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Identification of COVID-SARS Variants Based on Lactate Test Results

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***Abstract*—**In this research, it was examined whether individual COVID variants cause differences in the lactate curve of cyclists. After all, the virus variants attacked different organs in our body during the infections. During our tests, we used a traditional lactate step test, the results of which were compared with the values before the infection. In the tests, it has been proven that different virus variants show unique lactate curves. In this way, based on the lactate curve, it is possible to identify which variant caused the disease. Thanks to this, the return time has been shorten, because we can apply the best return protocol after infection to the competitors.

***Keywords*—**SARS-CoV-2, lactate step test, virus mutation, lactate profile.

I. Introduction

"SEVERE acute respiratory syndrome coronavirus 2" (SARS-CoV-2) is a beta coronavirus belonging to the coronavirus family, the first infected person was identified in China in December 2019. The rapidly spreading COVID-19 syndrome was classified as a global epidemic by the WHO in April 2020 [1]. The new SARS-CoV-2 enters the target cell via ACE2 receptors. Since 83% of these receptors are located on the epithelial II cells of the lung alveoli, the lung is the primary target of viral infection. Another large number of ACE2 receptors are found on the cells of the kidney, heart, endothelium, and colon, which explains the involvement of organs in the creation of multiorgan dysfunction [2].

In the first wave in Hungary (February-July 2020), most of the diseases with serious outcomes were caused by diffuse alveolar damage indicated by Acute Respiratory Distress Syndrome (ARDS), mainly in the elderly population (over 65 years of age). Over activity of T cells leads to an increased concentration of cytokines, the syndrome called cytokine storm is also one of the main causes of fatal infections [3]. High activity of the blood coagulation cascade leads to DIC (Disseminated Intravascular Coagulation) syndrome - blood coagulation disorders: bleeding or the formation of blood clots [4]. COVID-19 can also be associated with diseases affecting the cardiovascular system. The most common symptoms; are fever, headache, cough, runny nose, loss of sense of smell and taste, shortness of breath, less common; intestinal symptoms, altered state of alertness, and dizziness [5], [6]. Factors influencing the serious course of the disease are gender, age, existing chronic diseases (high blood pressure, diabetes, obesity, chronic lung diseases, heart, liver, and kidney diseases, tumors, allergies, asthma), and socioeconomic parameters (social status, diet, geographical and ethnic differences, level of care) [12], [7].

During the second wave (July 2020-January 2021), many more patients were identified in Hungary, many more young people, which resulted in a much lower rate of fatal infections. In Fig. 1, we can see the age group distribution of the victims of COVID-19 during the first three waves.

During the third wave (February-April 2021), according to virologist Dr. Miklos Rusvai, the diseases were caused by mutations that spread more and more quickly in Hungary and were much more infectious than the original virus, especially the so-called omicron virus [8], which spreads 40-80% faster [9]. Davies et al., in their research, found that the mortality of this variant was 55% higher than that of previous variants [10]. Due to the larger number of cases with a more serious outcome, the chance of developing cytochrome storm and metabolic abnormalities is also higher.

The basic hypothesis of our research was that each COVID-19 variant/mutant results in a unique lactate curve. After all, as we know, each variant attacked different areas of the human body.

During COVID-19, many athletes have indicated that their metabolism has changed. This is what Ardestani and Azizi [14] also in his research. In their research, they write about how the glucose level in the body of the spores changes as a result of COVID-SARS (Fig. 2).

If changes in glucose can be traced, it also follows that lactate production also differs from the state before the infection. We investigated this in our research.

II. Material and Method

In this research, we investigated how infections and waves with different symptoms change the metabolism. Our hypotheses are:

- change the lactate curve measured during the step test
- if it changes, they must show uniqueness of this change.

During the research, we tried to conduct the sampling in such a way that at the time of the survey, the competitors had almost the same indicators (Chronic Training Load, Acute Training Load, Training Stress Balance) as before the infection. In this way, we can more precisely compare whether there is a measurable difference or not.

