

*Playing Russian Roulette with Every COVID-19 Injection: The Deadly Global Game*¹

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Abstract

Are the promoters of the COVID-19 injections leading, pushing, and even forcing the people of the world to play a global game of Russian Roulette? The actuarial statistics and clinical facts are revealing harmful and deadly consequences on a global scale. For the sake of those still able to choose whether to spin the cylinder and pull the trigger one more time, I am conducting a guided tour. I will show why there are exponentially many more ways for the experimental injections to cause disease and death than health and well-being. Briefly put, valid transcription of mRNA from our own DNA leads to well-formed proteins that work as designed for cytoplasm, organelles, cells, tissues, and functional organ systems of the body. By contrast, the foreign (xeno) nucleic acid sequences, the mXNAs, in the “modified mRNA” in the COVID-19 injectables are more likely to harm the body’s systems than to benefit them. The relevant research shows why the mXNAs are incompetent to communicate effectively with the complex native context *in vivo* — the human biosignaling systems that are not mechanical but are articulated in multiple layers, interconnected, and for practical purposes, infinitely varied systems of information processing. Survival and longevity depend on valid communications among nuclear and mitochondrial DNA, their RNAs, and the protein language systems specified before a person’s birth. When these complex biosignaling systems fail, disorders and diseases follow. In catastrophic failures, death occurs. The story is not simple and the tour I am conducting is challenging with more than a few twists, turns, and digressions. However, I believe, persons experimented on — almost all of them unknowingly, some with partial knowledge who were unwilling recipients — account for more than half the world population. The stakes are high, even life or death, so I think many of the recipients of one or more injections will take the guided tour.

Keywords: COVID-19 vaccine recipients, engineered mXNA, modified mRNA, N1-methylpseudouridine, spike protein coding sequenc, uridine substitutions, XNA

Introduction

By now, it is reported that 4.9 billion people have received at least one (Pharmaceutical Technology, 2022) of the 12.7 billion doses of the COVID injections that have been administered (Vaccine

¹ My style in this paper is intentionally directed toward what I believe are the unavoidable conclusions flowing from clinical findings — from the facts of what is being found in the fluids being injected and in the bodies of recipients. We are learning from those still alive and from those already dead that the so-called “vaccines” contain harmful foreign materials that were not disclosed to the public. My review of relevant literature and the clinical outcomes discussed here is exclusively about the emerging empirical (scientific) facts as contrasted with the false corporate narrative consisting of — I hate to say it and wish I were wrong — propaganda (see the citations from Broudy and colleagues that follow). The clinical facts reveal that the pharmaceutical-medical-government complex has been orchestrating a false narrative supported by deceptive concealments and even lies. The relevant science is about whatever is in the injected fluids that ends up in the blood and organs causing many to get sick and even drop dead with huge unnatural clots in their bodies.

Tracker, 2022). Some took the shots willingly, and others to keep from being denied access to schools, restaurants, travel, or employment (Verkerk et al., 2022). At first many were anxious to submit to one of the injections, now not so many. Others were reluctant or refused COVID-19 injections from the beginning, some at the cost of their livelihood (Provost et al., 2022).

At the time of this writing, as the death toll mounts up (Dowd, 2022a, 2022b; Oller & Santiago, 2022; Mercola, 2022a, 2022c), and many ordinary people, such as I am, are reporting friends, family, and loved ones who took one or more of the shots experiencing neurological disabilities, inexplicable and highly aggressive resurgent cancers, circulatory disorders, thrombocytopenias, myocarditis, or death. They are seeing consequences from injections that are evidently worse than the disease as predicted by Seneff & Nigh, 2021; also Seneff, Nigh, Kriakopoulos, & McCullough, 2022) and even worse than losing a job — the folks now refusing the injectables have become a large majority. As of today, only 0.9% are taking all the “boosters” to become “fully vaccinated” (Pharmaceutical Technology, October 4, 2022) while 99.1% are not. I expect many of those who have taken one or more of the experimental injections will want to follow along on the tour now underway right here.

DECEIVING NATIVE BIOSIGNALING SYSTEMS

It is noteworthy that the genetic engineers producing the COVID-19 Pfizer and Moderna recipes (BNT162b2, mRNA-1273) — before they were placed in vials and then injected into billions of people — did so with the explicit intention of deceiving the body's native biosignaling systems — our DNA, RNA, and protein languages. The creation of the foreign, xenogenetic, mXNA spike coding sequence, was intended to deceive the native systems from the start. Nance and Meier wrote:

... we summarize the development and function of m1Ψ in synthetic mRNAs [p. 748]. ... [this] non-natural RNA nucleobase N1-methylpseudouridine (m1Ψ; Figure 1b [my Figure 1]) ... enhances immune evasion and protein production [p. 748]. ... The modified nucleobase *helps cloak mRNA vaccines from the immune system* [my emphasis, p. 753].

Nance and Meiers (p. 752), and others (see Parr et al., 2020), also say that the longevity (half-life) and consequent protein producing power of the mRNA producing spike is enhanced by transforming it with m1Ψ to an mXNA. Oddly, Nance and Meier seem to forget that they said a few pages earlier that the synthetic nucleic acids “are nonreplicating” “naturally decompose” and “do not integrate into genomes” (p. 748). But which parts of this mainstream story, if any, are true?

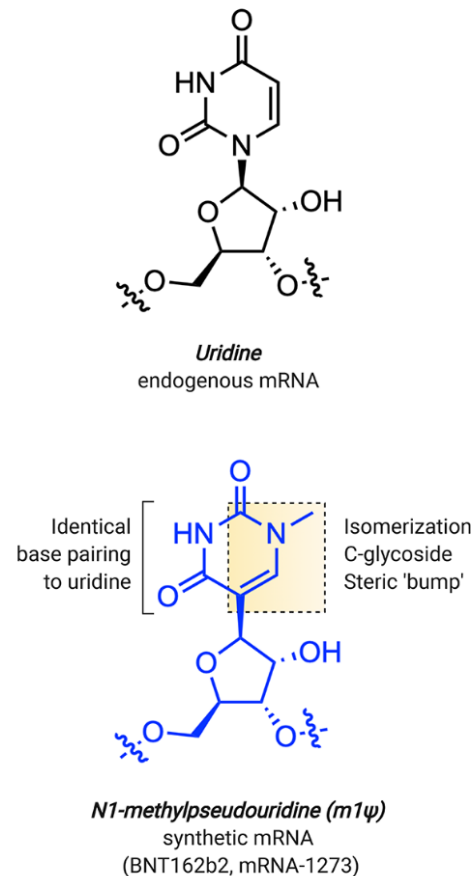


Figure 1. Comparing the native uridine in natural mRNA with the xenogenetic mXNA made to deceive the body's biosignaling systems (Figure 1b from Nance & Meier, 2021, p. 749, not subject to US copyright).

A Critical Substitution

What I want to devote attention to here is the substitution of N1-methylpseudouridine (m1Ψ) for the native uridine of natural mRNA. The global experiment with mXNA injections depends on research dating back more than a half century. Nance and Meier cite the work of Chamberlin et al. (1970, 1983) which identified a bacterial enzyme in *E. coli* that “can produce RNAs longer than 20,000 nucleotides without making an error” (p. 749). Such a statement seems to imply that the mXNA string in the SARS-CoV-2 modified spike coding sequence with only 3,821 nucleotides can be flawlessly produced, and reproduced in perpetuity, in all the injections and in human bodies. They say it will be “taken up by muscle and infiltrating immune cells that use it to produce spike protein” which will “be displayed on the cell surface, allowing it [the disease agent spike] to be recognized by the immune system”. They say that all this will cause “the production of antibodies and T-cells that protect against natural infection and prevent serious disease”. However, it must be re-emphasized that the mXNA coding sequences are *unnatural*, and there are tens of billions of them in each injection (Fleming, 2021, p. 99). Even in the updated COVID-19 “booster” injections, half the payload still consists of a synthetic mXNA coding sequence for the original SARS-CoV-2 spike protein, while the other half, also coded as mXNAs, is aimed at more recent variants (Lin, 2022). Nance and Meier say that the spikes generated by the mXNAs “cannot cause COVID-19” disease. They are supposed to “naturally decompose”. Nance and Meier also assert that the mXNA does “not integrate into genomes” (p. 748).

THE MAINSTREAM NARRATIVE, DEPENDS ON A BIOLOGICAL LIE

Nance and Meier (2021) stress that it is the “cloaking” of mXNA that causes the body’s natural immune cells to regard the man-made material as native (natural) mRNA derived from the body’s own nuclear DNA. This concoction hangs upon a fundamental lie aimed at deceiving the body’s own biosignaling systems. Can the rest of the claims for how the deception will work be trusted? Aldén et al. (2022) found the claim that mXNA does not incorporate itself into DNA to be false. The early prediction that the mXNA will be degraded and rapidly disappear was, however, shown by Röltgen et al. (2022) to be patently false. Two months after injection, the foreign mXNA was still active in recipients (also see Bansal et al., 2021). The presupposition that it would remain localized in the muscle at the site of the injection, and would not travel to the heart, lungs, and brain also turned out to be false (Classen, 2021; Baumeier et al., 2022; McLernon, 2022; Østein et al., 2022; Oster et al., 2022). Similarly, the claim that the mXNA injections would prevent disease from COVID-19 and its successors also proved false (Oller & Santiago, 2022; Mercola, 2022b, and see especially Mercola’s references). Independent research has shown that a higher number of doses of the injectables is actually associated with a greater likelihood of hospitalization, diagnosis of COVID disease, and death following the diagnosis (Gazit et al., 2021; Brown et al., 2021; Seneff & Nigh, 2021; Classen, 2021; Berdine, 2022). Moreover, increasingly it has become clear that persons with “long COVID” are more apt to have received one or more of the COVID-19 injections than not (Patterson et al., 2022) and there is pervasive clinical evidence that the COVID-19 vaccine based spike protein can cause disease and even death by itself (Bansal et al., 2021).

