Coronavirus – Using Your DNA Against You

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Abstract

This paper discusses how coronaviruses may attack cells and why the most dangerous, MERS, SARS, and SARS-CoV-2 (Covid-19), are so infectious. Independent research has found multiple nucleotide sequence matches between all the coronaviruses and the human genome. Those sequences are the same as some of the DHU loops and T-loops of human tRNA. Using those loops and their anticodon matches, viruses may be able to fool the nuclear membrane in cells to allow at least part of the virus to enter and associate with the human DNA, creating more opportunities for further infection. MERS and SARS only have T-loop double matches; SARS-CoV-2 has double matches in both the DHU and T-loops. Our immune system may be compromised and may no longer be able to stop the virus and other diseases from attacking organs throughout the body. Vaccines that attack the virus protein shell spikes while ignoring the virus inside are doomed to failure from the Darwin effect, but recognizing these loop matches suggests a possible approach to successful coronavirus vaccines. For SARS, eliminating the nucleotide sequence GTTCATGTC may create a mild virus and stimulate our immune system to recognize and attack the full virus; for MERS, eliminating the nucleotide sequence GTTTGATTC may do the same thing. All variants of SARS-CoV-2 suggest that GTTCATGTC and TAGTGGTGAG be eliminated, but the omicron variants also include a match for GATCAAAAC. This extra double match in the omicron variants suggests why they are more infectious. Note that SARS and SARS-CoV-2 have the same matched nucleotide sequence. I speculate that these matches indicate an evolutionary path from SARS to SARS-CoV-2 to omicron, all to improve its infection ability. The other SARS-CoV-2 variants are unsuccessful attempts. In my opinion, SARS, SARS-CoV-2, and omicron are the same disease that has continued to evolve and should be under a single general label. Only the infection process is considered in this paper, not the innate virulence of the viruses.

Keywords: coronavirus, tRNA, DHU loop, T-loop, anticodon, SARS, MERS, SARS-CoV-2, Covid-19, omicron

Introduction

Coronaviruses are a large family of viruses with eight of them known to infect humans, and have been given that general name because their outside appearance is similar to a corona. The human infections are named 229E, NL63, OC43, HKU1, SARS, MERS, and SARS-CoV-2 and are shown in Figure 1.

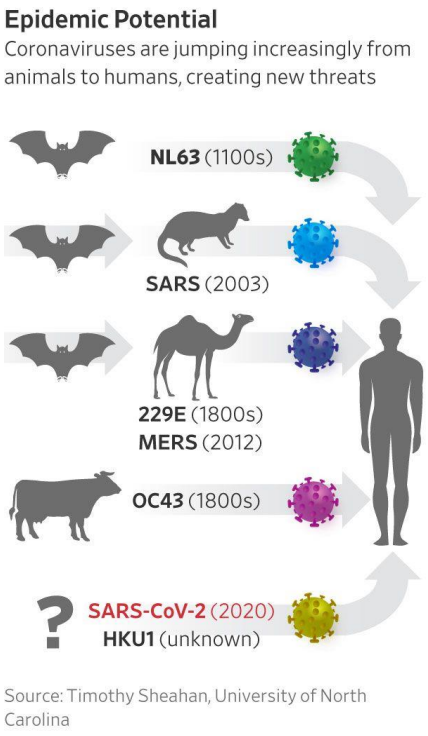

Figure 1. Human Coronaviruses

A newly jumped-to-humans coronavirus labeled CCoV-HuPn-2018 is described below.

People infected with coronaviruses are known to experience respiratory problems, from non-existent to severe. In addition, some of the people infected with SARS-CoV-2 and who recovered found themselves with health problems they never had before, including diabetes, high cholesterol, and damage to their heart, immune system, brain, and elsewhere.

You may be infected with 229E, NL63, OC43, or HKU1 many times in your life. All are associated with the common cold, with symptoms including a runny or stuffy nose, sneezing, mild cough, watery eyes, sore throat, headache, some aches

and pains, and perhaps fatigue. In 2021 the coronavirus CCoV-HuPn-2018 was found by Vlasova to have migrated from a canine intestinal disease to a human respiratory disease, with effects similar to the common cold varieties. Other viruses also cause these symptoms, including rhinoviruses and adenoviruses. All of the coronaviruses tend to occur in very young children and older people with preexisting conditions.

Severe Acute Respiratory Syndrome (SARS) was first identified from an outbreak in China in 2003 with severe flu-like symptoms causing hospitalization and death, spreading to 26 countries totaling more than 8,000 cases. SARS infections require close person-to-person contact and have basically been limited to hospital settings. Only four cases have been reported since 2003, all from laboratory accidents.

Middle East Respiratory Syndrome (MERS) was first reported in Saudi Arabia in 2012 and has since spread to other countries, including the United States, by travelers from the Arabian Peninsula who have been in contact with sick camels or diseased camel food products. In this case the fever, cough, shortness of breath, and other symptoms are more severe, affecting people of all ages, with 30%-40% dying from it. Although coronaviruses are respiratory diseases that are usually transmitted through the air, human infection of MERS is typically by close contact with camels or an infected person, making the overall risk to the global population rather low. As of June 2021 about 2,600 cases have been reported.

