Calculating the COVID-19 infected and asymptomatic carrier distribution by blood type, and the COVID-19 death rate by race

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Abstract

Despite the apparent decline of COVID-19 in May, the resulting attempts to reopen saw the SARS virus running rampant in the form of a pandemic once again, even two months later in mid-July. During this time period, two major questions have been raised regarding the nature of COVID-19 and the response towards it. The first is whether or not human blood types have any relevance towards being infected. Due to the numerous and differing answers, we wanted to see if there were any other possible connections between blood type and infectivity. The second question that has been continually asked is why African Americans and Latinos have such a high death rate compared to how much of the population they make up while Caucasians have a notably lower death rate compared to their population. Some have speculated that there is a biological difference between them that has caused this while others cite the different social standings and subsequent environments that come as a result of that that makes some races more susceptible to COVID-19. Concerning this second question, we wanted to figure out if the death rate disparities were due to biological or social reasons. By creating a unique model and deriving two formulas from it, we solved the two aforementioned questions. Through this, we have uncovered what we believe to be key to understanding this pandemic more clearly: the role of asymptomatic carriers. We have also developed two calculations that determine the percentage of asymptomatic carriers out of those infected with COVID-19, as well as the percentage of a child becoming an asymptomatic carrier if both parents are asymptomatic. These ideas will be in our conclusion.

Background

The world being lulled into a false sense of security due to the apparent effectiveness of the various preventive measures combined with the rapidly deteriorating condition of the nations' economies led to a global relaxation on those aforementioned preventive measures. The economic shutdown was stopped and stores were reopened, only to see the number of cases in

the United States jump from 1.252 million by May 7 to 3.874 million by July 21, as well as the number of deaths (also in the United States) go from about 75,000 by May 7 to about 141,000 by July 21. The threat the virus posed towards my community—especially with the possibility of me having to return to school in-person in a state with as many cases as California—pushed me to further investigate the matter of the virus. While reading a CNN article discussing the possibility of blood types affecting Coronavirus infectivity, the idea of the virus carrying their host's blood group antigens was brought up, which propelled me further in investigating such an idea. Having plenty of time, I looked into pathology, immunology, genetics, the 2003 SARS virus, and influenza to try to uncover and unite what information might have been overlooked. The question of blood types affecting how the virus interacts with a body was first brought into the light by a Chinese study of COVID-19 patients in Wuhan and Shenzhen, which was then quickly followed up by another study of the same topic by Columbia University. However—even without any capacity to actually conduct scientific tests, being a junior at Maranatha High School—I wished to dive deeper into this question by collecting data and analyzing it to find my own answers.

Methods

Because we wanted to mathematically derive our answers, we started by setting up three hypotheses. Then we used them to create our model, which was used to derive two formulas. We named these two formulas Formula #1 and Formula #2. The former found the percentage of symptomatic carriers out of a race via their blood type while the latter calculated the racial death rates of the United States using their blood type.

Data and results gathered by various scientific institutions in Wuhan, China[1] and Columbia University in New York[2] were used as the launch pad for this investigation, with both concluding that people with Type O+ blood had a lower risk to be infected while those with Type A+ blood had a higher risk to be infected. However, the Massachusetts Institute of Technology [3] and Harvard Medical School[4] both spoke out, saying that due to there being not enough evidence, this potentiality of blood being related to infection should not be accepted as fact. Because of the numerous and differing answers, we wanted to see if there were any other possible connections between blood type and infectivity. Acting as the base for the rest of the project was the article, "Why do we have different blood types—and do they make us more vulnerable to Covid-19?", from CNN[5]. The article cites research director Jacques Le Pendu of the French medical organization Inserm to have claimed that there was a "likelihood that the virus will carry the infected person's blood group antigen" and as a result interact like how blood interacts during a transfusion; someone who had Type O blood might destroy the virus if that virus came from someone who had Type A blood. This idea provided the inspiration for our three new hypotheses that were developed to resolve what was left unanswered by the article.