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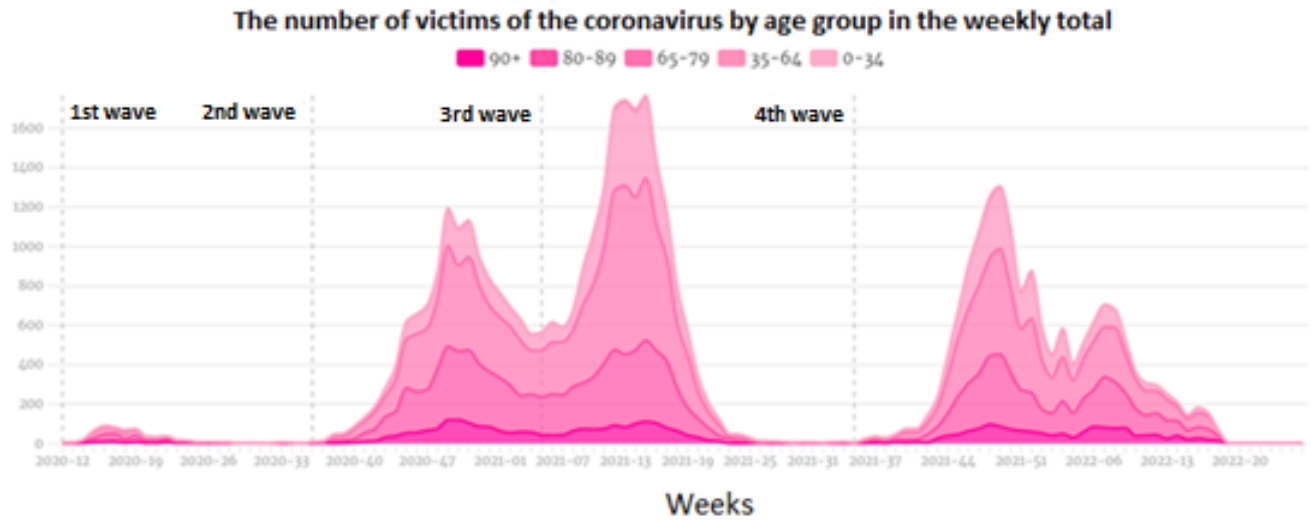

Fig. 1 The age group distribution of the victims of COVID-19 during the first three waves [13]

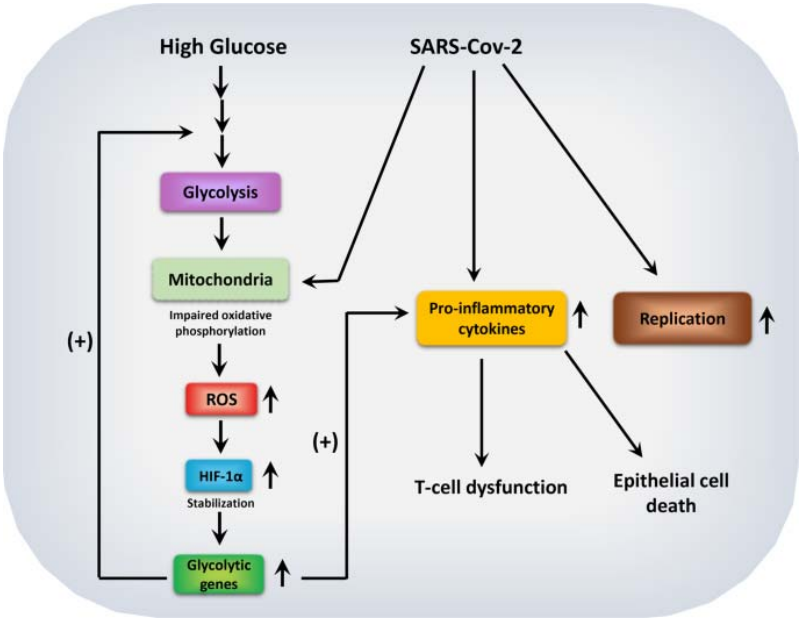

Fig. 2 Proposed model of how glucose induced metabolic reviewing potentiates SARS-CoV-2 replication and cytokine production [14]