Trouble with the Start Codon

As we are about to see, trouble with the genetically manipulated injections begins with the start codon in the mXNA. In all mRNA productions, the first question is where the ribosome is to begin

AUG UUC GUG UUC CUG GUG CUG CUG CCU CUG GUG UCC AGC CAG UGU GUG AAC CUG ACC ACC AGA ACA
 CAG CUG CCU CCA GCC UAC ACC AAC AGC UUU ACC AGA GGC GUG UAC UAC CCC GAC AAG GUG UUC AGA
 UCC AGC GUG CUG CAC UCU ACC CAG GAC CUG UUC CUG CCU UUC UUC AGC AAC GUG ACC UGG UUC CAC
 GCC AUC CAC GUG UCC GGC ACC AAU GGC ACC AAG AGA UUC GAC AAC CCC GUG CUG CCC UUC AAC GAC
 GGG GUG UAC UUU GCC AGC ACC GAG AAG UCC AAC AUC AUC AGA GGC UGG AUC UUC GGC ACC ACA CUG
 GAC AGC AAG ACC CAG AGC CUG CUG AUC GUG AAC AAC GCC ACC AAC GUG GUC AUC AAA GUG UGC GAG
 UUC CAG UUC UGC AAC GAC CCC UUC CUG GGC GUC UAC UAC CAC AAG AAC AAC AAG AGC UGG AUG GAA
 AGC GAG UUC CGG GUG UAC AGC AGC GCC AAC AAC UGC ACC UUC GAG UAC GUG UCC CAG CCU UUC CUG
 AUG GAC CUG GAA GGC AAG CAG GGC AAC UUC AAG AAC CUG CGC GAG UUC GUG UUU AAG AAC AUC GAC
 GGC UAC UUC AAG AUC UAC AGC AAG CAC ACC CCU AUC AAC CUC GUG CGG GAU CUG CCU CAG GGC UUC
 UCU GCU CUG GAA CCC CUG GUG GAU CUG CCC AUC GGC AUC AAC AUC ACC CGG UUU CAG ACA CUG CUG
 GCC CUG CAC AGA AGC UAC CUG ACA CCU GGC GAU AGC AGC AGC GGA UGG ACA GCU GGU GCC GCC GCU
 UAC UAU GUG GGC UAC CUG CAG CCU AGA ACC UUC CUG AAG UAC AAC GAG AAC GGC ACC AUC ACC
 GAC GCC GUG GAU UGU GCU CUG GAU CCU CUG AGC GAG ACA AAG UGC ACC CUG AAG UCC UUC ACC GUG
 GAA AAG GGC AUC UAC CAG ACC AGC AAC UUC CGG GGC AGC CCA CCG AAU CCA UCG UGC GGU UCC CCA
 AUA UCA CCA AUC UGU GCC CCU UCG GCG AGG UGU UCA AUG CCA CCA GAU UCG CCU CUG UGU ACG CCU
 GGA ACC GGA AGC UCA GCA AUU CGG UGG CCG AUC ACU CCG UGC UGU ACA ACU CCG CCA GCG UCA
 GCA CCU UCA AGU GCU ACG GCG UGU CCC CUA CCA AGC UGA ACG ACC UGU GCU UCA CAA ACG UGU ACG
 CCG ACA GCU UCG UGA UCC GGG GAG AUG AAG UGC GGC AGA UUG CCC CUG GAC AGA CAG GCA AGA UCG
 CCG ACU ACA ACU ACA AGC UGC CCG ACG ACU UCA CCG GCU GUG UGA UUG CCU GGA ACA GCA ACA ACC
 UGG AUC CCA AAG UCG GCG GCA ACU ACA AUU ACC UGU ACC GGC UGU UCC GGA AGU CCA AUC UGA AGC
 CCU UCG AGC GGG ACA UCU CCA CCG AGA UCU AUC AGG CCG GCA GCA CCC CUU GUA CCG GCG UGG AAG
 GCU UCA ACU GCU ACU UCC CAC UGC AGU CCU ACG GCU UUC AGC CCA CAA AUG GCG UGG GCU AUC AGC
 CCU ACA GAG UGG UGG UGC UGA GCU UCG AAC UGC UGC AUG CCC CUG CCA CAG UGU GCG GCC CUA AGA
 AAA GCA CCA AUC UCG UGA AGA ACA AAU GCG UGA ACC UCA ACU UCA ACG GCC UGA CCG GCA CCG GCG
 UGC UGA CAG AUG GCA ACA AGA AGU UCC UGC CAU UCC AGC AGU UUG GCC GGG AUA UCG CCG AUA CCA
 CAG ACG CCG UUA GAG AUC CCC AGA CAC UGG AAA UCC UGG ACA UCA CCC CUU GCA GCU UCG GCG GAG
 UGU CUG UGA UCA CCC CUG GCA CCA ACA CCA GCA AUC AGG UGG CAG UGC UGU ACC AGG ACG UGA ACU
 GUA CCG AAG UGC CCG UGG CCA UUC ACG CCG AUC AGC UGA CAC CUA CAU GGC GGG UGU ACU CCA CCG
 GCA GCA AUG UGU UUC AGA CCA GAG CCG GCU GUC UGA UCG GAG CCG AGC ACG UGA AUA AUA CCG ACG
 AGU GCG ACA UCC CCA UCG GCG CUG GAA UCU GCG CCA GCU ACC AGA CAC AGA CAA ACA GCC CUC GGA
 GAG CCA GAA GCG UGG CCA GCC AGA GCA UCA UUG CCU ACA CAA UGU CUC UGG GCG CCG AGA ACA GCG
 UGG CCU ACU CCA ACA ACU CUA UCG CUA UCC CCA CCA ACU UCA CCA UCA GCG UGA CCA CAG AGA UCC
 UGC UCG UGU CCA UGA CCA AGA CCA GCG UGC AGC GCA UGU ACA UCU GCG GCG AUU CCA CCG AGU
 GCU CCA ACC UGC UGC UGC AGU ACG GCA GCU UCU GCA CCC AGC UGA AUA GAG CCC UGA CAG GGA UCG
 CCG UGG AAC AGG ACA AGA ACA CCC AAG AGG UGU UCG CCC AAG UGA AGC AGA UCU ACA AGA CCC CUC
 CUA ACA AGG ACU UCG GCG GCU UCA AUU UCA GCC AGA UUC UGC CCG AUC CUA GCA AGC CCA GCA AGC
 GGA GCU UCA UCG AGC ACC UGC UGU UCA ACA AAG UGA CAC UGG CCG ACG CCG GCU UCA UCA AGC AGU
 AUG GCG AUU GUC UGG GCG ACA UUG CCG CCA GGG AUC UGA UUU GCG CCC AGA AGU UUA ACG GAC UGA
 CAG UGC UGC CUC CUC UGC UGA CCG AUG AGA UGA UGC CCC AGU ACA CAU CUG CCC UGC UGG CCG GCA
 CAA UCA CAA GCG GCU GGA CAU UUG GAG CAG GCG CCG CUC UGC AGA UCC CCU UUG CUA UGC AGA UGG
 CCU ACC GGU UCA ACG GCA UCG GAG UGA CCC AGA AUG UGC UGU ACG AGA ACC AGA UGC UGC UGA
 ACC AGU UCA ACA GCG CCA UCG GCA UGC AGA UCC AGG ACA GCC UGA GCA GCA CAG CAA GCG CCC UGG GAA
 AGC UGC AGG ACG UGG UCA ACC AGA AUG CCC AGG CAC UGA ACA CCC UGG UCA AGC AGC UGU CCU CCA
 ACU UCG GCG CCA UCA GCU CUG UGC UGA ACG AUA UCC UGA GCA GAC UGG ACC CUC CUG AGG CCG AGG
 UGC AGA UCG ACA GAC UGA UCA CAG GCA GAC UGC AGA GCC UCC AGA CAU ACG UGA CCC AGC AGC UGA
 UCA GAG CCG CCG AGA UUA GAG CCU CUG CCA UAG UGG CCG CCA CCA AGA UGU CUG AGU GUG UGC UGG
 GCC AGA GCA AGA GAG UGG ACU UUU GCG GCA AGG GCU ACC ACC UGA UGA GCU UCC CUC AGU CUG CCC
 CUC ACG GCG UGG UGU UUC UGC ACG UGA CAU AUG UGC CCG CUC AAG AGA AGA AUU UCA CCA CCG CUC
 CAG CCA UCU GCC ACG ACG GCA AAG CCC ACU UUC CUA GAG AAG GCG UGU UCG UGU CCA ACG GCA CCC
 AUU GGU UCG UGA CAC AGC GGA ACU UCU ACG AGC CCC AGA UCA UCA CCA CCG ACA ACA CCU UCG UGU
 CUG GCA ACU GCG ACG UCG UGA UCG GCA UUG UGA ACA AUA CCG UGU ACG ACC CUC UGC AGC CCG AGC
 UGG ACA GCU UCA AAG AGG AAC UGG ACA AGU ACU UUA AGA ACC ACA CAA GCC CCG ACG UGG ACC UGG
 GCG AUA UCA GCG GAA UCA AUG CCA GCG UCG UGA UCG ACA UCC AGA AAG AGA UCG ACC GGC UGA ACG AGG
 UGG CCA AGA AUC UGA ACG AGA GCC UGA UCG ACC UGC AAG AAC UGG GGA AGU ACG AGC AGU ACA UGA
 AGU GGC CCU GGU ACA UCU GGC UGG GCU UUA UCG CCG GAC UGA UUG CCA UCG UGA UGG UCA CAA UCA
 UGC UGU GUU GCA UGA CCA GCU GCU GUA GCU GCC UGA AGG GCU GUU GUA GCU GUG GCA GCU GCU GCA
 AGU UCG ACG AGG ACG AUU CUG AGC CCG UGC UGA AGG GCG UGA AAC UGC ACU ACA CAU GA

Figure 2. The mRNA code shows all 728 N-1 methylpseudouridines (m1Ψ) as uridines, U. The trinucleotide start codons, AUGs, are highlighted in yellow (14 start codons), and the canonical stop codons, UGAs, are shown in turquoise (49 stop codons). Although UAA and UAG can also function as stop markers in the protein manufacturing systems of the ribosome, there are none in the synthetic spike protein mRNA.

reading codons for the assembly of the amino acids that will, theoretically, build up the protein or any peptide portion of one. Then, there is the question of where the assembly is supposed to end. In theory, the start codon works something like a capital letter at the beginning of a sentence and

the intended stop codon is like the period at the end. If the coding sequence could be read perfectly, it should produce a complete SARS-CoV-2 spike protein with no errors every time. But, even if the sequence in the Pfizer and Moderna mXNA injectables had been manufactured so the mXNA could work as advertised every time (which did not happen; see Rose, 2022), the cloaked mXNA itself would still have to be disposed of. Neither the mXNA, nor its nanolipid container, nor any of its protein products belong in the body. They are toxic disease agents that cause problems on their own. They would do so even if they were of very high quality. But they are not of high quality (Rose, 2022) and, additionally, as I will show on this tour, the intended spike protein is not the only possible protein/peptide product spinning off from the billions of mXNAs delivered in the COVID-19 injections to the body's native ribosomal systems (Fleming, 2021, p. 99).

WHERE DOES THE RIBOSOME BEGIN AND END PRODUCTION?

As shown in Figure 2, there are 14 canonical start codons of the shape AUG (highlighted in yellow in Figure 2; but transformed from AUG in the mXNA to A**m1Ψ**G). Also, there are 49 canonical stop codons of the shape UGA (highlighted in turquoise in Figure 2; transformed in the mXNA to **m1Ψ**GA). For the moment, we will set aside the 728 **m1Ψ** substitutions and we will return to them a little later on in the tour. For now, we note that the multiplicity of start and stop codons leads to what are called “open reading frames” (ORFs) in the mXNA coding sequence of Figure 2. It is a foreign mXNA string before it is interpreted in any way because the SARS-CoV-2 spike is not native among any of the 30,036 proteins catalogued in the NCBI databases from human cells (Siegel, 2022). Each of those ORFs consists of a string of nucleotides — A, G, **m1Ψ**, and C — that can theoretically be parsed into consecutive triplet codons. Except for the **m1Ψ** substituted for U, those translatable codons would determine whatever protein/peptide product should be produced by the ribosome one amino acid at a time. An additional complexity contributing to the number of viable ORFs in the coding sequence for SARS-CoV-2 (my Figure 2) is that the parsing of triplets for the whole ORF can begin with the A of the AUG codon, or with the U, or with the G. Assuming the **m1Ψ** works somewhat like the U in native mRNAs, the “Kozak sequence” guiding normal translation should apply. That sequence, and deviations from it, account for the next stop on the tour.