Hypotheses

1. The SARS-CoV-2 virus has four variants, with the original antigens of the virus having been replaced with specifically human blood group antigens: A antigens, B antigens, and Rh(D) antigens. The first variant, SARS2-A, has A antigens and Rh(D) antigens. The second, SARS2-B, has B antigens and Rh(D) antigens. The third, SARS2-AB, has A antigens, B antigens, and Rh(D) antigens. The last, SARS2-O, has only Rh(D) antigens.
2. The virus's variants' A and B antigens are minutel flawed. When a virus carrying A antigens infects a body with Type AB+ blood, the antigens of the virus are detected as foreign invaders, with the same being applicable for when a virus with B antigens infects a body with Type AB+ blood.
3. The four SARS-CoV-2 virus types all exist in equal numbers.

When a SARS2-A virus enters a body with Type A blood, both the A antigens and Rh(D) antigens on the virus allow it to not trigger the innate immune system of the body. This is due to it having nearly identical antigens. From there, the virus is able to enter into a cell, with its A and Rh(D) antigens acting as a key. After replicating itself, the virus can leave the cell without lysing it. Due to SARS-CoV-2 operating much like how the influenza A virus acts, it will instead bud, resulting in the mature viruses leaving the cell host without destroying it. This means that the adaptive immune system will not be triggered either. Since no threat was initially detected, the antigen-presenting cells (APCs) have no foreign antigens to bring back to the lymph nodes, all of which would normally start a cellular manhunt for the intruder. Most importantly, the antigens work as two-way keys. In conclusion, because it has those human antigens, the SARS2-A virus is able to slip into the body, enter a cell, reproduce within it, leave it, and continue reproducing all without exciting either the innate or adaptive immune systems. In this case, the body shows no symptoms despite being a carrier of the virus—a situation that creates an asymptomatic carrier of this specific variant of SARS-CoV-2. However, this is only the case when the blood group antigens of the virus and body are compatible. When this compatibility is present, though, the infected person becomes an asymptomatic carrier. If the SARS2-A virus had entered into a body with Type B blood, then the infected body would detect the A antigens as irregular through its HLAs (human leukocyte antigens), which would cause the body to view the virus as a pathogen and, in turn, throw the innate immune system into action. However, due to the SARS2-A having the Rh(D) antigen, which is on the body's whitelist (that is, recognizable as normal in the body by the HLAs), the virus could still enter into the body's cells. After budding from that cell, though, it will still be noticed by the APCs due to its B antigen. Both examples of the SARS2-A invading—no matter how far it gets before being detected—would cause people to show symptoms. When the virus and body's antigens are incompatible, the body reacts as if it had been infected with influenza, causing the flu-like symptoms COVID-19 has been observed to impart on those it infects. This results in the infected person becoming a symptomatic carrier.

Model Table

The following table outlines the outcomes of the four SARS-CoV-2 types infecting the four major blood groups (A+, B+, AB+, O+). This is based off of the previous three hypotheses. Note that two of the results of infecting AB+ is incongruent with how blood transfusions works, since a virus with A or B antigens still results in a symptomatic carrier.

▢ - Asymptomatic carrier

△ - Symptomatic carrier

Virus types	Infected blood type	Infection result	
SARS2-A	A+	▢	2 △: When all 4 SARS2 types infect Type A+ blood, 2 of the infections will result in a symptomatic carrier.
SARS2-B	A+	△	
SARS2-AB	A+	△	
SARS2-O	A+	▢	
SARS2-A	B+	△	2 △: When all 4 SARS2 types infect Type B+ blood, 2 of the infections will result in a symptomatic carrier.
SARS2-B	B+	▢	
SARS2-AB	B+	△	
SARS2-O	B+	▢	
SARS2-A	AB+	△*	2 △: When all 4 SARS2 types infect Type AB+ blood, 2 of the infections will result in a symptomatic carrier.
SARS2-B	AB+	△*	
SARS2-AB	AB+	▢	
SARS2-O	AB+	▢	
SARS2-A	O+	△	3△: When all 4 SARS2 types infect Type O+ blood, 3 of the infections will result in a single symptomatic carrier.
SARS2-B	O+	△	
SARS2-AB	O+	△	
SARS2-O	O+	▢	

*Results founded according to Hypothesis #2

Formula #1

The Percentage Base formula (or PB formula) was created through the Model Table. This formula calculates the percentage of symptomatic carriers out of an area's population according to that population's blood group distributions.