Twelve competitive cyclists took part in our research. They had been tested for lactate from the years before the virus, so we had a basis for comparison of the condition before and after the infection. The average age of the riders is 21 ± 3 years, their body weight is 61 ± 2 kg, and the gender distribution is seven women and five men. The lactate test was performed on the Cyclus2 lab ergometer, and the lactate measurement was performed with the Lactascout 4.0 machine. The Freiburg 2 model was used to determine the degree of acidification. Sampling was done from the fingertip, as we also took the sample from there during the previous tests. We strove to ensure that the sampling took place as accurately as possible, similar to the previous ones. The measurements were performed at the same time as the previous tests, thus reducing the daily fluctuations of the biometric parameters. All of the athletes who participated in the research suffered from the infection. We examined three riders after the first wave, four riders after the second wave, and five riders after the third wave. Before the test, all the athletes participated in a 3D echocardiogram, a lung filter, and two blood samples were taken, where the change in the troponin level was monitored, and they also followed the return protocol for the sport [11].

During the lactate test, the initial resistance was set at 1.5 w/kg. The resulting value was rounded up. So, for a 63 kg competitor, the first step was 100 w. The resistance was increased every 3' by +20 w until it died out. On the two days before the test, the competitors did not do sports or perform high-intensity exercise to keep the starting lactate as low as possible.

III. Results

As we mentioned earlier, we tried to choose athletes who had

suffered from an infection, from whom we already had previous lactate test results and whose indicators were almost identical. We used three indicators during the study.

Chronic Training Load (CTL) is an exponentially-weighted average of the last 42 days of training. It reflects the training you have done over the last 3 months, but because the average is exponentially weighted, the workouts you did 15 days ago will impact your fitness more than the workouts you did 6 weeks ago.

Acute Training Load (ATL): by taking an exponentially weighted average of that stress from the past 7 days we are able to calculate your ATL (referred to as Fatigue in Training Peaks), or an estimate of your fatigue accounting for the workouts you have done in the past two weeks.

Training Stress Balance (TSB): a negative TSB indicates that you are carrying a lot of fatigue and are not on form. However, by tapering you can shed fatigue at a greater rate than you lose fitness and come into form (positive TSB) on the day that matters most important.

In Figs. 3-6, the orange curve represents the lactate values measured before the infection, while the other colors (blue, red, yellow) represent the new lactate values of the athletes who have undergone the infection. After the first wave, which mainly attacked the lungs and the older generation, we noticed that the body made much better use of the available O2. Acid tolerance improved. Compared to previous values, acidification occurred later. When the body was no longer able to break down lactate back into pyruvate, the degree of acidification was steeper. We can see this in Fig. 3.