Violating the Expected Kozak Sequence in mXNA

With respect to the translation process, it is also known that certain other nucleotides in the vicinity of the start codon, either upstream or downstream from it, influence the translation from mRNA to a protein/peptide sequence. Counting nucleotides away from the initial AUG in the upstream direction, positions are numbered -1, -2, and so forth, and in the downstream direction, they are numbered +1, +2, and so on. The biologically preferred sequence in eukaryotes, and especially in human beings, is known as the “Kozak sequence” (Kozak, 1981, 1984a, 1984b, 1986, 1989, 1990, 1997, 1999, 2001; Schneider & Kozak, 2001; Kozak, 2002, 2003; “Kozak Consensus Sequence,” 2022). Deviations from that sequence, in general, will negatively impact the fidelity of the translation process. As a result, the problem of how to punctuate the coding sequence contained in the ORF, given the 728 **m1Ψ** substitutions for natural uridine, is particularly problematic. Because we know that only one error in an amino acid sequence owing to a deviation from the expected Kozak sequence in ordinary natural mRNA can cause a potentially fatal disease (e.g., see De Angioletti et al., 2004), it is unlikely that the **m1Ψ** aberrations in the mXNA will produce good outcomes.

Homologues, Allergies, and Autoimmune Disease

They are likely, based on sound theory and already emerging clinical evidence, to be the proximate cause of disease and death in many recipients. Among the dangers pointed out by various authors (e.g., see Seneff et al., 2022, and also my own comment on Hughes, 2022, in this journal; Santiago, 2022) is the possibility that some of the products spinning off from the mXNA will be similar enough to native proteins, to become incorporated into cells, tissues, and organ systems where they do not belong and cannot function normally. Among the known clinical consequences of such near homologues are autoimmune disorders (Lyons-Weiler, 2020; Vojdani & Kharrazian, 2020; Vojdani et al., 2021). What happens is that the body's defenses detect foreign proteins that so closely resemble its own proteins that it not only begins to attack and destroy the homologues, but also its own proteins, cells, and tissues. In mild cases, allergies will be diagnosed. In more severe ones, fatal autoimmune diseases will emerge. The delay in the appearance of such conditions after the injections may make it difficult to pin down the exact causes of the diseases and deaths that follow, but the theory is clear: near homologues are dangerous in the manner described by Lyons-Weiler and others.

Using the NCBI ORF Finder

To illustrate the difficulty implicit in the published coding sequence for the SARS-CoV-2 spike protein, we can go to the NCBI ORF Finder at [this link](#), plug in that sequence (spelled out in Appendix, Part A) and run the software to find the ORFs.² If the software is set to search the sequence for all possible ORFs (ones that could produce a protein/peptide of 30 amino acids or more) using only the canonical DNA start codon ATG (which is transcribed as AUG in natural mRNA) there are 31 ORFs that could produce possible strings of amino acids. Of those, 14 have one or more known homologs at 100% and many others that share lesser similarity to human proteins in the “UnitProtKB/Swiss-Prot (swissprot)” database; if the software is set to regard ATG and other initiation codons as start positions, there are 68 ORFs, and if it is set to take any triplet of nucleotides that can be read as a codon there are 123 ORFs. Because the databases are known to be incomplete and imperfectly maintained, the search algorithms cannot possibly find all the actual homologous proteins, peptides, and the much shorter “motifs” known to serve as binding sites for the body's immunoglobulins enabling the antibodies and immune cells to attack as they seek to defend against foreign entities.

Using the discovery approach most favorable to the global genetic-engineering experiment underway, there remain 31 possible protein/peptide strings of amino acids to be accounted for. Only one of them can be the intended SARS-CoV-2 spike. Bearing in mind that the addition of the 728 **m1Ψ** substitutions to convert the mRNA into mXNA opens up many more possible outcomes, two questions need to be considered: (1) What is the likely result of the first injection as it spins off unknown protein/peptide products one after another in the organ systems of the body over a period of months? And, (2) what will be the result of adding one injection on top of another in an indefinite series? Appealing to my allegorical title, if a person has already spun the cykinder and pulled the trigger one or more times, should they do it again? A great deal hinges on how the ORFs will shape up once the 728 **m1Ψ** substitutions are introduced into at least 30 billion plus strings of

² Any reader wishing to replicate the findings reported in this paragraph concerning ORFs can follow these simple instructions: (1) go to the linked NCBI public website; (2) copy the coding sequence from the Appendix Part A into the “Enter Query Sequence” box; (3) set the “Minimal length ORF (nt)” at 30; (4) leave the “Genetic Code:” set at “Standard”; (5) select the “ORF start codon to use”; and (6) repeat the last step for each of the three choices specifying what to regard as a start codon.

mXNA, or up to 3.3 times that number in the Emergency Use Authorized (EUA) injections (Fleming, 2021, p. 99). The mainstream corporate narrative urges everyone to keep taking the shots.

What Are the Odds of Landing on an Empty Chamber?

What increases the odds against any good outcomes is that the multiplicity of possible protein/peptide products spinning off from the foreign mXNA coding sequence in the BNT162b2 and mRNA-1273 injectables is the fact that as much as 45% of the total payload in the injections consists of flawed fragments of mXNA (Rose, 2022). Therefore, even if we knew precisely what the intended spike proteins would do, no one can predict what the fragments will do. Seneff and Nigh (2021) explained why:

The European Medicines Agency (EMA) Public Assessment Report is a document submitted to gain approval to market the vaccine in Europe. . . . One concerning revelation is the presence of “fragmented species” of RNA in the injection solution. . . . These fragments, if translated by the cell following injection, would generate incomplete spike proteins, again resulting in altered and unpredictable three-dimensional structure and a physiological impact that is at best neutral and at worst detrimental to cellular functioning. There were considerably more of these fragmented forms of RNA found in the commercially manufactured products than in the products used in clinical trials [p. 60].

But, supposing the fragments of mXNA turned out to be harmless, what about the intended spikes and the unintended protein/peptide products spinning off from the other 30 ORFs?

Clinical Outcomes

Disorders, diseases, and deaths are the overwhelming preponderance of actual clinical outcomes now being confirmed. People are revulsed by pictures of what embalmers are now extracting from the blood vessels and organs of the corpses of vaccinated individuals (Trigoso, 2022). Meanwhile, the diseases and unexplained deaths of recipients of the injections continue to mount up (Berdine, 2022; Mercola, 2022a, 2022c; Oller & Santiago, 2022). Surely, the enormous clot-like structures being removed from the corpses of COVID vaccinees require explanation. Former proponents of the injections such as Aseem Malhotra, one of the MDs who promoted the injectables at the beginning of their rollout, now believes injections should be halted immediately (Malhotra, 2022). Based on the many ORFs in the intended coding sequence, not to mention the high percentage of fragmented mXNAs in the injectables, the foreign entities being injected can only be expected to disrupt the body's biosignaling systems (Gryder et al., 2013; Oller, 2010, 2014; Kennedy et al., 2016). Even the promoters of the genetic engineering experiment — funded and protected from law suits by the pharmaceutical-medical-government (PMG) complex (the CDC, FDA, Department of Defense, etc.; see Fleming, 2021; also Children's Health Defense, 2021) — acknowledge that correct translations of natural mRNA is “context-dependent”. However, the so-called “science” underlying the rollout of the mXNA experiment is local, shallow, and does not take account of the multi-layered biosignaling context of the whole body. The mainstream corporate narrative depends on “mechanisms” that do not have free will, do not have genuine creative powers of thought, and are not morally responsible for their actions (but see Kyrie & Broudy, 2022).

WHAT DOES IT MEAN FOR MRNA TO BE “CONTEXT-DEPENDENT”?

Shi et al. (2019), Boo and Kim (2020), and Nance and Meier, 2021) — all funded mainly by the PMG according to their published statements — acknowledge that alterations in natural mRNA can only be understood adequately in terms of the containing context. The rule is that containing contexts always outrank and over-ride the ones contained within them (see Gryder et al., 2013; Oller,

2010, 2014; Kennedy et al., 2016). Nonetheless, the experiments suggesting how the injectables might work in theory are based on *in vitro* de-contextualized studies of mXNAs, or on models depending of studies with mice or animals that did not survive the experiments (see Blaylock, 2022). The “safety” studies do not take account of the containing context dependencies that determine how the global experiment underway in the bodies of live human beings will unfold.

There Are Multiple Layers of Context

Shi et al. (2019), in a paper with 536 citations on the Web of Science³ on October 5, 2022, stressed the fact that there are “multiple layers” of relevant context in the native transcription of RNAs from DNA and the interaction of natural mRNA with the ribosome systems where protein products, enzymes, and essential peptides are produced. They say:

We emphasize the importance of context for RNA modification regulation and function [p. 640]. . . . some of the key questions remaining to be addressed in the field are most likely to be context-dependent. The writers and erasers [operating on natural mRNA coding sequences] likely target different groups of transcripts in different cell types and in different biological processes. The erasers and different readers may also modulate . . . specific regions of a transcript, leading to different functional outcomes. How are the selectivity, both transcript selectivity and site selectivity, achieved? Different readers may affect distinct sets of transcripts in different cell types or tissues. How are these readers recognizing their target transcripts? How are writers, readers, and erasers regulated and integrated into diverse biological signaling and regulation? RNA and RNA modifications effector proteins can exist in different cellular compartments. The correct cellular context should be an essential element of future functional investigations of RNA modifications [p. 647].

Nance and Meier (2021, p. 752) acknowledged the importance of context, saying: “Natural RNA modifications are known to be context dependent”, so it might be expected that answers to questions about how mXNAs would work in living humans would be sought in advance. But, in citing Boo and Kim (2020), who remarked that that “hundreds of different RNA modifications have been . . . shown to regulate mRNA stability, consequently affecting diverse cellular and biological processes” (p. 400), Nance and Meier are effectively acknowledging that the “hundreds” of known “different RNA modifications” could not have been taken into consideration. Boo and Kim conclude (p. 405) in calling for the construction of a complete “list of RNA modifications that affect mRNA stability” and that can “elucidate the underlying molecular mechanisms”.

But the List Does Not Exist

But, there is no such list and the “mechanisms” so far “elucidated” in the research literature are shallow, exclusively local (in isolated cell cultures or in short-lived experiments with nonhuman models), and cannot enable reasonable forecasting of what the biosignaling systems of the human body will do with the mXNAs. For all these reasons the “mechanisms” already “elucidated” are only tangentially and hypothetically applicable to the ongoing global experiment. The human genome as contained in nuclear and mitochondrial DNA consists of a string of about 3 billion base pairs in each of the body’s billions of nucleated cells and 16,569 base pairs in a much greater multitude of mitochondria. All of them talk to each other and to RNAs and proteins. The interactions that we know are taking place by the trillions of trillions consist of DNA with DNA, DNA-RNA, RNA-RNA, DNA-protein, RNA-protein, and protein-protein cross-talk. How this multitude of multitudes of interactions work in human bodies is yet to be well understood and cannot logically be explained in terms of localized “mechanisms”. In the meantime, the genetic engineers are relying mainly on isolated human cell lines processed *in vitro*. What *in vivo* studies they have done have

³ This paper was the second most cited out of 306 articles in the core index that deal with “context dependent mRNA” functions.

involved the level of a bacterium, yeast, or in some cases “transgenic” mice, or other nonhuman mammals that did not survive the experiments (see Blaylock, 2022, and his references). The clinical studies with human beings, we know (Horowitz, 2021) have been too brief, poorly designed, falsely represented to the public, and have left the essential question about preventing transmission of disease unaddressed (see Bob Roos, Member of the European Parliament, 2022). In fact, a Pfizer spokesperson on October 11, 2022, admitted that the question of whether or not the BNT162b2 product could prevent transmission of the SARS-CoV-2 virus, was never addressed. She says, “Did we know about stopping immunization [transmission] before it was sent to the market? No, uh, these uh, we had to really move at the speed of science.” She went on in the hearing to mention that everything had to be done “at risk”. So, researchers might ask, what studies were relied upon? It turns out that what the genetic engineers knew when they started the global experiment was largely based on incomplete understanding of how transcription and translation work in organisms at the level of prokaryotes such as bacteria, or even monocytic eukaryotes such as yeasts.