Variables:

- A = percentage of Type A+ blood in population
- B = percentage of Type B+ blood in population
- AB = percentage of Type AB+ blood in population
- O = percentage of Type O+ blood in population

PB (Total infected percentage base) = 1/2A+1/2B+1/2AB+1/3O

- A+, B+, and AB+ blood types have a constant of ½ because, according to the Model Table, 2 of the infections produce a symptomatic carrier (assuming all 4 are different virus types).
- O+ blood type has a constant of ⅓ because, according to the Model Table, 3 of the infections produce a symptomatic carrier (assuming all 4 are different virus types).

A' (A+ type infected percentage) = A/2PB
B' (B+ type infected percentage) = B/2PB
AB' (AB+ type infected percentage) = AB/2PB
O' (O+ type infected percentage) = O/3PB

The Chinese study group recorded the blood group percentages out of a group of 3694 healthy citizens and the infected percentages by blood type out of a group of 1775 patients who had COVID-19.

Wuhan Table #1

Blood group	Population blood groups	Infected
A+	32.2% (1188)	37.7% (670)
B+	24.9% (920)	26.4% (469)
AB+	9.1% (336)	10.0% (178)
O+	33.8% (1250)	25.8% (458)

Calculations via Wuhan Table #1 second column data:

PB (Total infected percentage base) = 1/2A+1/2B+1/2AB+1/3O

PB = (½*32.16) + (½*24.90) + (½*9.10) + (⅓*33.84) = 44.36
A' = A/2PB = 32.16/2(44.36) = 36.2%
B' = B/2PB = 24.90/2(44.36) = 28.1%
AB' = AB/2PB = 9.10/2(44.36) = 10.3%
O' = O/3PB = 33.84/3(44.36) = 25.4%

We put our calculation results and the input data together to create underneath Wuhan Table #2.
Wuhan Table #2

Blood group	Population (Wuhan data)	Infected (Wuhan data)	Infected (Calculated)	p-value
A+	32.2%	37.7%	36.2%	0.015
B+	24.9%	26.4%	28.1%	0.017
AB+	9.1%	10.0%	10.3%	0.003
O+	33.8%	25.8%	25.4%	0.004

Wuhan Shenzhen study
We used the same formula (Formula #1) that we used to check the Wuhan study's data to also check the data from the Wuhan Shenzhen study. The original data will be placed in Shenzhen Table #1 and the results in Shenzhen Table #2.

Shenzhen Table #1

Blood group	Population blood groups	Infected
A+	28.8% (6728)	28.8% (82)
B+	25.1% (5880)	29.1% (83)
AB+	7.3% (1712)	13.7% (39)
O+	38.8% (9066)	28.4% (81)

Shenzhen Table #2

Blood group	Population (Shenzhen data)	Infected (Shenzhen data)	Infected (Calculated)	p-value
A+	28.8%	28.8%	33.08%	0.147
B+	25.1%	29.1%	28.83%	0.009

AB+	7.3%	13.7%	8.38%	0.388
O+	38.8%	28.4%	29.7%	0.046

In the "Infected" column of Shenzhen Table #1 for AB+ individuals, the 39 people used for the group is significantly smaller than the 178 used to the Wuhan study, resulting in more inaccurate values from our calculations. When such sample amounts are too low, as was seen in the number of Shenzhen infected AB+ people (39), it affects the accuracy of the results, which in turn causes the difference between the given data and our calculated data to be greater.

Columbia University NYP study

We will use the same formula (Formula #1) and its calculating processes that we used to check the Wuhan study's data to also check the data from Columbia University's NYP. The original data will be placed in NYP Table #1 and the results in NYP Table #2.

NYP Table #1

Blood group	Population blood groups	Infected
A+	32.7% (35643)	34.2% (233)
B+	14.9% (16229)	17.0% (116)
AB+	4.2% (4582)	3.1% (21)
O+	48.1% (52406)	45.7% (312)

NYP Table #2

Blood group	Population (NYP data)	Infected (NYP data)	Infected (Calculated)	p-value
A+	32.7%	34.2%	39.0%	0.140
B+	14.9%	17.0%	17.8%	0.047
AB+	4.2%	3.1%	5.0%	0.613
O+	48.1%	45.7%	38.2%	0.164

Comparing the size of the infected group of AB+ individuals used by the Wuhan Shenzhen study (39) and that of Columbia University's study (21), the near decrease by half between the former and the latter have also resulted in a near doubling in the difference between our calculated infected percentage and the percentage presented by their data. The difference went from 0.388 with the Shenzhen data to 0.613 with the Columbia data. Like what we have mentioned

concerning the Wuhan Shenzhen study, all the other differences have also gone up between the Shenzhen calculations and the NYP calculations.