SARS-CoV-2 (Covid-19) was first noticed in January 2020 in Wuhan, China and quickly spread around the world by infected people who traveled. Symptoms typically develop approximately 5 days after infection, but can appear from 2 to 14 days afterwards and people testing positive for the disease may appear without symptoms up to being severely ill. As of December 2022 there were over 650 million cases and 6.7 million deaths worldwide, with the United States having the most infections by far, with over 102 million cases and over 1.1 million deaths. While much is unknown about this disease, people may remain contagious for at least 10 days and should self-quarantine during that period. SARS-CoV-2 seems to spread most easily through airborne droplets (coughing, sneezing, or even talking) and may remain active in the air for longer periods of time. As with previous pandemics, the best advice is to wear a mask in public, avoid crowds, avoid indoor activities, and wash your hands frequently.

Viruses are small bundles of genetic material that don't have cells but are protected by coats of protein. They're usually not considered living things because they need to infect host cells to generate their metabolism and to reproduce, but they do have genes and evolve by natural selection. Their status as living or nonliving became more controversial with the discovery of pandoraviruses in 2013 that are bigger than many bacteria and, according to Pennisi and also by Nadège, their position in the tree of life is unclear. Viruses can be either DNA or RNA based and they infect all forms of life. As Figure 2 shows, they come in a variety of shapes.

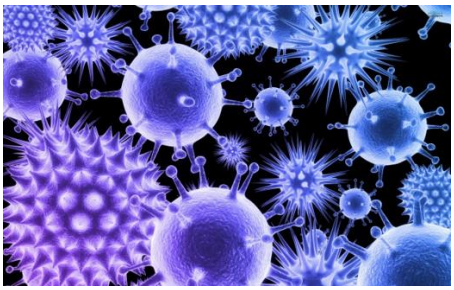

Figure 2. Virus shapes

Figure 3 shows the classic way that viruses work.

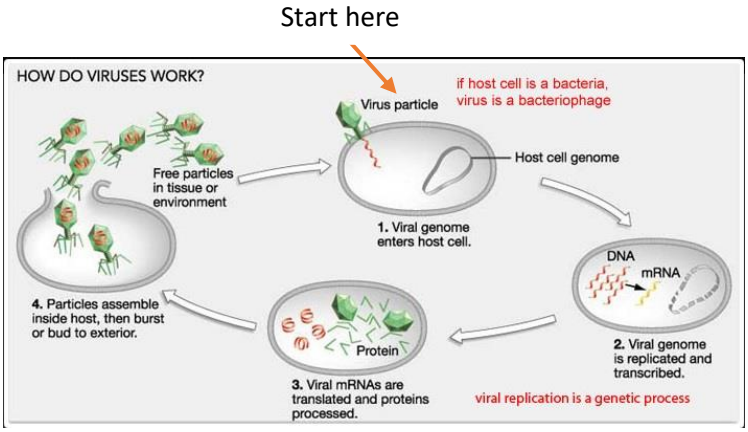

Figure 3. How Viruses Work
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Starting at the top, the virus lands on a cell, pierces the cell wall, and injects the virus material into the cell, part of which is messenger RNA (mRNA) which is used to make more copies of the virus that burst through the cell wall to infect

other cells, destroying the cell in the process. This illustration shows a DNA-based virus, but one based on RNA would do the same thing.

Figure 4 shows the specific process used for SARS-CoV-2 infections but may be extended to all coronaviruses.

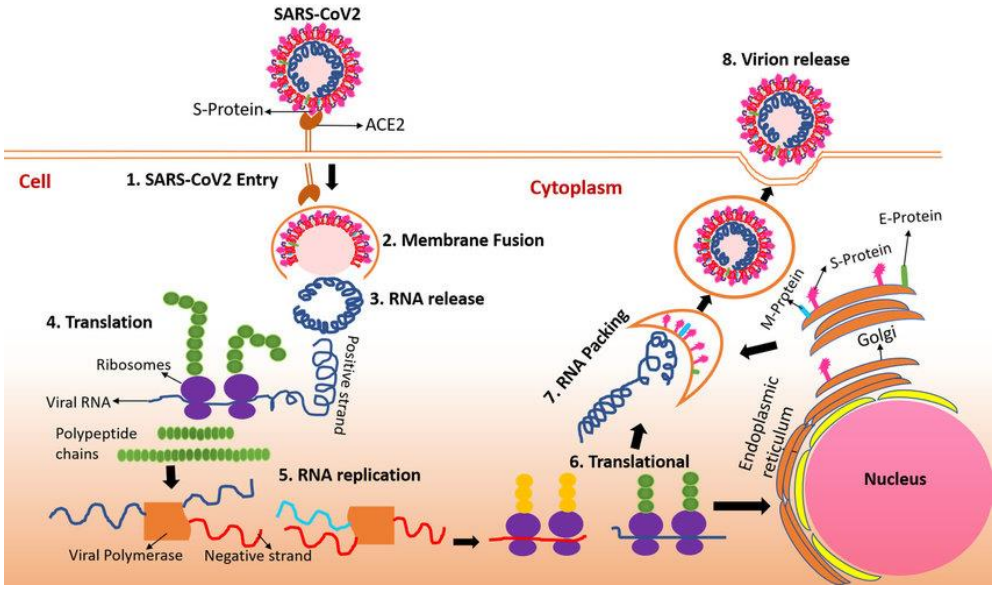

Figure 4. SARS-CoV-2 Infection Process
From Boopathi, Poma, and Kolandaivel

As you can see, after the virus enters the cell cytoplasm it unwinds and is fully copied, after which it can try to enter the cell nucleus or be coated in a protein envelope and released to infect other cells via budding rather than by destroying the cell. This way of exiting permits the virus to use this cell to make more copies for continued infections. Weiss & Gottlinger describe how a similar method is used by the human immunodeficiency virus (HIV).