Generalizing from Yeast to a Human Being? Seriously folks?

In eukaryotes such as humans are, nearly all of our cells, excepting our red blood cells, have a defined nucleus containing the library of DNA information for the integration of all our organ systems. Timmers and Tora (2018) point out that prokaryotes, such as bacteria, unlike yeasts do not have a defined nucleus contained within the well-guarded series of baffles inside the entothelial reticulum of eukaryotes as in *homo sapiens*. Also, the protein producing systems in nucleated cells are more productive in eukaryotes than in prokaryotes. Timmers and Tora note that whereas “a single mRNA molecule . . . yields on average ~ 1,000 protein molecules in yeast” in more complex eukaryotic cells like our own, one mRNA molecule typically produces “3,000–10,000” protein molecules. Given the 30 billion or so mRNA molecules injected into any human recipient of at least one of the experimental COVID-19 products (see Fleming, 2021, p. 99), if all goes according to the plan of the genetic engineers, thousands of billions of the SARS-CoV-2 spike protein should be produced in every recipient. To form an idea of how much the genetic engineers know about what is likely after multiple doses, we might consider how well they are doing in deciphering the control processes (the buffering functions) in the simplest prokaryotes known to biology. What is known for sure about the buffering functions in, say, a particular yeast? Timmers and Tora put it this way:

Careful studies in yeast have revealed the phenomenon of transcript buffering, and we provide future directions into the understanding of the molecular mechanisms [“systems” would be a better word-choice here] involved. Discovery of this phenomenon relied on methods to measure the absolute rates of both mRNA synthesis and mRNA degradation. Several open questions exist, such as what is the origin and nature of the buffering signal? [They don’t know where the buffering signals come from in a yeast?] Does the signal emanate from the cytoplasm to be received in the nucleus or is the communication bidirectional? What is the dynamic range of transcript buffering? Is this process connected to translational efficiency . . . ? . . . We expect . . . a surge in investigations and understanding of transcript buffering in mammalian cell systems. This understanding will aid in devising more effective therapeutic applications of such inhibitors to balance or to further imbalance mRNA synthesis and degradation in pathological conditions. By the same token, this will bring our appreciation of these opposite processes controlling mRNA levels in eukaryotes to the next level.

The upshot is this: if the genetic engineers do not yet understand how “transcript buffering” works at the level of a bacterium or yeast, how can they know how their mRNA experiment is going to work over the long haul in the far more complex systems of human beings?⁴

The next series of stops on the tour will involve specific clinical outcomes. Some of them seem likely to follow from protein/peptide/motifs spinning off from the billions of exemplars of the

⁴ Thanks to a comment from Dr. Anya Kreynes, dated January 28, 2023 and addressed to the Editor in Chief, I have corrected and clarified all the references to yeasts on this page. The error pointed out by Dr. Kreynes was the false statement that yeasts are prokaryotes when what was intended was to emphasize that unlike bacteria, they are monocytic eukaryotes, though still far less complex than human nucleated cell systems.

mXNA spike coding sequence in injected persons. In the language of the PMG, what are the observed “side effects” of the injections?

Playing Global Russian Roulette

In my metaphor of “global Russian Roulette”, the pistol is the syringe in the hand of some clinician and each injection amounts to another spin of the cylinder and another pull of the trigger. According to the mainstream corporate narrative about the risk/benefit ratio — from the CDC, the FDA, and other parties who are getting rich, or richer, on the continued use of the COVID-19 injectables (see the Children’s Health Defense Team, 2021) — there are many chambers in the cylinder and the risk of harm is outweighed by the likelihood that pulling the trigger will destroy many COVID disease agents and will keep the player from asymptotically transmitting, or getting sick, or even dying with COVID-19 disease. If the benefits are large and risks are small, why not spin the cylinder and take the shot? But, the relevant independent research with ordinary and electron microscopes shows that all *of the chambers are loaded with injurious and potentially lethal components*. Those components are coming to light in the vials of fluids in vials of the injectables and in the blood of patients receiving them (see Hughes, 2022, also see my answer to him about what is in the “vaccines”, Santiago, 2022; and Tuuminen 2022 on Benzi Cipelli, 2022).

THE FINAL CLINICAL OUTCOME — SORRY YOU’RE DEAD

As Oller and I showed in an earlier paper (2022), hospital data, actuarial records, autopsies, and clinical observations of injuries and deaths show that there really is something like a game of Russian Roulette being played out, not in Monte Carlo, but on a global scale. The all-cause-mortality results suggests about a 40% across the board increase (Berdine, 2022; Mercola, 2022a, 2022c) since the “emergency use” injectables were authorized. This index is the gold standard for health because it can hardly be mis-measured. It is difficult to manipulate or make mistakes in measuring mortality. Dead is dead and hard not to notice. The body also has to be disposed of. By contrast, disease incidence, number of cases diagnosed, prevalence of this or that injury or infection, hospital admissions, visits to emergency rooms, heart attacks versus fatal seizures, etc., can easily be mistaken or misrepresented, but death itself is hard to hide and is not easily mistaken for any other condition. That being established, it becomes irrational now that shut-downs have been discontinued in 2022 to deny that the COVID-19 injections must now be taken as the main worldwide factor, as Ed Dowd (2022a, 2022b) has concluded, to explain the surging numbers of dying people, particularly in the 18 to 39 age bracket. Mercola’s analysis (2022a, 2022c), and see his references to the work of others, also seems conclusive. The injections are the most likely cause of the continued increase in all-cause-mortality.

Foreign Entities in Blood of Recipients and in the Injections

There is mounting clinical evidence from examinations of foreign particles, entities, and constructions in the blood of recipients compared with microscopic investigations finding the same strange entities — except for the enormous blood clots in the veins and arteries of dead vaccinees (Trigoso, 2022) — in the warmed up fluids contained in COVID-19 “vaccine” vials (Jeon, 2022; Lee et al., 2022; Benzi Cipelli, 2022; Hughes, 2022). The clotting can be explained: the vials contain many sharp edged foreign constructions, ones that often resemble graphene oxide and similar materials used in microcomputing devices. Those foreign objects damage the red blood cells and the vessels they have to flow through. The CDC denied that anything like graphene materials were included in any of the EUA injectables, but Jeon (2022), followed up by Lee et al. (2022), found foreign entities difficult to explain in any other way. Later, Benzi Cipelli et al. (2022) corroborated the findings of

Jeon, and of Lee et al., and Hughes (2022) presented additional corroborating findings from many other distinct investigations by different teams of competent and independent researchers — ones that have no financial interests to cause them to prefer one outcome over another. They are simply trying to find out what is going wrong with this global experiment in biological genetic engineering.

CARDIAC INJURIES AND CATASTROPHIC SYSTEMS FAILURES

When hundreds of healthy performing athletes are falling dead on camera (Heilman, 2022), when 561 fully vaccinated children and adolescents are more likely to be taken to see the doctor than the matched group of 561 less vaccinated ones in the same practice (Lyons-Weiler & Blaylock, 2022), when tens of thousands of mature adults are being diagnosed with circulatory problems and multiple systems disorders that depend on the flow of blood and lymph (Blaylock, 2022; Seneff et al., 2022), we must suspect that the foreign objects in the blood — and in the lymph ducts based on findings of Lee, et al. (2022) with centrifuged blood plasma from recipients of the injections — are coming directly from the SARS-CoV-2 mXNA-containing injections. The shots are implicated. Also, the problems appear to be worse and more likely to be serious or fatal after more than one of the injections (Gundry, 2021; Guetzkow, 2022; Krug et al., 2022; Patone et al., 2022; Sun et al., 2022; Xie et al., 2022). If one jab is injurious, up to 4 additional shots of similar composition (the US FDA approved a fifth injection of Pfizer or Moderna, seven months ago, on March 30, 2022; Associated News Services, 2022) will probably be more so. Evidence of injuries, by his own testimony, caused Surgeon General, Joseph A. Ladapo, MD, PhD (2022) to issue the press release on October 8, 2022 (Durden, 2022) recommending against COVID-19 injections for persons from infancy through age 39. The Florida Department of Health found

an 84% increase in the relative incidence of cardiac-related death among males 18-39 years old within 28 days following mRNA [make that an mXNA] vaccination.

In Italy a population-based study of about 3 million patients associated the mXNA injections to myocarditis/pericarditis. Massari et al. (2022) reported the highest risk in 12 to 39-year-olds within a week of an mXNA injection. Then, on January 25, 2022, Oster et al., reported an increased risk of myocarditis, especially in adolescent/young males, following the first mXNA injection and found it to increase even more after the second shot. It was noteworthy that 96.4% of patients in the Oster study (784/813) were hospitalized. Their condition was obviously serious.

INJURIES TO REPRODUCTIVE SYSTEMS

The next stop on our tour is about the impact of COVID-19 injections on reproductive systems. The first published work on bad clinical outcomes for female reproductive systems showed strange entities in blood samples from women with irregular menstrual cycles or vaginal bleeding (Jeon, 2022; Y. M. Lee et al., 2022; K. M. Lee et al., 2022). Semen count and motility were also observed to be reduced in sperm donors after exposure to a BNT162b2 injection (Gat et al., 2022). Without going into too much detail, it is with respect to fertility, embryological growth, and tumor producing hormones, where the profoundly important “Kozak sequence” (see the Appendix, Part B, Figure 3) — a biologically preferred coding sequence in natural mRNAs being translated into protein/peptide amino acid sequences — seems to come into play with respect to the COVID-19 injectables. Because of the many m1Ψ substitutions in the unnatural mXNA coding sequence, the role of the Kozak sequence is less certain than with any natural mRNA produced by the native DNA of the body. Among the possible protein/peptide products in the 31 ORFs mentioned earlier are multiple

strings that are near homologues of ones known to be involved in both male and female human reproductive systems.

ANTIBODY DEPENDENT ENHANCEMENT

Another of the clinical outcomes being observed by independent researchers and practitioners is what is called “antibody dependent enhancement” (ADE). The risk pointed to in this line of research is similar to, and dependent upon, what Lyons-Weiler (2020) termed “pathogenic priming”. Cardozo and Veasey argued in 2020 that participants in the initial COVID-19 experimental trials were inadequately informed of the risk associated with ADE. They explained why persons used in the trials could become sensitized to “more severe disease than if they were not vaccinated”. They argued that the risk was present for all the injectables regardless of whether they were “composed of protein, viral vector, DNA or RNA and irrespective of delivery method” (p. 1). They noted that patients were not being adequately informed even in the “clinical trial protocols” and subsequent clinical outcomes after the rollout of Pfizer, Moderna, and AstraZeneca injectables have confirmed their fears. In a study of 22,072,550 cases, 848,911 hospitalizations, and 175,070 deaths in the UK attributed by them to COVID-19, Emani et al. (2022) acknowledged that

since December 20, 2021 . . . [there have been] a significantly increased proportion of SARS-CoV2 cases, hospitalizations, and deaths among the vaccinated; and a decreased proportion of cases, hospitalizations, and deaths among the unvaccinated.