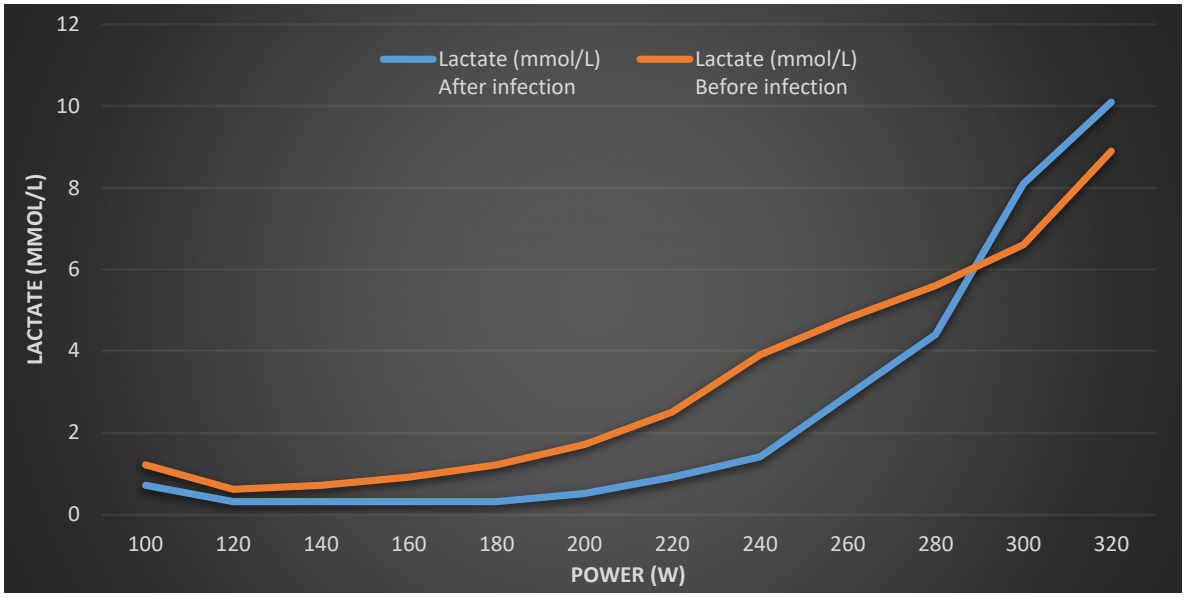

Fig. 3 Lactate profile after the 1st wave of SARS-CoV-2

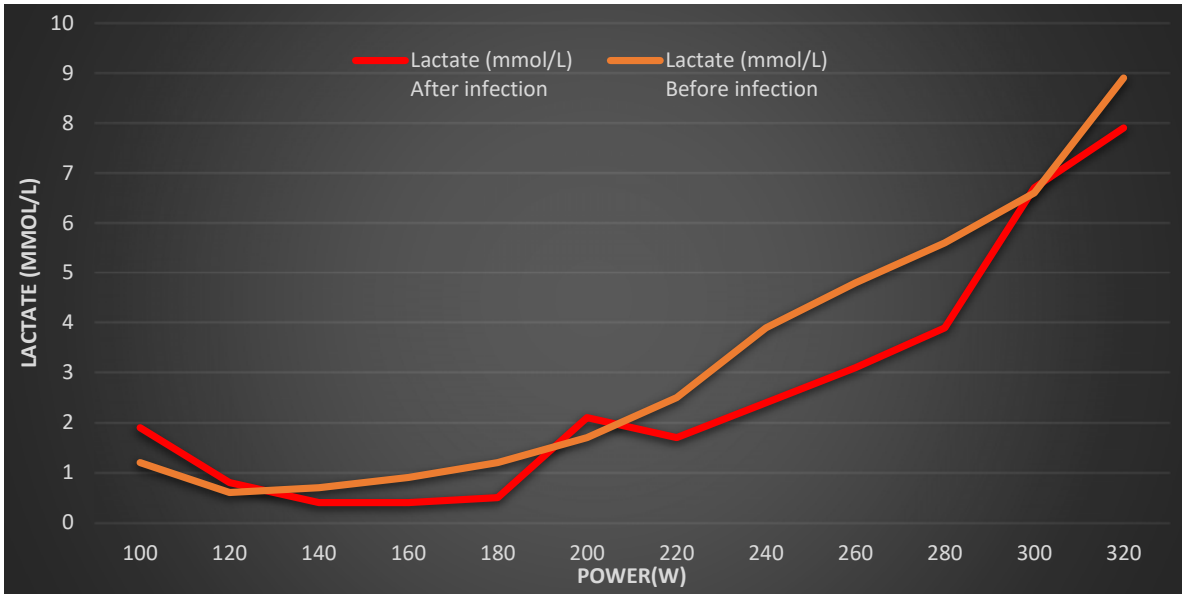

Fig. 4 Lactate profile after the 2nd wave of SARS-CoV-2

As can be seen in Fig. 3, acidification occurs later in those who have undergone the infection. This is marked with a blue line. Orange shows the results before infection. In the measurements performed after the second wave, we noticed that the lactate value measured at the end of the first step was higher compared to the value before the infection, which was followed by a substantial compensation. We can see this in Fig. 4.

As we can see in Fig. 4, the compensation following the initial high value is broken by an outlier. This spike was not observed in the measurements made after the first wave. Furthermore, what is still important to mention is that the level of acidification falls short of the steep gradient experienced after the first wave; just as the last lactate value does not reach the value before the infection.

After the 3rd wave, the lactate profile of the athletes changed again. We can see this in Fig. 5. In this wave, the virus mostly attacked the mitochondrial network and the young.