Our immune system has ways to stop viruses from injecting their contents into cells and infected cells have ways to alert the immune system that they have been infected. The viruses, on the other hand, have their ways to suppress our immune system functions if they can enter the cell nucleus. We can feel all this happening when we get a cold, as our symptoms intensify and then weaken as our immune system gets the upper hand.

Cells normally make the material they need with their own mRNA. The virus, however, brings its mRNA and the cell innocently uses its normal mechanism to make many duplicates of the virus.

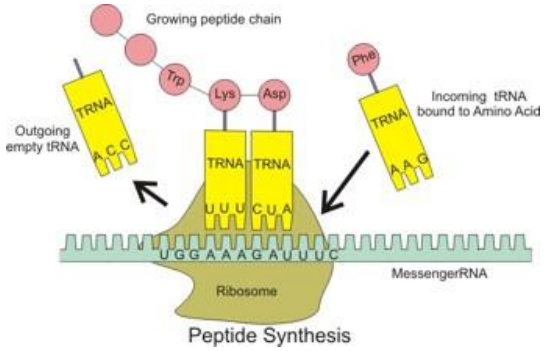

Figure 5. Process for Making Cell Material
Drawn by Boumphreyfr

As Figure 5 shows, transfer RNA (tRNA) components carry amino acids to a ribosome where the amino acids are linked to peptide chains to form the proteins used throughout the body. Ribosomes are large and complex molecular machines and are found in all living cells. Protein creation happens when a nucleotide code within the tRNA matches the complementary nucleotide code of the mRNA being used by the ribosome. Then the ribosome binds the amino acid to the chain, releases the empty tRNA, and continues moving across the mRNA to bind other amino acids from other tRNAs. This same process is used by the cells all the time and also with virus mRNA for the cell to innocently make duplicate viruses.

Methods

I wondered if there was any clear link between the coronaviruses and humans. Since all the coronaviruses are RNA-based and humans are DNA-based the link couldn't be direct, but I started looking at the nucleotides to see if anything popped out. The coronaviruses each have about 30,000 nucleotides while the human genome has over three billion. I decided that a one-in-a-million chance of a nucleotide string appearing in both the coronaviruses and the human DNA would be good enough. All cells use combinations of four nucleotides (adenine, guanine, cytosine, and thymine) in their construction. These nucleotides are indicated as A, G, C, and T. For four nucleotides, that means a string of 10 would

occur one chance in a million at random (4^{10}), so I looked for a string at least that long.

The National Center for Biotechnology Information (NCBI), a part of the National Institutes of Health (NIH), provides a free online database of information about many living things, including full nucleotide sequences for both the human genome and the coronaviruses known to infect humans. That was the data I needed for this search, and I started with SARS-CoV-2.

To my surprise, I found three strings – TAGTGGCTAG, TAGTGGTGAG, and TAGTGGTTAT – which matched some of the DHU loops of human tRNA. As you saw above, tRNA is an important cellular component used for making proteins. Here's what tRNA looks like as a two-dimensional cloverleaf structure as described by Robert W. Holley (Figure 6).

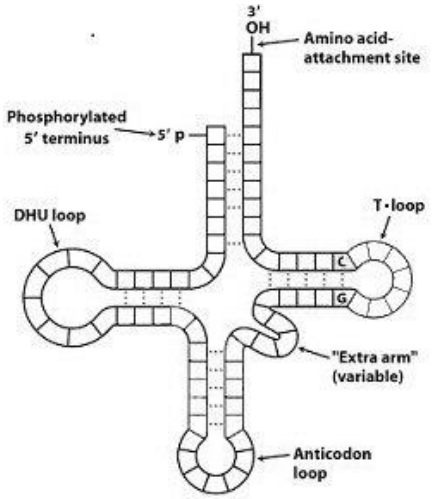

Figure 6. tRNA Structure

Adapted from: *BIOCHEMISTRY 7E* by J.M. Berg, J. Tymoczko, and L. Stryer, Copyright 2012 by W.H. Freeman and Company. Used with permission by the publisher.

Starting at the 5' end there are seven nucleotides forming half of the main stem, followed by a two-nucleotide bridge to the D-arm. Four nucleotides form half the D-arm stem, followed by the DHU loop containing seven to ten nucleotides, then the other four nucleotides of the stem. A one-nucleotide separator is followed by five nucleotides forming half of the Anticodon arm stem, then seven nucleotides for the Anticodon loop (the middle three nucleotides are the amino acid code), followed by five nucleotides for the other half of the stem. This is

followed by a variable number of nucleotides forming an "extra arm". Five nucleotides form half of the T-arm stem, followed by seven nucleotides for the T-loop, then five nucleotides for the other half of the T-arm stem. Finally, seven nucleotides forming the other half of the main stem are followed by up to three nucleotides that act as the attachment point for the amino acid being transported at the 3' end.