According to our Model Table and formula [PB (Total infected percentage base) = 1/2A+1/2B+1/2AB+1/3O], we made a calculation to get the value for **the infected rate of O+ blood type people compared to those with either of the three other blood types:**
ID (Infection difference) = ½ - ⅓ = 17%.

Formula #2

This formula calculates the rates of COVID-19 deaths by race in the entire United States. Upon being infected by a SARS2-O, all four blood types will result in asymptomatic carriers. Also, only Type O+ people will be symptomatic *when infected by any of the three SARS-CoV-2 virus types*, with the exception of SARS2-O. This is the reason why the distribution of Type O+ blood for the four major races is used to determine their death rates.

Definition:

- **Virus Density Constant**: The VDC is used to assume that the virus has a total strength value; this value will not increase or decrease, it is a constant. The virus attacks all races with indifference (according to our model and Formula #2 itself, we will explain this in the conclusion). However, after looking at the CDC's COVID-19 statistics, we want to uncover why it seems like the virus divides this strength into different amounts according to different races.
- **Racial herd behavior viral allocations (Allocation values)**: When a race is attacked by the SARS-CoV-2, *their herd behavior in response towards the attack can affect how badly they are affected by the virus*, which is represented by our allocation value from the VDC. However, the behavior of people can change, be it suddenly or gradually, causing their allocation value to also change to further reflect their actions. So, we can say that the overall behavior of the race affects how much the virus divides its strength into different amounts. We defined four variables—WA, LA, BA, and AA—to represent the racial herd behavior viral allocations.
- **Common Herd Behavior Virus Allocation Set (Common Set)**: The Common Herd Behavior Viral Allocation Set is the set of allocation values that has proven to be the most common and accurate values for each race in our death rate calculations.
    - WA (Caucasian allocation value) = 1.00
    - LA (Latino allocation values) = 0.80
    - BA (African American allocation values) = 1.50
    - AA (Asian allocation values) = 1.00

Variable table:

Races	Population Percentages	Allocation Values
Caucasian	WP	WA
Latino	LP	LA
African American	BP	BA
Asian	AP	AA

While the four allocation values of the races are variable, the product of these four values must be the VDC, or the virus's total strength value, 1.2.
VDC = WA * LA * BA * AA = 1.2

Constants:

- Wo (percentage of Caucasians with Type O+ blood) = 37%
- Lo (percentage of Latinos with Type O+ blood) = 53%
- Bo (percentage of African Americans with Type O+ blood) = 47%
- Ao (percentage of Asians with Type O+ blood) = 39%
- VDC (Virus Density Constant) = 1.2

The Common Set's Use: Using the Common Herd Behavior Viral Allocation Set, we have accurately calculated both the national death rates and the death rates of 20 states and also defined this as the first group. These 20 states make up 56% of the 36 states we did calculations for.
Altered Allocation Values: A few states required that the viral allocation values be altered to acquire accurate death rates. There were 16 other states (44% of the states we did calculations for) that required viral allocations that were altered from the Common Set to find an accurate rate; this is group two.
Formula #2 as an Observational Tool: These 36 states, which were what we used for testing Formula #2, could all have their racial death rates be accurately calculated by Formula #2. Due to the high success rate of this formula, we believe it can be a viable tool for identifying if there are any anomalies in the death rates that have occurred due to oddities in human behavior or errors in data collection, which would then cause our formula to fail. When our formula fails to be accurate, it can act as an alarm against irrational behavior in the pandemic on the national stage.

Racial death value equations:

WD (Caucasian) = WP * Wo * WA
LD (Latino) = LP * Lo * LA
BD (African American) = BP * Bo * BA
AD (Asian) = AP * Ao * AA

National Racial Death Rates:

The CDC derived the following data out of 112,827 COVID-19 deaths in the United States. They recorded the death rates of the four major races via percentages. However, because the CDC recorded the percentage of deaths of all races in the United States, including smaller minorities such as Native Africans and Pacific Islanders, the percentage of deaths of these four major races were recorded as the percentages in column 2 of Table #3. Column 3 shows the percentages of column 2 after being converted to only account for those four races.

