-2 spike protein contains a sequence motif similar to known nAChR antagonists, such as α-bungarotoxin from snake venom. Molecular simulations indicate that a specific region of the spike protein (Y674-R685) can bind to nAChRs, potentially disrupting cholinergic signaling. This interaction might be linked to some of the neurological symptoms observed in COVID-19 patients.

https://www.cell.com/biophysj/fulltext/S0006-3495(21)00146-6

3. Elevated Levels of Snake Venom-like Enzyme in Severe COVID-19

A study conducted by researchers at the University of Arizona found that severe COVID-19 cases exhibited elevated levels of secreted phospholipase A2 group IIA (sPLA2-IIA), an enzyme with high sequence similarity to a key enzyme found in snake venom. This enzyme is known for its role in inflammation and cell membrane degradation. The study suggests that high levels of sPLA2-IIA may contribute to COVID-19 severity and mortality.

https://news.arizona.edu/news/venom-coursing-through-body-researchers-identify-mechanism-driving-covid-19-mortality