For a specific example, here's a complete human tRNA with the DHU loop TAGTGGTGAG, one of the nucleotide sequences found in SARS-CoV-2:

TCCTCGTTAGTA**TAGTGGTGAG**TATCCCCGCCTGTCACGCGGGAGA
CCGGGGTTCGATTCCCCGACGGGGAG

And here are all its tRNA parts:

TCCTCGT	5' stem
TA	bridge to the D-arm
GTA**T**	D-arm stem
AGTGGTGA	DHU loop
GTAT	D-arm stem
C	separator
CCCGC	Anticodon arm stem
CTGTCAC	Anticodon loop (codes for aspartic acid)
GCGGG	Anticodon arm stem
AGAC	"extra arm"
CGGGG	T-arm stem
TTCGATT	T-loop
CCCCG	T-arm stem
ACGGGGA	3' stem
G	amino acid attachment point

Notice that for SARS-CoV-2 the DHU loop includes the top nucleotides of the stems, resulting in TAGTGGTGAG. Including the top stem nucleotides is accepted practice for the T-loop, as shown in Figure 6, and is also followed for the tRNA loops in my work.

Looking carefully at the stems, you'll see that very specific nucleotides are used. For example, the two parts of the Anticodon arm stem are CCCGC and GCGGG. The first nucleotides on both sides nearest to the Anticodon loop are C and G. This pair forms a very strong bond (as does A and T) and these strong bonds are

what tRNA tries to have for all its stems. In addition, all three loops in a tRNA are associated, with the DHU and T-loops corresponding to the amino acid code in the Anticodon loop. The result is a tightly built component the cell uses to transport amino acids from one place to another.

Figure 6 shows the tRNA structure in two dimensions, but the actual component in our cells is three-dimensional and does a lot of twisting and turning, as illustrated in Figure 7 with the colors expressed in the companion drawing.

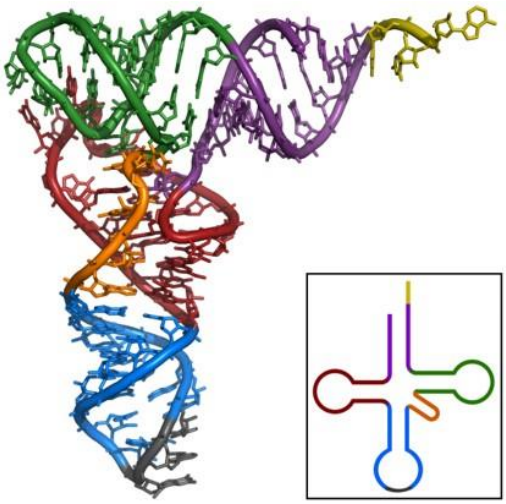

Figure 7. Three-Dimensional tRNA Structure
Study.com

While SARS-CoV-2 has some of the identical T-loops and DHU loops found in human tRNA, the virus doesn't have the associated stems. The virus only mimics the loop parts of human tRNA. The other coronaviruses also have extended matching nucleotide strings for T-loops and DHU loops of tRNA. Further inspection of the Homo sapiens tRNAs from a file provided by the University of California Santa Cruz (see References) revealed that some DHU loops were shorter or longer than 10 nucleotides, so all 74 of the known and unique human tRNA DHU loops were checked against all of the human coronaviruses. All 45 of the known and unique human tRNA T-loops were also checked against all of the human coronaviruses. Both the T-loops and DHU loops appear to relate to specific amino acids, although some relate to multiple ones. Only the anticodon codes known in human tRNAs were considered in the coronaviruses. A full list of the 20 amino acids and their associated human tRNA loops is shown in the appendix as Table A-1.

Results

Table 1 shows the discovered coronavirus-to-human DHU loop matches for each of the human coronaviruses, with their associated amino acids and any found human anticodons. Anticodons always begin 12 nucleotides from the end of the DHU loop, and that's where they must be in a coronavirus to match the DHU loop. An anticodon, as illustrated in Figure 6, contains the complementary nucleotides to the amino acid codon in the mRNA string and, since the direction is from the 5' end (see Figure 6), they're also reversed. So, an anticodon of AAC would be the amino acid GUU (valine). The Accession Code is used with the NCBI database to locate the specific virus information.