Table #3: Racial percentage of deaths

Race (four major races targeted in project)	Percentage of deaths out of 112,827 from the CDC	Percentage of deaths according to four major races
Caucasian	49.9%	53%
Latino	17.2%	18%
African American	22.2%	23%
Asian	5.2%	6%

Below are the population distributions used for the variables (WP, LP, BP, AP) after being converted to only account for those four races:

Table 4:

Four Major US Races	United States Population	Variable
Caucasian	63%	WP
Latino	19%	LP
African American	13%	BP
Asian	5%	AP

The formula below was used to calculate each race's death value (not death rate):

WD = WP * Wo * WA
LD = LP * Lo * LA
BD = BP * Bo * BA
AD = AP * Ao * AA

We used the Common Set to calculate each race's death value:
WD = 63% *37% * 1 = 0.2331
LD = 19% * 53% * 0.8 = 0.0806
BD = 13% * 47% * 1.5 = 0.0917
AD = 5% * 39% * 1 = 0.0195

The racial death values from above were placed into column 4 of Table #5, and then converted into the racial death rate percentages in column 5.

Table #5: Calculated national death values and rates

Four Major US Races	United States Population	Racial death rate (CDC)	Racial death values	Racial death rate (Calculated)	p-value
Caucasian	63%	53%	0.2331	55%	0.04
Latino	19%	18%	0.0806	19%	0.06
African American	13%	23%	0.0917	22%	0.04
Asian	5%	6%	0.0195	5%	0.17

Our calculated racial death rates were placed in column 5 to allow for a comparison with the CDC's racial death rates.

State Racial Death Rates:
After calculating the national racial death rate percentages with Formula #2, we then used it to find the racial death percentages of several states. We have calculated the racial death rates of 36 states. Most of the racial death rates of the other 14 states were not calculated because of unreliable data. We formed two groups according to whether or not the Common Set was used. The first group's calculated results, which used the Common Herd Behavior Viral Allocations Set, had high accuracy when compared to the provided death rates. The second group consists of calculations where the Common Set was not employed. The Common Set was instead shifted to new sets of allocation values to calculate accurate death rates because of a change in herd behavior. Since we used allocation values to describe the outcome of a race's behavior in response to the virus, a change in behavior would have been reflected in a set of new values.

First group: Consists of 56% of the results out of the 36 states with calculated results. All calculations used the Common Herd Behavior Viral Allocation Set and were highly accurate.

The examples below are of the state death rate calculations. All the calculations will be listed in the "Formula #2 death rates by race for 36 states" document. Details will be listed in the "References" section[6].

Colorado death rate by race: RDR = Racial Death Rate

Races	Population	RDR (CDC)	RDR (Calculated)	p-value
Caucasian	68%	62%	65%	0.05
Latino	22%	22%	24%	0.09
African American	4%	7%	7%	0.00
Asian	3%	3%	3%	0.00

New York death rate by race:

Races	Population	RDR (CDC)	RDR (Calculated)	p-value
Caucasian	74%	63%	68%	0.08
Latino	12%	15%	13%	0.13
African American	9%	18%	16%	0.11
Asian	4%	4%	4%	0.00

Second group: The second group consists of calculations where the Common Set was not employed. Instead, new sets of allocation values were used to calculate accurate death rates. This group makes up 16 of the 36 state calculations—44% of these calculations.

Below is where we changed the herd behavior viral allocation values because the Common Set gave inaccurate results. Rows that are highlighted green show when that race's death rate is exceptionally lower than what is common, and rows that are highlighted red show when that race's death rate is exceptionally higher than what is common. The need to change the allocation values from the Common Set points towards some sort of social behavior from a race or special treatment towards that race that saw an extreme growth or decrease in their death rate. We can observe an obvious situation where a change in the death rate from the green row will cause a shift to the race of the red row. As to how this occurred, it will be explained in the conclusion.

California death rate by race:

Allocation values: WA = 0.88, LA = 0.90, BA = 1.50, AA = 1.00

Races	Population	RDR (CDC)	RDR (New set)	p-value	RDR (Common set)**
Caucasian	39%	31%	31%	0.00	34%
Latino	41%	48%	47%	0.02	42%
African American	5%	9%	8%	0.11	9%
Asian	15%	13%	14%	0.08	15%

**Common Set rates are provided to allow for comparisons of the RDR between it and the CDC. If there is a major disparity between the RDR of the Common Set and the CDC, it reflects two possible situations: that race's death rate is lower than usual if the Common Set's rates are much higher than the CDC's (lower death rate highlighted in green), or that race's death rate is higher than usual if the Common Set's rates are much lower than that CDC's (higher death rate highlighted in red).