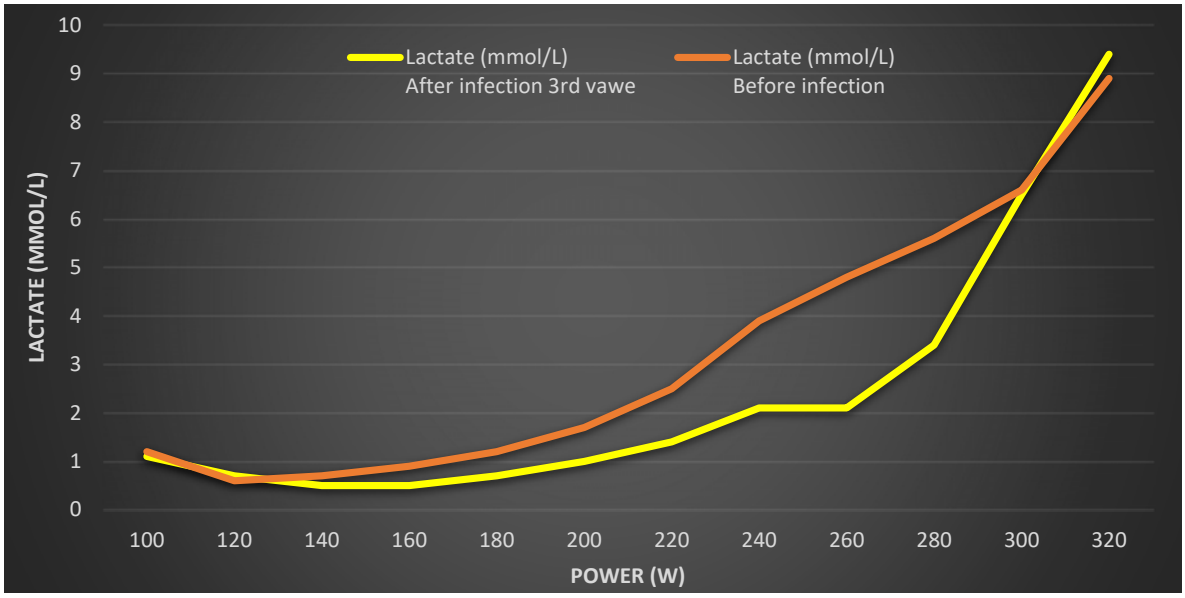

Fig. 5 Lactate profile after the 3rd wave of SARS-CoV-2

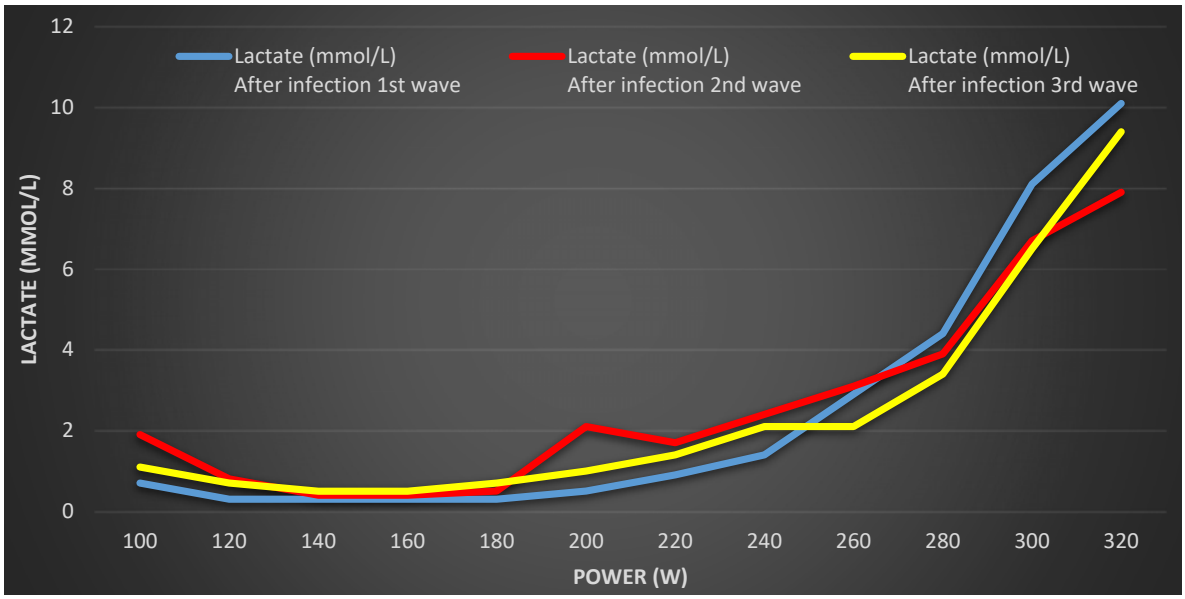

Fig. 6 Lactate values in 1st, 2nd and 3rd wave

As we can see in Fig. 5, the value seen at the end of the first step was almost identical to the value measured in the period before the COVID-SARS. Before the appearance of the virus, the value was 1.2, now it is 1.1. The small spike seen during wave 2 has also disappeared from the long low. The next significant difference is that the rate of acidification has also slowed down. However, the last measured value was much higher compared to the values measured during the 2nd wave. However, it falls short of the value measured during the first wave.

In Fig. 6, we can see the results of the measurements carried out during the first three waves of SARS-CoV-2.

The blue line indicates the lactate values measured after the first wave, the red line the second wave, and the yellow line the third wave. During the three waves, we obtained values different from the state before the infection. But this is not surprising, since the different waves attacked different parts of the human body. We can remember that during the first wave, the virus mainly attacked the lungs, while during the other waves, for example, the mitochondria were in the crosshairs.

During the measurements, we found that each wave had a specific image during the load tests. This also means that based on the lactate profile, we can say with great certainty which virus strain caused the athletes' illness.

Among the waves we examined, the values measured after the first and third were the most similar to each other. These had a long phase where the body was still able to convert lactate back into pyruvate. In wave 2, a spike appeared at this stage, followed by repeated compensation. The relatively high last value was also similar in the case of the first and third waves. These values exceeded the values measured before the infection. In the second wave, we measured the lowest value at the end of the tests. Furthermore, it is true for all waves that during the first 5-6 steps the lactate value remained lower than the values experienced before the infection. Table I contains the data used in the research.