Table 1

Human tRNA DHU Loops in Coronaviruses with Associated Amino Acids and Found Anticodons

Coronaviruses (Accession Code)	Human tRNA DHU Loop	Associated Amino Acids	Found Anti-codon
229E (NC_002645.1)	TGATGGATAA	Arginine (Arg)	None
	CACAGGTAG	Cysteine (Cys)	None
	CAGTGGTAG	Alanine (Ala) Cysteine (Cys) Glycine (Gly) Valine (Val)	None
	AAGTGGTAA	Threonine (Thr)	TGT
NL63 (NC_005831)	TAGTGGTAAG	Glycine (Gly)	None
	AAGTGGTAA	Threonine (Thr)	None
	TAGTGGTAT	Glycine (Gly) Proline (Pro)	None

	CAATGGTAG	Glycine (Gly) Tryptophan (Trp)	None
OC43 (NC_006213.1)	CAATGGATAA	Arginine (Arg)	None
	CAGTAGGTAG	Leucine (Leu) Methionine (Met)	None
	GAGTGGTTAT	Valine (Val)	None
	TAGGGGTAG	Cysteine (Cys)	None
	TAATGGTAAG	Glutamine (Gln)	None
	CAGGGGTAG	Cysteine (Cys)	None
HKU1 (NC_006577.2)	TAATGGATAA	Arginine (Arg)	None
	TAATGGTAAG	Glutamine (Gln)	None
	TAGTGGTAT	Glycine (Gly) Proline (Pro)	None
CCoV-HuPn-2018 (MW591993.2)	TAATGGTGAG	Glutamine (Gln)	None
	TAATGGTAAG	Glutamine (Gln)	CTG
	CAATGGTAG	Glycine (Gly)	None
	TAGTGGTTAT	Valine (Val)	None
	AAGTGGTAA	Threonine (Thr)	None
SARS (NC_004718)	CAAGTGGTAG	Alanine (Ala)	None
	TAATGGTCAG	Glutamine (Gln)	None

	CAGTGGTAG	Alanine (Ala) Cysteine (Cys) Glycine (Gly) Valine (Val)	None
MERS (NC_019843.3)	CAGTTGGTTAG	Isoleucine (Ile)	None
	CAACGGTAG	Tryptophan (Trp)	None
	CACAGGTAG	Cysteine (Cys)	None
	CAGTGGTAG	Alanine (Ala) Cysteine (Cys) Glycine (Gly) Valine (Val)	None
	CAGGGGTAG	Cysteine (Cys)	None
	CAGGTGGTAG	Cysteine (Cys)	None
SARS-CoV-2 [Covid-19] Alpha (MZ930641.1) Beta (MZ944846) Gamma (MZ930670.1) Delta (MZ950690.1) Mu (MZ952579.1) Omicron [B.1.1.529] (OL672836.1)	TAGTGGCTAG	Glutamic acid (Glu)	None
	TAGTGGTGAG	Aspartic acid (Asp) Glycine (Gly)	Gly CCT
	CAACGGCAG	Tryptophan (Trp)	None
	TAGTGGTTAT	Valine (Val)	None

Table 2 shows the discovered coronavirus-to-human T-loop matches for each of the human coronaviruses with their associated amino acids and any found human anticodons. The position of the anticodons is variable, depending on the "extra arm" (Figure 6) in the tRNA from which they're copied, and that's where the anticodon must be in a coronavirus to match the T-loop. The length of the "extra arms" was found from the tRNAs listed in the University of California data file. The anticodons are complimentary so an anticodon of CAA would be the amino acid GUU (valine).

Table 2

Human tRNA T-Loops in Coronaviruses with Associated Amino Acids and Found Anticodons

Coronaviruses (Accession Code)	Human tRNA T-Loop	Associated Amino Acids	Found Antico-don
229E (NC_002645.1)	GTTTGAGTT	Arginine (Arg)	None
	GTTTGAACC	Asparagine (Asn)	None
	GTTTGATTC	Glycine (Gly) Tyrosine (Tyr)	None
	GTTTGAAAC	Valine (Val)	None
NL63 (NC_005831)	GTTCAAGCC	Isoleucine (Ile)	None
	GTTCAACCC	Phenylalanine (Phe)	None
	GTTTGATTC	Glycine (Gly) Tyrosine (Tyr)	None
OC43 (NC_006213.1)	GTTTGAGTT	Arginine (Arg)	None
	GTTCGAGAC	Asparagine (Asn)	None

	GATCAAAAC	Valine (Val)	None
	GTTTGAAAC	Valine (Val)	None
	GTTTGAATC	Leucine (Leu)	None
	GTTCAACCC	Phenylalanine (Phe)	None
HKU1 (NC_006577.2)	None	None	None
CCoV-HuPn-2018 (MW591993.2)	GATCAATGC	Alanine (Ala)	None
	GTTTGAGTT	Arginine (Arg)	None
	GTTCGAGAC	Asparagine (Asn)	None
	GTTCAATTC	Aspartic acid (Asp) Glycine (Gly) Tyrosine (Tyr)	None
	GTTTGAATC	Leucine (Leu)	None
SARS (NC_004718)	GATCAATGC	Alanine (Ala)	None
	GATCGATGC	Alanine (Ala)	None
	GTTTGAACC	Asparagine (Asn)	None
	CGTGCCCCA	Lysine (Lys)	None
	GTTCATGTC	Lysine (Lys)	TTT
MERS (NC_019843.3)	GATCAATGC	Alanine (Ala)	None
	GTTTGAGTT	Arginine (Arg)	TCC
	GTTTGAACC	Asparagine (Asn)	None