The obvious conclusion the authors do not state overtly is that, not only are the injections increasingly ineffective as the doses accumulate, but they are increasingly likely to be harmful and even fatal to recipients. The persistent reference in mainstream publications to the worse than non-existent “vaccine effectiveness” — worse because the injections are sickening and killing people — enables the many who are in step with the mainstream cadence to continue promoting the next spin of the cylinder in the ongoing global game of Russian Roulette.

Natural Immunity Is Better

While the mainstream promoters of the injectables keep urging more and more shots, evidence that natural immunity is better weighs overwhelmingly against repeated mRNA injections. The so-called “vaccines” in the global experiment are neither safe nor effective.

Israel

Gazit et al. (2021) reported on a study of 2.5 million people in Israel where the state has enacted, arguably, the world’s most enforced COVID-19 mandates for lockdowns and injections. They found that

SARS-CoV-2-naïve vaccinees had a 13.06-fold (95% CI, 8.08 to 21.11) increased risk for breakthrough infection with the Delta variant compared to those previously infected, when the first event (infection or vaccination) occurred during January and February of 2021. The increased risk was significant ($p<0.001$) for symptomatic disease as well. When allowing the infection to occur at any time before vaccination (from March 2020 to February 2021), evidence of waning natural immunity was demonstrated, though SARS-CoV-2 naïve vaccinees had a 5.96-fold (95% CI, 4.85 to 7.33) increased risk for breakthrough infection and a 7.13-fold (95% CI, 5.51 to 9.21) increased risk for symptomatic disease. SARS-CoV-2-naïve vaccinees were also at a greater risk for COVID-19-related-hospitalizations compared to those that were previously infected.

Qatar

Chemaitelly et al. (2022), between February 28, 2020 and June 5, 2022 (approximately 27 months) used three different matched cohorts of 407,214 “unvaccinated” persons in Qatar divided into two groups

in each of their studies. The two groups compared in each of the three cohorts consisted of persons who were diagnosed as having had a (1) “SARS-CoV-2 primary (first) infection prior to vaccination (designated the primary-infection cohort) versus those who were also unvaccinated but not infected, and (2) “the national (control) cohort of . . . infection-naïve and unvaccinated (designated the infection-naïve cohort)” (p. 6). They asked how likely is re-infection with severe, critical, or fatal results for either group. They found that natural immunity gained from a “primary infection . . . was 97.3% (95% CI: 94.9-98.6%)” effective against re-infection “irrespective of the variant of primary infection or reinfection, and with no evidence for waning” (p. 15).

Iceland

Eythorsson et al. (2022) in Iceland reported in their “population-based cohort study” that “reinfection during the first 74 days of the Omicron wave in Iceland” revealed unsurprisingly that a longer lapse of time after the “initial infection was associated with a higher probability of reinfection” but they did report being surprised to find that persons who received more than one dose of the COVID-19 injectables had a “slightly higher probability of reinfection compared with 1 dose or less” (p. 3 of 4).

WHY IS THE GLOBAL EXPERIMENT GOING WRONG, OR, IS IT GOING AS INTENDED?

It might be wise to ask what is going wrong with the COVID-19 injections? The next stop on our tour is in a laboratory where “transgenic mice” were studied to deliberately produce the sort of auto-immune response via pathogenic priming to a DNA gene of influenza virus. Our stopover there leads to a more sinister question: what if the global experiment is not actually taking an unintended course toward disorders, diseases, and deaths on a wide scale? Is it impossible that such catastrophic results, are perhaps being intentionally programmed into human DNA by the genetic engineers in the background? From the research reported by Jeon, (2022), Y. M. Lee et al. (2022), and Hughes (2022), we can be certain that there are foreign materials in the COVID-19 injectables that are not supposed to be there. Given Broudy’s arguments about how the mainstream corporate narratives have been guided by tried and true propaganda techniques (Broudy & Arakaki, 2020; Broudy, 2021; Broudy & Hoop, 2021; Broudy & Kyrie, 2021; Kyrie & Broudy, 2022), would it be a impossible leap of logic to suppose that serious deception is taking place on a worldwide scale?⁵

Transgenic Mice

In 2000, Reed et al. published an article pertaining to a “transgenic” mouse — one incorporating the influenza virus into its own genome. The study is important because it anticipates the very sort of “transhumanist” genetic engineering discussed in detail by Kyrie and Broudy (2022) that seems now to be underway with the COVID-19 injectables being used not on mice but on human beings. With respect to the genetically modified mice, Amy J. Reed explained to Franklin Hoke (2001) why the experiment with mice incorporating a foreign influenza gene into their own genome was seen as revolutionary. “Other scientists have seen data like this [showing self-immune disease] in their experiments,” said Reed, “but they weren’t able to assure themselves that they were monitoring a self-reactive immune response sparked by a viral infection”. However, with mice whose DNA was genetically modified to incorporate the influenza gene, the researchers knew exactly what was causing the immune defenses of the mice to attack and destroy their own tissues — it was the influenza gene incorporated into the mouse’s own DNA. Could something similar be happening now in human beings on a global scale?

⁵ Here from an anonymous source is a series of physical demonstrations of how resonant properties of events involving material entities can enter into lockstep cadences. Is it inconceivable that injected individuals might be manipulated in the manner of “transgenic mice” or perhaps influenced in yet-to-be-determined ways by foreign materials injected into their bodies? We do know that some of the mXNA delivered in the injectables is incorporated in the recipient’s nuclear DNA (Aldén et al., 2022).

Transgenic Humans?

The problem to which I am calling attention in this paper is that the mXNA of the COVID-19 injections is not native mRNA and, according to its proponents, is designed not only to “cloak”

itself from the host’s immune defenses, but also to cause each of the invaded and commandeered ribosomes to persevere through multiple repetitive readings of the same mXNA coding sequence. The idea, according to proponents of that synthetic nucleic acid — one of the many being created by genetic engineers (Chaput & Herdewijn, 2019), all of them properly abbreviated as XNAs — is to “enhance the synthesis of antigens [more spikes]” in order to “drive high levels of [spike] protein production” (Nance & Meier, 2021). The important problem to which I have called attention in this article is that in addition to the multitude of supposedly perfect SARS-CoV-2 spike proteins, which are themselves potentially lethal over the long haul as we have seen in this review, there are a plethora of other possible outcomes from the specific mXNA coding sequence designated as a “modified” mRNA that is designed supposedly only to produce that particular spike. Unsurprisingly, some of the harmful products that are predicted with bioinformatic tools are already being seen in the adverse events associated with the COVID-19 injectables.

Conclusions

The world’s population is being experimented on with injections of largely unknown composition and unknown long-term consequences. When the rollout of COVID-19 injectables began, many people were evidently eager to receive the first injection. The emergency use products were advertised by the government oversight agencies, by the pharmaceutical manufacturers, and by the mainstream media world-wide, to have the power to prevent infection and possible death by SARS-CoV-2 and its downstream variants. Enthusiasm for the injectables was countered at the start by relatively few dissenters who doubted one or another aspect of the mainstream corporate narrative. As pressure from the government oversight agencies increased to threats of travel bans, job losses, and worse, some dissenters received one or more injections against their will (see [Uninformed Consent, 2022](#)). As the global experiment continues to unfold, it appears now that the number of those who are still willing and able to receive additional injections has waned from more than half the world’s population to somewhere in the neighborhood of a little less than 1% for US citizens taking the fifth “booster” as of March 30, 2022 (Associated News Services, 2022). In this paper, I have conducted a tour of some of the reasons for the distrust of the mainstream corporate narrative playing out in the media. For a confirmation that I am reading things correctly in this respect, see the conversation between Megyn Kelly and Joseph Ladapo, MD, PhD, at [this link](#). In the final analysis, the decision about whether to take an injection or not, should rest with the individual, not the government. With respect to children, most parents are like Megyn, Ladapo, and me: we are unwilling to hand over such an important choice to any bureaucrats at the CDC, FDA, etc. We are adamantly opposed to handing such decisions off to transnational entities such as the World Health Organization, the World Economic Forum, or any consortium of such entities.

**Part A. The World Health Organization’s International Nonproprietary Names
record, 2020, Number 11889 — DNA Coding Sequence for the SARS-CoV-2
Spike Protein**

What follows here is the DNA coding sequence of 3,821 nucleotides underlying the BNT162b2 and mRNA-1273 spike protein of SARS-CoV-2 as published by the World Health Organization's . To get the mRNA version it is only necessary to substitute U for T in all 728 positions where T appears, and to get the mXNA version, it is necessary to substitute m1 Ψ in that same position for the U 728 times.