TABLE I
LACTATE VALUES FROM BEFORE THE SARS-COV-2 INFECTION UNTIL THE 3RD WAVE

Power (W)	Lactate (mmol/L) After infection 1st wave	Lactate (mmol/L) After infection 2nd wave	Lactate (mmol/L) After infection 3rd wave	Lactate (mmol/L) Before infection
100	0.7	1.9	1.1	1.2
120	0.3	0.8	0.7	0.6
140	0.3	0.4	0.5	0.7
160	0.3	0.4	0.5	0.9
180	0.3	0.5	0.7	1.2
200	0.5	2.1	1	1.7
220	0.9	1.7	1.4	2.5
240	1.4	2.4	2.1	3.9
260	2.9	3.1	2.1	4.8
280	4.4	3.9	3.4	5.6
300	8.1	6.7	6.5	6.6
320	10.1	7.9	9.4	8.9

IV. CONCLUSION

After the first wave, which mainly attacked the respiratory system, we could observe that the competitors performed below the pre-infection values (Fig. 3). This is the result of the body learning to deal with the lack of oxygen that occurs during infection, so a supercompensation has occurred. It can also be thought of as the effect of a high-altitude training camp.

During the measurements made after the second wave, we noticed that a spike appeared in the lactate values. Before infection, this spike was not visible in the tempo zone. We see this in Fig. 4.

After the third wave, the spike that gives the uniqueness of the second wave disappeared in the lactate curve (Fig. 5). Here, our tempo zone has smoothed out, and we got better acidity values than before the infection.

In Fig. 6, we can see the relationship between the lactates measured in the first three waves. As can be seen in the figure, the curves do not overlap, they diverge nicely. Since the lactate values were also low in the endurance and tempo zones, they cannot be tested with statistical tests, since the significance test would not yield results either.

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