	GTTTGATTC	Glycine (Gly) Tyrosine (Tyr)	None
	GTTCATGTC	Lysine (Lys)	None
SARS-CoV-2 [Covid-19] Alpha (MZ930641.1) Beta (MZ944846) Gamma (MZ930670.1) Delta (MZ950690.1) Mu (MZ952579.1) Omicron [B.1.1.529] (OL672836.1)	GTTTGAGTT	Arginine (Arg)	None
	GATCAATGC Omicron variant BQ.1.1 only	Alanine (Ala)	None
	GTTTGAGCC	Lysine (Lys)	None
	GTTCATGTC	Lysine (Lys)	TTT
	GATCAAAAC	Valine (Val)	CAA Omicron variant only
	GTTTGAAAC	Valine (Val)	None

Discussion

A coronavirus infects a cell and its mRNA is used to make copies. Our cells normally put little snippets of their cytoplasm on the outside of the cell wall for immune system inspection. If the cell is infected, it's likely a bit of that cytoplasm will include virus proteins that will be recognized by our immune system. Then the immune system uses various methods to attack and destroy the cell, bringing the virus into the open for disposal.

The coronaviruses can't stop the cell from displaying those bits of its cytoplasm, so I suggest the virus evolved with copies of the tRNA loops in an attempt to enter the cell nucleus and associate part of the virus RNA with our DNA. Chatterjee et al. and Takano found that tRNA does, in fact, go in and out of both the cytoplasm and the nucleus. Research by Zaitseva et al. used artificial means to create amino acid deficiencies in the nucleus that allowed HIV to "hijack" (their term) a tRNA for the virus to enter the cell nucleus. For example, if a nucleus is looking for the amino acid glycine, a virus that pretends to be a tRNA carrying glycine might be able to sneak into the nucleus. The nuclear membrane is an important line of defense for our DNA, so it carefully inspects components that try to enter. At the same time, viruses try to mutate to find a way past this security. Small viruses such as polio and hepatitis may find hijacking to be the only way to enter the nucleus. On the other end of the scale, Legendre et al. show that the huge pandoraviruses contain their own full tRNA structures and wouldn't have to hijack anything. Could coronaviruses use special tRNA methods to get inside the nucleus?

As you see in Figure 6, the Anticodon loop contains the nucleotide code for the amino acid. But the anticodon is only three nucleotides long. For four nucleotides (A, T, C, G), that means a string of three would occur at random 4^3 times, or one time in 64. But the tRNA DHU loop also associates with specific amino acids and can be ten nucleotides long, making that possibility of error 4^{10} or about one in a million. I speculate the nuclear membrane looks at both the DHU loop and its matching anticodon, which starts 12 nucleotides from the end of the DHU loop, to better ensure that it's a tRNA for an amino acid it wants.

Table 2 shows another possible way into the cell nucleus via a T-loop match with its anticodon. Extended T-loops are nine nucleotides long, resulting in one chance at random of 4^9 or about 262,000. I speculate the nuclear membrane looks at both the T-loop and its anticodon to better ensure that it's a tRNA for an amino acid it wants.

Using the data in Tables 1 and 2; information from Figures 1, 2, 4, and 6; and our observations of infections from each of the coronaviruses may help us understand their evolution, how they may use tRNA loop copies, and why some coronaviruses are more infectious than others.

We know that coronaviruses NL63, 229E, OC43, HKU1, and possibly CCoV-HuPn-2018 are considered to be similar to the common cold, and only people

with compromised immune systems are at risk of developing more serious illnesses. While all of the coronaviruses have both T-loop and DHU loop copies as shown in Tables 1 and 2, only 229E, CCoV-HuPn-2018, and all the variants of SARS-CoV-2 have a DHU loop and anticodon copy in the right place (Table 1). I speculate they have the opportunity, when admitted to a cell nucleus, to cause changes that help their infection agendas. However, 229E, first noticed in the 1800s, may not have evolved to where it can cause more trouble and appears as a common cold. CCoV-HuPn-2018, recently migrated from an intestinal disease in canines to a respiratory disease in humans, may not be able to interfere with human cell functions and also appears as a common cold. SARS-CoV-2, however, has been shown to be more aggressive.

The T-loop data in Table 2 show double matches only for SARS, MERS, and SARS-CoV-2. I speculate they have the ability, when admitted to a cell nucleus, to cause changes that help their infection agendas. SARS, MERS, and the earlier versions of SARS-CoV-2 each have a single T-loop/anticodon double match. As a respiratory disease spread through the air, SARS-CoV-2 may be more infectious than the close contact needed by SARS and MERS. The omicron variant of SARS-CoV-2 may be even more infectious because it has two T-loop double matches, giving it a better chance to enter a cell nucleus.

Inspecting Tables 1 and 2 more closely, you'll notice that SARS has a T-loop match for the amino acid lysine and SARS-CoV-2 has that same match in addition to a DHU loop match for the amino acid glycine. Omicron has the same amino acid matches as SARS-CoV-2 plus another T-loop match for the amino acid valine. I speculate that these matches indicate an evolutionary path from SARS to SARS-CoV-2 to omicron, all to improve its infection ability. The other SARS-CoV-2 variants are unsuccessful attempts. In my opinion, SARS, SARS-CoV-2, and omicron are the same disease that has continued to evolve and should be under a single general label.