ATGTTTCGTGTTCTCTGGTGCTGCTGCCCTCTGGTGTCCAGCCAGTGTGTGAACCTGACCACCAGAACAACAGCTGCCCTCCAGCCTACACCAACAGCCTTACCAGAGGCGTGTACTACCCCGACAAGGTGTTTCAGATCCAGCGTGTCTGCACTCTACCCAGGACCTGTTCTCTGCCTTCTTTCAGCAACGTGACTCTGGTTCCACGCCATCCACGTGTGCCGGACCAATGGCACCAGAAGATTTCGACAACCCCGTGCTGCCCTTCAACGACCGGGTGTACTTTGCCAGCACCGGAGAAGTCCAACATCATCAGAGGCTGGATCTTTCGCGACCACAGCTGGACAGCAAGACCAGAGCCTGTGATCGTGAACAACGCCACCAACGTGGTATCAAAGTGTGCGAGTTTCAGTTCTGCTCAACAGACCCTTCTCTGGGCGTCTACTACCACAAGAACAACAAGAGCTGGATGGAAAAGCGAGTTTCCGGGTGTACAGCAGCGCCAACAACCTGCACCTTCGAGTACGTGTCCAGCGCTTTCCTGATGGACCTGGAAGGCAATCGGAAGGCAATCAAGCACTTCAAGATCTACAGCAAGCACACCCTTCAACCTCTGTGCGGCTTGCCTCAGGGCTTCTCTGCTGTGGAACCCCTGGTGTGATCTGCCCATCGGCATCAACATCACCCGGTTTCAGACACTGTCTGGCCCTGCACAGAAGCTACTCTGACACTGGCGATAGACAGCAGCGGATGGACAGCTGGTGGCGCCGCTTACTATGTGGGCTACCTGCAGCCTAGAACCCTTCTGCTGAAGTACAACGAGAACGGCACCA TCACCAGCCGCGTGGATTGTGCTCTGGATCCTCTGAGCGAGCAAAAGTGCACCTTGAAGTCTCTACCGTGGAAAAGGGCATCTA CCAAGACCAGCAACTTCCGGGGACGCCCAACCTCCATCCATCGTGGCGTTCCCAATATACCAATCTGTGCCCTTCCGGCGAGGTGTT TCAATGCCACCAGATTTCGCTCTGTGTACGCTTGAACCCGGAAGCGGATCAGCAATTCGCTGGCCGACTACTCCGTGCTGTATACA CTCCGCCAGCTTCAGCACCTTCAAAGTGCTACGGCGTGTCCCTACCAAGCTGAACGACCTGTGCTTCAAAAACGTGTACGCCGACA GCTTCGTGATCCGGGGAGATGAAGTGTGCGGCAAGTTGCCCTTGCACAGACAGGCAAGATCGCCGACTACAACATCAAGCTGCCCG ACGACTTCAACGGTGATGTGTGATTCGCTTGGAAACAGAACAACTTGGACTCCAAAGTCGGCGGCCAACTACATACTTACCTGTACCGGCT GTTCCGGAAGTCCAATCTGAAGCCCTTCGAGCGGGACATCTCCACCAGATCTATCAGCGCCGCGCAGCACCCCTTGTAACGGCGTG GAAGGCTTCAACTGCTACTTCCCACTGCAGTCTACGGCTTTCAGCCCAAAATGGCGTGGGCTATCAGCCCTACAGAGTGGTGG TGCTGAGCTTTCGAAGTCTGTCATGCCCTGCCACAGTGTGCGCGCCTAAGAAAAAGCACCATCTCGTGAAGAACAAATGCGTGAATTTCAACTTCAACGGCGCTGACCGGCGCGTGTGTGACAGAGCAAGAAGTTCTTCCCTGCCATCCAGCAGTTTGGCGGGGATATCGCCGATACCACAGACGCCGTAGAGATCCCCAGACTGGAATCTTGACATACCCTTGCAGCTTCGGCGAGTGTCTG TGATCACCCCTGGCACCAACACCAGCAATCAGGTGGCAGTGTCTGTACCAGGACGTGAACCTGTACCGAAGTGGCCGTGGCCATTCA CGCCGATCAGCTGACACTCATGTGCGGGGTGTACTTCCACCGAGCAAAATGTGTTTCAGACAGACAGCCGCTGTCTGATCGGAGCC GAGCAGCTGAACAATGCTACGAGTGGCAGATCCCCATCCGCGCTGGAATGTTCGCGCCAGCTACGACACAGACAAACAGCCCTC GGAGAGCCAGAAGCTGTGGCCAGCCAGAGCATATTCCTACACAATGTCTCTGGCGCCGAGAACAGCGTGGCCTACTCCAACA ACTCTATCGCTATCCCCACCAACTTCACCATCAGCGTGAACACAGAGATCTTGCCTGTGTCCATGACCAAGACCAGCGTGGACTGC ACCATGTATATCTCGCGCGGATTCACACAGAGTGTCTCCAACTGTCTGTGCACTACGGCAGCTTCGTACCCAGCTGAATAGAGCCCT GACAGGGATCGCGCTGGAAACAGACAAGTACCCCAAAGAGGTGTGTTCGCCCAAAGTGAAGCAGATCTACAAGACCCCTCTATCAAG GACTTCGGCGGCTTCAATTTCAGCCAGATTTCGCCGATCTAGCAAGCCCAAGCAAGCGGAGCTTCATCGAGGACTGTCTGTTCACA AAAAGTGACACTGGCCGACGCCGGCTTCATCAAGCAGTATGGCGATTGTCTGGGCGACATTGGCCGAGGGATCTGATTTGCG CCCAGAAGTTTAACCGAGCTGACAGTGTCTGCTCTGCTGACCCATGAGATGATCGCCCAAGTACACATCTGCGCTGTGGCCGG CACAATCACAAGCGGCTGGACATTTGGAGAGAGCGCGCTCTGCAGATCCCCCTTTGCTATGCAAGTATGCGCTACCGGTTCAACGGC ATCGGAGTGACCCAGAATTGCTGTACGAGAACCAGAAGCTGATCGCAACCAAGTTCAACAGCGCATCGGCAAGATCCAGGACA GCCTGAGCAGCACAGCAAGCGCCCTGGGAAAGCTGCAGGACGTGGTCAACCAGAATGCCCAGGCACTGAACACCCTGGTCAAGC AGCTGTCTCTCAACTTTCGGCGCCATCAGCTCTGTGTGTAAGCATATCTCTGAGCAGACTGACACCTCTGTAGGGCGAGGTGCAGAT CGACAGACTGATCAGGAGGCACTGACAGCTCCAGACATACGTGACCCAGCAGCTGTACAGAGCCGCGGAGATTAGAGCCTCT GCCAATCTGGCGGCCACCAAGATGTCTGAGTGTGTCTGGGCCAGAGCAAGAGAGTGGACTTTTGGCGCAAGGGCTACCACCTG ATGAGCTTCCCTCAGTCTGCCCTCACGGCGTGGTGTCTTCTGCAGTGTACATATGTGCCCGCTCAAGAGAAGAATTTACCACCCG C TCCAGCCATCTGCCAGCAGCGGCAAGGCCACTTTCCTAGAGAAGGCGTGTTCGTGTCCAACCGCCACCATTTGTTCTGTGACACG CGGAACCTTCTACGAGCCCGATCATCACCCAGACAACACTTCTGTCTTGGCACTCGCAGCTGATCGGCATTTGTGAACA ATACCGTGTACGACCCTCTGCAGCCCGAGCTGGACAGCTTCAAAGAGGAAGTGGACAAGTACTTTAAGAACCACAAAGCCCGA CGTGGACCTGGGCGATATCAGCGGAATCAATGCCAGCGTCTGTGAACATCCAGAAAGAGATCGACCGGCTGAACGAGGTGGCCA AGAATCTGAACGAGAGCCTGATCGACCTGCAAGAACTGGGGAAGTACGAGCAGTACATCAAGTGGCCCTGGTACATCTGGCTGACACG CTTTATCTGCCCGACTGATTTGCCATCGTGTGGTTCACAACTCATGCTGTGTGTCATGACCAGCTGTCTAGCTTACCTGAAGGGCTG TGTGAGCTGTGGCAGCTGCTGCAAGTTTCGAGGAGGACGATTCTGAGCCGCTGCTGAAGGGCGTGAAGCTGCAGTACACATGA

Part B. What Is the Kozak Sequence and Why Is It Important to the mXNAs in the BNT162b2 and mRNA-1273 Injections?

What is the Kozak sequence?

The Kozak sequence is the optimal nucleic acid pattern believed by many to ensure ribosomal recognition of the start codon at the initial stage of translating an mRNA sequence of nucleotide bases into a sequence of amino acids to build a peptide/protein product. The one that is intended to be produced in the BNT162b2 and mRNA-1273 injections is precisely the amino acid sequence listed in Part C of this Appendix. In natural mRNA, the canonical Kozak sequence is embedded in the 5' end within what is referred to as the “untranslated cap region”. That region at the beginning of the mRNA is the critical part that introduces the mRNA to the ribosome. As it communicates with the biosignaling systems in the cell, it functions something like a passport with a built-in birth certificate that identifies this particular mRNA as native, i.e., self, rather than foreign. If the identification of the mRNA as belonging to the self, rather than coming from some potential pathogen/invasor of the cell, goes as expected, the ribosome accepts the mRNA message as generated from the body’s own natural DNA and is thus authorized to begin constructing the protein/peptide products specified by that mRNA. If all goes well, that particular mRNA in the cytoplasm of the cell will not be attacked and destroyed by the police/military cytotoxic immune defense cells of the body. The unknown quantity added to the mix 728 times in the coding sequence for the SARS-CoV-2 spike is the xeno-component, N1-methylpseudouridine (m1Ψ). Also, that part of the mXNA in the injections (see Part A above in this Appendix) comes into play after the “untranslated region” consisting of the protective cap. According to promoters of the injections, Nance and Meier (2021, p. 750, that cap consists of the following 54 nucleotides: GAGAAUAAACUAGUAUUCUUCUGGUCCCCACAGACUCAGAGAGAACCCGCCACC. The crucial point to keep in mind with respect to this cap sequence is that it contains 10 uridine (U) nucleotides that the mXNA replaces with 10 N1-methylpseudouridines (m1Ψ). Will these unnatural nucleotides in the recipe automatically have the results predicted by Nance and Meier (2021)?

Why is the Kozak sequence important?

The canonical Kozak sequence (2002) is described in Figure 3 relative to the BNT162b2 cap which is followed by the intended spike protein coding sequence beginning with the yellow highlighted AUG four lines from the top at right hand side of the figure. As shown there, the Kozak consensus sequence appears in the BNT162b2 cap reading from the 5' toward the 3' end of the mRNA as -GCCACC AUG-. In 1984, Kozak showed that a single nucleotide change in mRNA could have a dramatic impact on translation. Two years later, in 1986, she showed that translation is particularly sensitive to changes in positions -1, -2, and +4 in the coding sequence relative to the triadic start sequence, AUG. Seventeen years later, de Angioletti in 2003 (published in 2004) found that positions substantially farther removed from the AUG start sequence can play a major role: “...+45 G → C is the first mutation found in the Kozak sequence (GACACCATGG) of the β-globin gene and the first one at the position -6 upstream [from] the ATG [the DNA source of the mRNA AUG start sequence]”. This one change results in a potentially fatal form of anemia. More than another decade later, Li et al (2017) found important changes owed to nucleotides at positions -15 and -9.

Such research findings provide the basis for the discussion to follow where I concentrate on positions the +2, +4, and -3 in the Kozak sequence as defined in Figure 3. Returning then to the potential consequences of the m1Ψ substitutions, Parr et al. (2020) reported high protein expression, evasion of cytotoxic immune cells, and enhanced sensitivity to translation regulating microRNAs and RNA binding proteins from mXNA-containing m1Ψ substitutions for uridine

studied in cell lines *in vitro* — in non-living artificially manipulated systems. The implied suggestion is that **m1Ψ** substitutions might show all the same advantages over native mRNA *in vivo* — that is in living human recipients of the COVID-19 injections. But is that true? The clinical results do not seem to be consistent with the *in vitro* findings of Parr et al. The reason? The experiments with isolated cell lines and components transfected into them or otherwise manipulated artificially cannot take account of the larger context, multiple layers deep, that we know exists in the human body. In that light, the *in vitro* experiments of Parr et al. and those cited by them are apt to be misleading if generalized to the global human population.

When a ribosome begins reading the coding sequence in an XNA, presumably it is looking for the canonical start codon, an AUG. However, in natural mRNAs the start codon may also be a CUG, or GUG. In fact, according to Kearse and Wilusz (2017), the alternate start codons are used more frequently, than formerly supposed:

Although it was long thought that eukaryotic translation almost always initiates at an AUG start codon, recent advancements in ribosome footprint mapping have revealed that non-AUG start codons are used at an astonishing frequency. These non-AUG initiation events are not simply errors but instead are used to generate or regulate proteins with key cellular functions, for example, during development or stress. Mis-regulation of non-AUG initiation events contributes to *multiple human diseases, including cancer and neurodegeneration* [my emphasis]

But, what will happen with repeat AUG start codons influenced by **m1Ψ** in coding sequences that are not conformable to the optimal Kozak sequence of Figure 3? It is reasonable to expect that secondary structures formed from the experimental mXNA in the COVID-19 injectables are likely to activate or suppress normal cell activities in unpredictable but almost certainly harmful ways. For some of the likely problems see Almeida et al. (2013), Borger et al. (2020), Seneff et al. (2022). What is certain is that a misreading can lead disorder, disease, and even death. Xia (2021) wrote:

The two mRNA [actually mXNA] vaccines [Pfizer and Moderna] both used GCCACCAUG, but not the codon after the start codon AUG, for two good reasons. First, while the -3R [purine A or G] has been demonstrated repeatedly to enhance translation initiation, the effect of +4G, as well as nucleotides downstream, on translation initiation has been inconclusive. The preponderance of +4G was explained by the amino acid constraint hypothesis as follows. About 60% of the proteins experience N-terminal methionine excision (NME) which requires a small and nonpolar amino acid such as alanine and glycine. Alanine is encoded by GCN and glycine by GGN, leading to a high frequency of G at the +4 site. There is little evidence that +4G and downstream nucleotides contribute to translation initiation. Second, the second amino acid in the spike protein is phenylalanine, which ensures that NME does not happen. Changing it to GCG (encoding alanine) would result in NME, leading to unpredictable changes in the S [spike] protein. For these reasons, the first codon is not considered in Kozak consensus optimization.