Connecticut death rate by race:

Allocation values: WA = 1.50, LA = 0.53, BA = 1.50, AA = 1.00

Races	Population	RDR (CDC)	RDR (New set)	p-value	RDR (Common set)
Caucasian	66%	73%	73%	0.00	60%
Latino	17%	9%	9%	0.00	18%
African American	10%	15%	14%	0.07	17%
Asian	5%	1%	4%	1.00	5%

The calculation of the Asymptomatic Carrier Percentage:

Formula #3 was created to calculate the Asymptomatic Carrier Percentage (ACP), which shows the chance for someone who is infected with COVID-19 to be an asymptomatic carrier. It was based off of the model shown in the Model Table.

ACP = (¼ * 2/4) + (¼ * 2/4) + (¼ * 2/4) + (¼ * ¼) = 43.75%

- The first term represents Type A+ infections, the second Type B+ infections, the third Type AB+ infections, and the fourth Type O+ infections.

- First ¼ in every term represents the division of the SARS-CoV-2 virus into four types (SARS2-A, SARS2-B, SARS2-AB, SARS2-O).
- The second fraction in each term represents the likelihood of that blood type being infected and the host becoming an asymptomatic carrier (see Model Table).

According to the CDC (Center for Disease Control and Prevention), the percentage of asymptomatic infections is around 40%.

The calculation of the chance of an asymptomatic child in an asymptomatic family:
Looking at our research, we believe that asymptomatic carriers play a crucial role in the spreadability and sustainability of this pandemic. To take our consideration of the threat of asymptomatic carriers a step further, we have also calculated the probability of a child becoming an asymptomatic carrier in a family with two asymptomatic parents. Using our Model Table, we have created two tables to find this answer.

Viruses carried by blood type table

Asymptomatic carrier blood type	Type(s) of virus carried
A+	A+, O+
B+	B+, O+
AB+	AB+, O+
O+	O+

Probability of asymptomatic child by parents' blood types

Parent blood types	Types of virus carried	Possible blood types of child	Calculation of child's asymptomatic probability	Results
A+ & A+	A, O	A+, O+	(½ * 2/2) + (½ * ½)	¾
A+ & B+	A, B, O	A+, B+, AB+, O+	(¼ * ⅔) + (¼ * ⅔) + (¼ * ⅓) + (¼ * ⅓)	½
A+ & AB+	A, AB, O	A+, B+, AB+	(⅓ * ⅔) + (⅓ * ⅓) + (⅓ * ⅔)	5/9
A+ & O+	A, O	A+, O+	(½ * 2/2) + (½ * ½)	¾
B+ & B+	B, O	B+, O+	(½ * 2/2) + (½ * ½)	¾
B+ & AB+	B, AB, O	A+, B+, AB+	(⅓ * ⅔) + (⅓ * ⅔) + (⅓ * ⅓)	5/9
B+ & O+	B, O	B+, O+	(½ * 2/2) + (½ * ½)	¾

AB+ & AB+	AB, O	A+, B+, AB+	(⅓ * ½) + (⅓ * ½) + (⅓ * 2/2)	⅔
AB+ & O+	AB, O	A+, B+	(½ * ½) + (½ * ½)	½
O+ & O+	O	O+	(1 * 1/1)	1

Asymptomatic child percentage= 1/10 * (¾ + ½ + 5/9 + ¾ + ¾ + 5/9 + ¾ + ⅔ + ½ + 1) = 68%

Results

Through Formula #1's calculations, we have acquired a very accurate infection rate by blood group using Wuhan's study data and result. This accuracy supports our hypotheses and model and strengthens the credibility of our results. There are two results from our first calculation. The first is that O+ blood type people are just less than 17% less likely to be symptomatically sick compared to those with any of the three other blood types. The second result is that there is a deep connection between blood types and COVID-19 infection.

The calculations we derived from Formula #2 are more complex relative to those of Formula #1. Having carefully scrutinized our calculations, we have come upon two unique points of view. First, there is a constant we call the VDC (Virus Density Constant) that represents the viruses' whole strength, which is distributed to different races according to the race's herd behavior. The diverse reactionary results to the viruses are not caused by a race's biology or social conditions, since neither can cause the value changes we have seen in a few months' time while herd behavior can. Second, there is a simultaneous interflow of death and infection rates between different races when one or more races change their majority's actions.