The nuclear membrane should only admit tRNAs it thinks the nucleus needs. Shaheen et al. found that if the nucleus is deficient in amino acids, specific tRNAs are built and sent to the cytoplasm to bring them back. For MERS, SARS, and SARS-CoV-2, this may allow at least part of the virus to enter the nucleus. Campbell and her colleagues found that a significantly higher proportion of the amino acid lysine is needed during mitosis. Referring to Table 2, you see that the SARS and SARS-CoV-2 have double matches for lysine, while the MERS double match is for tyrosine. I suggest the specific matches in SARS and SARS-

CoV-2 make their entry to the nucleus easier, allowing those coronaviruses to be more infectious than MERS.

cell type	turnover time
small intestine epithelium	2-4 days
stomach	2-9 days
blood Neutrophils	1-5 days
white blood cells Eosinophils	2-5 days
gastrointestinal colon crypt cells	3-4 days
cervix	6 days
lungs alveoli	8 days
tongue taste buds (rat)	10 days
platelets	10 days
bone osteoclasts	2 weeks
intestine Paneth cells	20 days
skin epidermis cells	10-30 days
pancreas beta cells (rat)	20-50 days
blood B cells (mouse)	4-7 weeks
trachea	1-2 months
hematopoietic stem cells	2 months
sperm (male gametes)	2 months
bone osteoblasts	3 months
red blood cells	4 months
liver hepatocyte cells	0.5-1 year
fat cells	8 years
cardiomyocytes	0.5-10% per year
central nervous system	life time
skeleton	10% per year
lens cells	life time
oocytes (female gametes)	life time

Figure 8. Cell Replacement Schedule
Harvard University

Figure 8 shows the cell replacement schedule from cell mitosis. Notice that the lung cells are replaced every eight days, corresponding to the time people infected with SARS-CoV-2 report their breathing problems. Yoo et al. found the gene ORF6 in SARS-CoV-2 that inhibits our immune system and allows the SARS-CoV-2 disease to spread more easily. This same gene is in SARS, but not in any of the other coronaviruses. Immune system suppression is a key factor to make a disease more infectious.

Inclusion of human DHU and T-loop copies linked with anticodon matches appears to be an evolutionary tactic meant to improve infection success. As described by both Marshall and Brody, our immune system may become

overwhelmed fighting MERS, SARS, or SARS-CoV-2, allowing latent diseases within our body to attack other organs. We should be prepared for future coronaviruses to also use the tRNA loops with anticodon matches as an effective means for more comprehensive and successful infections.

Vaccine Development

The best way to limit the effects of a disease is to develop a vaccine against it. SARS-CoV-2 vaccines have been developed in an incredibly short time, made to identify spikes in the virus protein shell and keep them from connecting with our cells so the virus is never injected. However, the virus within the shell is ignored in these vaccines. This approach initially works well but is doomed to fail because of the Darwin effect, described in the next paragraph.

Let's say 99.9% of the virus is contained in a shell identified by the vaccine. Those viruses will (hopefully) all be destroyed, but the 0.1% that missed being destroyed will escape and multiply. Also, viruses mutate frequently, and the easiest mutation is in the protein shell and spikes. Our immune system is usually able to handle these variants, but when one develops that infects enough of our cells, such as we've seen in the SARS-CoV-2 omicron variants, then our immune system is less effective. As shown in Figure 2, the variety of different protein shells is nearly unlimited. This cops-and-robbers game continues with new vaccines and newly mutated viruses, with the robbers always winning. A different kind of vaccine is needed.

If the speculation that SARS-CoV-2 enters the cell nucleus and associates its RNA with our DNA is correct, then perhaps identifying that process for our immune system would limit those infections. This might be done by eliminating the troublesome nucleotide sequences in the virus, creating a mild version that can be used as a vaccine. Those relatively benign viruses would be seen by our immune system as alien and be destroyed, while at the same time recognizing them, and the real virus, more easily later. This approach is similar to the successful vaccines developed for smallpox and polio, where immune system antibodies are created to fight the benign disease (the vaccine) and would also fight the active virus if it appears. For MERS, eliminate the nucleotide string GTTTGATTC for the benign vaccine; for SARS, eliminate the nucleotide string GTTCATGTC; for SARS-CoV-2, eliminate the nucleotide strings TAGTGGTGAG, GTTCATGTC, and GATCAAAAC. Positive results would

mean an overall virus vaccine has been created, rather than just a protein spike vaccine.

Summary

Each of the human-directed coronaviruses was found to have several nucleotide sequences that matched some of the T-loops and DHU loops of human tRNA. It was also found that several coronaviruses had anticodon codes, placed identically to a cellular tRNA, that matched the T-loop or DHU loop amino acid nucleotide sequence. Those sequences raised the possibility that a coronavirus could fool the nuclear membrane and enter the cell nucleus to associate itself with the human DNA. Those DHU and T-loop matches could allow coronaviruses to expand their infection agendas and possibly compromise our immune system, permitting other diseases within our bodies to attack their organs of choice. Such additional infections have caused significant problems for individuals who have recovered from SARS-CoV-2. Vaccines that attack the virus protein shell spikes are readily overcome by virus mutations, suggesting that successful vaccines should be directed towards the internal virus RNA infection process. For SARS-CoV-2, this means eliminating the nucleotide sequences TAGTGGTGAG, GTTCATGTC, and GATCAAAAC to create a benign virus as a vaccine; for MERS, this means eliminating the nucleotide sequence GTTTGATTC; for SARS, this means eliminating the nucleotide sequence GTTCATGTC.