Setting aside all the uncertainties Xia has raised, there is another potential problem noted by Kozak (1990). Shabalina (2013) wrote:

Emerging evidence shows that “silent” substitutions carry a wealth of information, which is written over the encoded amino acid sequence, and that this information can be used to regulate translation speed, protein homeostasis, metabolic fate and even posttranslational modifications....

Liu et al. (2021) found “after the first inoculation . . . consistent alterations in gene expression of many different immune cell types”. It can be inferred that the products spinning off from the commandeered ribosomes are causing damage and confusion in immune cells themselves. Widespread clinical evidence of harm has already been demonstrated in findings reported by Brown et al., (2021) in the *MMWR*; also by Kampf (2021a, 2021b, 2022), and by Oller and Santiago (2022). Röltgen et al. (2022) showed the foreign mRNA present and active up to 2 months after injection. The objective of any traditional vaccine, and of the COVID-19 injections as well, is to increase

Analyzing the BNT162b2 5' Cap Containing an Imperfect Kozak Sequence

| | | | | | | | | | | | | | | | | | | | | | | | |
|----------|---|-----|-----------------------|-----|-----|-----|-----|-----|-----|-----|-----|-----|--|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| (1) DNA: | ATG TTC GTG TTC CTG | | | | | | | | | | | | | | | | | | | | | | |
| (2) RNA | +4 | | | | | | | | | | | | | | | | | | | | | | |
| (3) | GAG AAU AAA CUA GUA UUC UUC UGG UCC CCA CAG ACU CAG AGA GAA CCC GCC ACC AUG UUC GUG UUC CUG | | | | | | | | | | | | | | | | | | | | | | |
| (4) AA: | Glu | asn | lys | leu | val | phe | phe | trp | ser | pro | gln | thr | gln | arg | glu | pro | ala | thr | met | phe | val | phe | leu |
| (5) | The Kozak consensus sequence for initiation of translation in vertebrates is (GCC)GCCRCCATGG, where R is a <u>purine</u> (A or G) that is always observed (Kozak, 2002) | | | | | | | | | | | | | | | | | | | | | | |
| (6) | The sequence was defined as | | 5'-(gcc)gccRccAUGG-3' | | | | | | | | | | Kozak 2002 | | | | | | | | | | |
| (7) | | | 5'-(gcc)gccAccAUGU-3' | | | | | | | | | | BNT162b2 * | | | | | | | | | | |
| (8) | | | 5'-(xxx)xxxAxxAUGU-3' | | | | | | | | | | coronavirus 2 Spike Protein (covid-19)** | | | | | | | | | | |

* World Health Organization. Messenger RNA Encoding the Full-Length SARS-CoV-2 Spike Glycoprotein. INN, 2020, 11889

** Severe acute respiratory syndrome coronavirus 2 isolate Wuhan-Hu-1, co - Nucleotide - NCBI
https://www.ncbi.nlm.nih.gov/nucleotide/NC_045512.2 (accessed Feb 8, 2021)

Figure 3. Row (1) shows the first of 14 start codons, the ATGs, in the DNA followed by five spike codons specifying amino acids in the targeted BNT162b2 (which is identical to mRNA-1273 according to Nance & Meier, 2021, p. 748); row (2) emphasizes the downstream location of the critical position +4 in the Kozak sequence; row (3) shows the opening, 5' cap in the “untranslated region” of the spike protein prior to the insertion of any m1Ψ substitutions for the 17 uridine (Us) that appear there; row (4) aligns the sequence of amino acids that would be produced by the ribosome if every codon were translated (of course, the actual translation is supposed to begin at AUG in Kozak positions +1, +2 and +3, whereas the preceding sequence of potential codons form part of the “cap” which remains untranslated and protects the mRNA from degradation; row (5) shows the Kozak sequence in vertebrates with an optional (GCC) followed by an obligatory GCCRCCATGG, where R can be either A or G; row (6) reiterates Kozak’s sequence from 2002 where lower case letters show optimal hypothetical elements and upper case those that are required; row (7) shows the portion of the mRNA cap that is supposed to contain a perfect replica of the Kozak sequence but comes up short in position +4 where instead of a G, we find a U that will be replaced by m1Ψ (presumably the “cloaking” component); row (8) confirms that the cap segment shown for BNT162b2 is homologous to the XNA skeleton in the counterpart region of SARS-CoV-2. The curious shortcoming of the unnatural injectables of BNT162b2 and mRNA-1273 occurs in the +4 position where they not only use a non-canonical U instead of the optimal G, but they replace the U with the wild card converting the whole mRNA into an mXNA with 10 unnatural m1Ψs in the cap and 728 in the intended spike protein coding sequence.

antibody levels against one or more specific disease agents. However, traditional vaccine components should not be present 60 days later. Something is causing the novel post-injection disease conditions, resurgence of cancers, formation of blood clots, cardiac issues, and the sudden death of formerly healthy persons. Xia (2021) wrote:

As mammalian host cells attack unmodified exogenous RNA, all U nucleotides were replaced by N1-methylpseudouridine ([m1]Ψ). However, [m1]Ψ wobbles more in base-pairing than U and can pair not only with A and G, but also, to a lesser extent, with C and U. This is likely to increase misreading of a codon by a near-cognate tRNA. When nucleotide U in stop codons was replaced by [m1]Ψ, the rate of misreading of a stop codon by a near-cognate tRNAs increased. Such readthrough events would not only decrease the number of immunogenic proteins, but also produce a longer protein of unknown fate with potentially deleterious effects.

Xia's findings demand attention. The first codon after the canonical start AUG, at +4, appears to be suboptimal relative to the desired Kozak sequence. As such it may allow for the ribosomal pre-initiation complex (PIC) to scan past the first AUG and start producing a protein/peptide product at one of the 13 other downstream AUG codons. Given that "...AUG G" with G in the +4 position is optimal (see Figure 3), but the BNT162b2 sequence is AUG UUC, or more accurately **Am1ΨG m1Ψm1Ψm1Ψ** which does not conform either to the SARS-CoV-2 spike protein which is AUG UUU (see Figure 1) nor does it conform to the canonical Kozak sequence, so what will the result be? According to Thomas et al. (1988) writing for the prestigious *Proceedings of the National Academy of Sciences of the United States of America*, UUC is more efficient than UUU. But the question for the COVID-19 mXNA is what efficiency does **m1Ψm1ΨC** have in comparison to a sequence with the shape, **m1Ψm1Ψm1Ψ**? How will the synthetic nucleotide affect the binding of the mXNA to the ribosome? Downstream from there, how will it impact protein folding?

The binding process in particular is complex and known to be dependent on interactions between many signaling components in addition to the codons themselves. Similarly, protein folding is critically important to functionality as McKernan et al. (2021) have explained:

Codon optimization describes the process used to increase protein production by use of alternative but synonymous codon changes. In SARS-CoV-2 mRNA vaccines codon optimizations can result in differential secondary conformations that inevitably affect a protein's function with significant consequences to the cell. Importantly, when codon optimization increases the GC content of synthetic mRNAs, there can be an inevitable enrichment of G-quartets which potentially form G-quadruplex structures. The emerging G-quadruplexes are favorable binding sites of RNA binding proteins like helicases that inevitably affect epigenetic reprogramming of the cell by altering transcription, translation and replication. In this study, we performed a RNAfold analysis to investigate alterations in secondary structures of mRNAs in SARS-CoV-2 vaccines due to codon optimization. We show a significant increase in the GC content of mRNAs in vaccines as compared to native SARS-CoV-2 RNA sequences encoding the spike protein. As the GC enrichment leads to more G-quadruplex structure formations, these may contribute to potential pathological processes initiated by SARS-CoV-2 molecular vaccination.

Since retroviruses have the capacity to incorporate mXNA in native DNA via reverse transcriptase (as shown *in vitro* by Aldén et al. 2022), it is relevant that Pachetti et al. (2020) identified a SARS-CoV-2 dependent RNA polymerase (RdRp) that seems to transform the single stranded mRNA into double-stranded DNA. Once transformed, the viral sequence can be integrated into the genome of infected cells. An example of a human retrovirus that does this is T-cell lymphotropic virus (HTLV-1), associated with certain T-cell leukemias and lymphomas (Zhang et al., 2017). There are also non-viral retro-elements known as "long interspersed nucleotide elements" (LINEs), which account for about 25% of the human genome (Deininger & Batzer, 2002; Michieletto et al., 2019; Müller et al.,

2018). In that regard, in the summer of 2021, Chandramouly et al. (2021; also see Science Daily, 2021) reported the following:

Cells contain machinery that duplicates DNA into a new set that goes into a newly formed cell. That same class of machines, called polymerases, also build RNA messages, which are like notes copied from the central DNA repository of recipes, so they can be read more efficiently into proteins. But polymerases were thought to only work in one direction DNA into DNA or RNA. This prevents RNA messages from being rewritten back into the master recipe book of genomic DNA. Now, Thomas Jefferson University researchers provide evidence that RNA segments can be written back into DNA via a polymerase called theta, which could have wide implications affecting many fields of biology.

I have already cited Aldén et al. (2022) who reported “that BNT162b2 mRNA is reverse transcribed intracellularly into DNA” in as few as 6 hours after “BNT162b2 exposure”. Boettler et al. (2022) provided more evidence of such effects after vaccination:

COVID19 vaccination can elicit a distinct T cell-dominant immune-mediated hepatitis⁶ with a unique pathomechanism associated with vaccination induced antigen-specific tissue resident immunity requiring systemic immunosuppression. Lay summary Liver inflammation is observed during SARS-CoV-2 infection but can also occur in some individuals after vaccination and shares some typical features with autoimmune liver disease. In this report, we show that highly activated T cells accumulate and are evenly distributed in the different areas of the liver in a patient with liver inflammation following SARS-CoV-2 vaccination. Moreover, within these liver infiltrating T cells, we observed an enrichment of T cells that are reactive to SARS-CoV-2, suggesting that these vaccine-induced cells can contribute to the liver inflammation in this context.

Singh et al. (2020) discussed the role of mitochondria in SARS-CoV-2 pathogenesis:

Based on available data for the SARS-CoV-1 virus, we suggest how CoV-2 localization of RNA transcripts in mitochondria hijacks the host cell's mitochondrial function to viral advantage. Besides viral RNA transcripts, RNA also localizes to mitochondria. SARS-CoV-2 may manipulate mitochondrial function indirectly, first by ACE2 regulation of mitochondrial function, and once it enters the host cell, open-reading frames (ORFs) such as ORF-9b can directly manipulate mitochondrial function to evade host cell immunity and facilitate virus replication and COVID-19 disease. Manipulations of host mitochondria by viral ORFs can release mitochondrial DNA (mtDNA) in the cytoplasm and activate mtDNA-induced inflammasome and suppress innate and adaptive immunity. We argue that a decline in ACE2 function in aged individuals, coupled with the age-associated decline in mitochondrial functions resulting in chronic metabolic disorders like diabetes or cancer, may make the host more vulnerable to infection and health complications to mortality. These observations suggest that distinct localization of viral RNA and proteins in mitochondria must play essential roles in SARS-CoV-2 pathogenesis.