Another two of our calculations were derived from a need to have more information concerning asymptomatic carriers. 43.75% of infected are asymptomatic carriers and 68% of children at home with asymptomatic parents will become asymptomatic carriers as well. When these children return to the infective network, such as going back to school, they may face the same challenges and dangers that adults do.

Conclusions

Regarding our first question, where we asked if there was any relation between blood types and COVID-19 infectivity, we disagreed with the conclusions of Wuhan and Columbia University's studies. They said that those with A+ blood are more susceptible to getting infected while O+ are

less susceptible. We also did not accept the conclusions of MIT or Harvard, in that there was not enough data to prove if there was or was not any relation between blood types and infectivity. With our model and formulas, we found out that blood types play an extremely important role in the spreading of the SARS-CoV-2 virus. Because viruses have taken advantage of human blood type antigens to invade the human body, our defence against the virus through the immune system must all be related to blood type. Through our calculation, O+ blood type people are just less than 17% less likely to be symptomatically sick compared to those with any of the three other blood types.

The second question was why the death rates of some races did not match up with the percentage of the population they made up and if such a discrepancy was caused by differences in biology or social conditions. We believe that the variance was due to neither biology nor social conditions, but herd behavior. From Formula #2, we used the Common Set to describe the different races' responses to the virus in the same state. 56% of calculations used the Common Set to find accurate racial death rates. This means the majority of states have the same response to the virus based on race. But our concern was the other 44%, where the Common Set could not explain the races' reactions to the virus until we found a new set that could accurately find the racial death rates. That was when we came to our realization that the four races in the same state could have diverse and simultaneous responses, but everything will be balanced in the end. The only plausible cause we could think of for this change and diversity is herd behavior. Neither genetics nor social conditions are able to change in as short a period of time and affect such a specific area as what was observed. The people's behavior, though, is able to change quickly enough that it is reflected relatively rapidly. This herd behavior works in a kind of push-and-pull: all the races enter the infectivity network, and when one race's asymptomatic carriers increase, the danger for the other races increases. This is because the race with more asymptomatic carriers feels more confident in being more social; they have less deaths and less apparent infections, so they are more willing to interact with others. However, being asymptomatic means there is more of the virus in their body, resulting in them being more infective. Couple that with the fact that more of them are outside, and it results in their deaths and infections going down while the deaths and infections of the other three races rise. In response, the other races' will also act a certain way, creating the aforementioned push-and-pull effect.

Within what is an infective network, we discovered an extremely important role in the asymptomatic carrier. That is the reason why we developed our final two calculations: the percentage of asymptomatic carriers and the percentage of a child becoming an asymptomatic carrier. These two values led us to bring up our reservations and suggestions to all people who are suffering under these SARS-CoV-2 viruses. Through our calculation, 43.75% of people are asymptomatic carriers. They have no symptoms, but they carry more viruses than symptomatic carriers with them and are ready to spread to others even if their viruses may disappear in ten

days. If a great number of asymptomatic carriers enter into the infective network, there will be a big crisis in their community unless they change their behavior and are willing to adopt the only three useful actions that protect others from being infected: wearing masks, social distancing, and not gathering. From another of our calculations, 68% of children in an asymptomatic family will become an asymptomatic carrier. This means that a child staying at home has a higher probability of not expressing their infection through symptoms while still having the same, large amount of viruses that adults do. When these children step into the infective network in ways like going back to school, they may start to spread viruses or change their role from an asymptomatic carrier to a symptomatic carrier. That results in them starting to have symptoms of COVID-19. This explains why after businesses and schools reopened, there has been a 90% increase in COVID-19 cases amongst kids over the last month.

According to our model and calculations, anyone who contracts COVID-19 has only two paths: a symptomatic carrier or asymptomatic carrier. An asymptomatic carrier can be infected continuously by two kinds of SARS-CoV-2, as well as having a 56.25% of becoming a symptomatic carrier instead upon being infected again. A cured COVID-19 patient has a second chance to be sick because they have a chance to be infected by another one of the four SARS-CoV-2 viruses. However, they only have this second chance if they have O+ or AB+ blood. Those with A+ or B+ have only one chance to be sick.

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