In addition, from a tRNA perspective SARS appears to have evolved into SARS-CoV-2 which has further evolved into omicron, all to improve its infection ability. The other SARS-CoV-2 variants are unsuccessful attempts. In my opinion, the amino acid matches indicate that SARS, SARS-CoV-2, and omicron are the same disease that has continued to evolve and should be under a single general label.

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Appendix

Table A-1
Amino Acids and
Associated Human tRNA Loops

Amino Acid	Associated tRNA T-Loop	Associated tRNA DHU Loop
Alanine (Ala)	GTTCGATCC, GATCGATGC, GATCAATGC, GTTCAATCC, GATCGACGC, GTTCGACCC	CAGTGGTAG, CAAGTGGTAG, CAAATGGTAG, CAAGCGGTAG, CAGCGGTAG, CAGGCGGTAG
Arginine (Arg)	GTTCGACTC, GTTCGAGTC, GTTCGAATC, GTTTGAGTT	CAATGGATAA, TAATGGATAA, TGATGGATAA, CAATGGATAG, CAATGGACGAG
Asparagine (Asn)	GGTTCGATC, GGTTCGAAC, GGTTCGAGC, GGTTCCAGC, GTTCGAGAC, GTTTGAACC	CAATCGGTTAG, CAATCGGCTAG, CAATGGGTTAG, TAGTCGGTTA
Aspartic acid (Asp)	GTTCAATTC, GTTCGATTC	TAGTGGTGAG, TAGTGGTTAG, TAGTGGTGAGTA, TAGTGGTGAGTG

Cysteine (Cys)	GTTCAAATC, ATTCAAATC, GTTCAAATC	CAGTGGTAG, TAGCGGTAG, CAGGTGGTAG, CAGGGGTAG, TAGGGGTAG, CACAGGTAG, CAGTGGGTAG
Glutamine (Gln)	GTTCGAGTC, GTTCGAATC, GTTCAAATC	TAATGGTTAG, TAATGGTGAG, TAATGGTAAG, TAATGGTCAG
Glutamic acid (Glu)	GTTCGACTC, GTTCGATTC	TAGTGGTTAG, TAGTGGCTAG, TAGCGGTTAG
Glycine (Gly)	GTTCAATTC, GTTCGATTC, GTTTGATTC, CTTCGATTC, GCTCGATTC	TAGTGGTGAG, TAGTGGTTAG, CAGTGGTAG, TCAGTGGTAG, TAGTGGTAAG, TAGTGGTAT, CAATGGTAG
Histidine (His)	GTTCGAATC	TAGTGGTTAG
Isoleucine (Ile)	GTTCGATCC, GGTTCGATC, GGTTCGAAC, GCTCGACTC, GTTCGAGCC, GTTCAAGCC	CAATCGGTTAG, CAGTTGGTTAG, CAGTTGGTCAG, CAGTCGGCTAG, CAGTTGGTAAG

Leucine (Leu)	GTTCGAATC, GGTTCGAAC, GGTTCGAGC, GTTCAAATC, GTTTGAATC	CAGTAGGTAG, GAGCGGTCTAA, GAGTGGTCTAA, GAGCAGTCTTAA, GAGTGGTTAA
Lysine (Lys)	GGTTCGAGC, ATTCGTGCC, CGTGCCCCA, GTTTGAGCC, GCTCGAGCT, GTTCAAGTC, GTTCATGTC	CAGTCGGTAG, CAGTCGATAG, CAGTCGGCAA, CAGTCGGTGG, CAGTTGGTAG, CAGTCAGTAG
Methionine (Met)	GTTCGAGCC, GTTCGAACC	CAGTAGGTAG, CAGTGGGCAG, CAGTAGGCAG, CAGCGGGCAG, CAGCTGGCAG
Phenylalanine (Phe)	GTTCGATCC, GTTCAATCC, GTTCAACCC	CAGTTGGGAG
Proline (Pro)	GTTCAAATC	TAGGGGTAT
Serine (Ser)	GTTCGAATC, GGTTCGAAC, GTTCAAATC	GAGTGGTTAA
Threonine (Thr)	GTTCGATCC, GGTTCGAAC, GTTCAAATC, GTTCGAATC, GTTCGACTC,	TAGTTGGTTAA, TAGCTGGTTAA, TAGCTGGTCAA, AAGTGGTAA, TAGTTGGCTAA, CAGCGGTTGG, CAGTGGTTAG, CAGGGGTTAG

Tryptophan (Trp)	GTTCGAATC, GTTCAAATC, GTTCAAGTC	CAACGGCAG, CAACGGTAG
Tyrosine (Tyr)	GTTCGAATC, GTTCAATTC, GTTCGATTC, GTTTGATTC	CAGTTGGTAG, CAGCTGGTAG
Valine (Val)	GTTCGATCC, GGTTCGAGC, GTTCGAAAC, GATCAAAAC, GTTTGAAAC, GGTCGAAAC	CAGTGGTAG, TAGTGGTTAT, TAGTGGTCAT, TAGCGGTTAT, GAGTGGTTAT