BNT162b2 is a viral transcript and as such may follow the same path as SARS-CoV-2 by interfering with mitochondrial function in more or less the same manner, relying on a convenient ORF, just as Singh et al. described for the original virus. As already alluded to, Aldén et al. (2022)

... detected high levels of BNT162b2 in Huh7 cells and changes in gene expression of long interspersed nuclear element-1 (LINE-1), which is an endogenous reverse transcriptase. Immunohistochemistry using antibody binding to LINE-1 open reading frame-1 RNA-binding protein (ORFp1) on Huh7 cells treated with BNT162b2 indicated increased nucleus distribution of LINE-1.

This finding suggests that mXNA can reverse transcribe itself into LINE-1 sequences in nuclear DNA. Zhang et al. (2021) actually anticipated in large measure, the later findings of Aldén et al., but with the original SARS-CoV-2 disease:

⁶ See footnote 3 just above here where researchers noted that the SARS-CoV-2 virus incorporated hepatitis C virus (HCV) along with human immunodeficiency virus-1 (HIV-1). For what other purpose could bioweapons designers have spliced these components together if not to cause reverse transcription of disease producing sequences into human DNA? Regardless what the intentions were, the clinical consequences suggest precisely those sorts of outcomes.

An unresolved issue of SARS-CoV-2 disease is that patients often remain positive for viral RNA as detected by PCR many weeks after the initial infection in the absence of evidence for viral replication. We show here that SARS-CoV-2 RNA can be reverse-transcribed and integrated into the genome of the infected cell and be expressed as chimeric transcripts fusing viral with cellular sequences. Importantly, such chimeric transcripts are detected in patient-derived tissues. Our data suggest that, in some patient tissues, the majority of all viral transcripts are derived from integrated sequences. Our data provide an insight into the consequence of SARS-CoV-2 infections that may help to explain why patients can continue to produce viral RNA after recovery.⁷

The findings of Xia (2021), Röltgen et al. (2022), Liu et al. (2021), McKernan et al. (2021), Zhang et al. (2021), Pachetti et al. (2020), Chandramouly et al. (2021), Aldén et al. (2022), and Boettler et al. (2022) call for further analysis. Particularly, since Reed et al. found in autoimmune disease in transgenic mice. What if some portion of the mXNA of the spike protein is integrated into human host genomes? It is known in advance that variations in positions -3, +2, and +4 in the Kozak sequence (see the Appendix, Part B for some of the details) can cause errors and the mXNA readings by the ribosome can only be complicated by **m1Ψ** nucleotide substitutions for uridine. It is yet to be determined if and how the ORFs in the mXNA of BNT162b2 and mRNA-1273 will be expressed in actual proteins and peptides and what impact they will have on human recipients of tens of billions of often imperfect replicas of mXNA (see Figure 2 and discussion there). Vojdani et al. (2021; also see Vojdani & Kharrazian, 2020, as well as Segal et al., 2018) point to some of the dangers of near homologs of essential human proteins and peptides at the risk of triggering cross-reactive autoimmune diseases in at least some recipients.

Part C. The Amino Acid Sequence of the SARS-CoV-2 Spike Protein

In this section, I present the amino acid sequence predicted by the genetic engineers promoting the SARS-CoV-2 mXNA injections containing the modified **m1Ψ** variant. according to *in vitro* studies (not inside human bodies) where the enzyme discovered by Chamberlin et al. (1970, 1983) reads the mRNA perfectly and produces exactly the hoped-for amino acid sequence with no errors and no fragmented pieces, the following illustration represents the linear sequence predicted for the spike protein contained in the SARS-CoV-2 mXNA injections. The single letters used are from the standard list of amino acids that can be found [here](#) under the heading, “Table of standard amino acid abbreviations and properties”. I have highlighted in bolded red two significant motifs that I want to discuss here. TQLPP and FTVEKG are the two motifs I have singled out. However, MPAS and MPRH are peptides that are also found in the protein amino acid sequence from the published mXNA. The latter two peptides, may also interfere with normal biosignaling. The underlying issue, as in autoimmune disorders generally, is the homology of such peptide sequences, with common protein “motifs” — the relatively short sequences of amino acids that are known to be involved in essential disorders and diseases. I will just address the two highlighted motifs in the order they appear reading from left to right. They are known as “motifs” precisely because plausible inferences can be made about the biological functions in which they may be involved in meaningful ways.

⁷ In this regard, it is interesting as Fleming argued (2021), based on the earlier analysis of the SARS-CoV-2 viral sequence by Huang et al. (2006) well before the outbreak ultimately traced to the Wuhan weapons laboratory in China, that the bioweapon released either accidentally, or intentionally, in 2019 contained four perfect sequences taken respectively from what they termed HCV (human hepatitis C virus), HIV-1 (human immunodeficiency virus -1), SARS-CoV-1, and SARS-CoV-2. Checking those sequences against the NCBI databases confirmed that they are exactly as portrayed by Huang et al. in 2006 and as re-iterated in the hardly readable fine-print one-page Appendix of Fleming (2021). The key question is, how did perfect peptides from HCV and HIV-1 end up in the SARS-CoV-2 virus? The research trail of funding, publications, and patents shows they were put there deliberately by bioweapons designers engaged in “gain of function” research (Oller, 2021).

| The Intended SARS-CoV-2 Spike Protein Amino Acid Sequence† in BNT162b2 and mRNA-1273 | Standard Abbreviation | Amino Acid |
|---|--------------------------|---------------|
| MFVFLVLLPLVSSQCVNLTTR TQLPP AYTNSFTRGVYYPDKVFRSSVLHSTQ | A | Alanine |
| DLFLPFFSNVTWFHAIHVSGTNGTKRFDNPVLPFNDGVYFASTEKSNIRG | C | Cysteine |
| WIFGTILDSKTQSLIVNNATNVVIVKCEFQFCNDPFLGVYYHKNNKSWM | D | Aspartate |
| ESEFRVYSSANNCTFEYVSQPFLMDLEGKQGNFKNLREFVFNIDGYFKIY | E | Glutamate |
| SKHTPINLVRLDPQGFSALEPLVDLPIGINITRFQTLALHRSYLTTPGDSSSG | F | Phenylalanine |
| WTAGAAAYYVGYLQPRITFLKYNENGTTTDAVDCALDPLSETKCTLKS FT | G | Glycine |
| VEKGI YQTSNFRGSPNPSCGSPISPICAPSARCSMPDPSLCTPGTGSLSAIA | H | Histidine |
| WPITPCCTTPPASAPSSATACPLPSTTCASQTCTPTASSGEMKCGRLPLDRQA | I | Isoleucine |
| RSPTTTTSCPTTSPAVLPGTATTWTPKSAATTTTCTGCSGSPISPSGTSPPRSIRP | K | Lysine |
| AAPLVTAWKASTATSHCSPTAFSPQMAWAISPTIEWWCASNCCMPLPQCAAL | L | Leucine |
| RKAPISRTNATSTSTAPAPACQATRSSCHSSSLAGISPIPQTIPLEIPRHWSWTS | M | Methionine |
| PLAASAECLSPAPTPAIRWQCCTRTTVPKCPWPFTPIHSHLHGCTPPAAMCF | N | Asparagine |
| RPEPAVSEPTTIATSATSPSALESAPATRHRQTALGEPEAWPARASPTQCLWA | P | Proline |
| PRTAWPIPTTSLSPPTSPAPQRSCLCPPRPAWTAPCTSAIIPPSAPTCCCSTAA | Q | Glutamine |
| SAPSEPPQGSPWNRTTRTPKRCSPKSRSTRPLLSRTSAASISARFCPLASPASGASS | R | Arginine |
| RTCCSTKHWPTPASSSSMAIVWATLPPGIFAPRSLTDQCCLCPMRSPSTHLP | S | Serine |
| WPAQSQAAAGHLEQAPLCRSPLLCRWPTGSTASEPRMCCSTRTRSSPTSSAPSA | T | Threonine |
| RSRTAAAQAPWESCRTWSTRMPRHPTWSSCPPTSAPSALCTISADWTLLRP | V | Valine |
| RCRSTDSQADCRASRHTPSSSEPPRLPLPWPPRCLSVWARAREWTFARA | W | Tryptophan |
| TTASLSLPLTAWCFCTHMCPLKRRISPLQPSATTAKPTFLEKACSCPTAPIGS | Y | Tyrosine |
| HSGTSTSPRSSPPTTPSCLATATSSALTIPCTTPSCLATATSSALTIPCTTQAPIW | | |
| TWAIASMPASTSRKRSTGTRWPRITRASTCKNWGSTSTSSGPGTSGWALSP | | |
| DLPSWSQSCCVAPAAVAARAVVAVAAAAASTRTILSPCRANCTTH | | |

† Owing to a typographical mistake copying the page number in a cut and paste operation to generate this sequence in Expaty at <https://web.expasy.org/translate/>, all of the proteins after the FTVEKG motif everything is changed in a correction on October 17, 2022.

TQLPP

TQLPP, as localized in the spike protein of BNT162b2, shares antibody binding for the same motif associated with thrombopoietin. According to Nunez-Castilla et al. (2022a, 2022b; also see Torabi et al., 2022), thrombocytopenia accounts for almost a 5-fold rise in COVID-19 patients as compared against deaths from that disease condition in the pre-COVID general population. In 2020, Figliozi et al. found that mortality with thrombocytopenia in patients with COVID was increased by a factor of 6.23 times above its likelihood in those who did not have COVID. What are the odds in patients who have received one of the mRNA shots compared against those who have not? Figliozi et al. suggest that the shots are implicated in causing thrombocytopenia and the TQLPP motif may well be playing a role. Why, in any event, was that motif included in the BNT162b2 and the mRNA-1273 coding sequence in the first place?

FTVEKG

Another suspect in the coding sequence is the motif consisting of FTVEKG which is associated with pollen allergies. What is it doing in the mRNA injectables? It is known to code for Phleum pratense (Phl p2) protein — a respiratory pollen allergen present in Timothy grass. That particular allergen affects an estimated 200 million people worldwide (Kurt et al., 2012). Pollen exposure is

known to influence the production of cytokines, and they are commonly associated with what Blaylock (2021; and elsewhere) termed a “cytokine storm”. It is the sort of allergic reaction that can precipitate in anaphylaxis. Damialis et al. (2021) wrote:

Coexposure to airborne pollen enhances susceptibility to respiratory viral infections, regardless of the allergy status. We hypothesized this could be also true for SARS-CoV-2 infections. To investigate this, we tested for relationships between SARS-CoV-2 infection rates and pollen concentrations, along with humidity, temperature, population density, and lockdown effects. Our unique dataset derives from 130 sites in 31 countries and across five continents. We found that pollen, sometimes in synergy with humidity and temperature, explained, on average, 44% of the infection rate variability. Lockdown halved infection rates under similar pollen concentrations.

Since the mRNA injectables are designed to bypass the body’s defenses, the FTVEKG motif may be a significant part of the explanation for COVID injections leading to anaphylaxis and all kinds of unexplained sudden deaths after one or more injections. The question remains: what is that suspect motif doing in the COVID genetic payloads? Could these elements, and who knows how many other potentially lethal components appear, have been purposely included in the spike coding sequence? What else is in the injectables? Does anyone still want to take another spin of the cylinder and pull the trigger again?

Conflict of Interest

The author has no conflicts of interest to report.

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