



Salvogene
SARS-CoV-2 Task Force:
Long-term damage caused by
Covid-19: Pathologists document
a battlefield in the heart muscle cells,
a gaping wound

Dear Premium Customers,

We have previously reported on the long-term damage that results from Covid-19, and examinations conducted by pathologists are providing an especially rich source of information confirming this. Some time ago, we wrote about a study at UKE in Hamburg, which is looking at the effect on various organs. In this Keynote, we will address the issue of long-term cardiovascular damage.

The realization that other organs in addition to the lungs are damaged by infection with the SARS-CoV-2 virus is now preoccupying American pathologists, and their investigations have shown that a patient's heart muscle cells are effectively taken apart.

In the context of our mission to optimize the health of our clients,

we place great emphasis on the role played by “inflammaging” and “silent inflammation”. By means of close monitoring, we aim to keep the levels of these two as low as possible and thus slow down the aging process. Our task here is considerably complicated by SARS-CoV-2. Our perspective on the virus is therefore always going to be different from that of doctors on the emergency ward. Among the considerations for our SARS-CoV-2 Task Force is what the heart vessels of an infected person will look like ten years down the line. Especially because SARS-CoV-2 is exceptionally vigorous in triggering inflammatory processes. This means that, for individuals who already have elevated levels of inflammaging and silent inflammation, the SARS-CoV-2 virus is highly effective in triggering and potentiating the tendency to inflammation. This runs totally counter to our strategy of maintaining good health.

In this area, we are also combining our knowledge with new findings from the University Hospital for Internal Medicine in Kiel, where a study of long-term cardiovascular effects has now been launched. The title of the study is COVIDOM, and it is set to run for 24 months.

SARS-CoV-2 accelerates the aging process of the internal organs

What we aim to achieve with our in-house AI program SAIP is the capacity to forecast what the heart vessels of infected persons will look like in five to ten years. This is roughly the timeframe for long-term effects that can already be clearly defined now. It has been established in principle that, in addition to the outwardly manifest consequences of the disease, the virus also accelerates the aging process of the internal organs without it becoming immediately apparent. It is therefore conceivable that a 30-year-old who becomes infected – perhaps even asymptotically – will have the lungs or heart of a 40-year-old in two years’ time or the lungs or heart of a 60-year-old in ten years’ time. This is a subject we are currently researching in detail. Ultimately, the decisive factor is not whether the disease has had severe symptoms, for example embolisms, but whether the infection has

changed the metabolism. This metabolic change can occur without any visible symptoms. We are working on the assumption that extensive damage to the blood vessels may occur and that this can lead to significant functional disorders in the internal organs over the long term.

Unfortunately, the pandemic has also brought routine check-ups to a standstill, partly because resources needed to be redirected, but also because many people were afraid that a visit to the doctor's consulting rooms carried the risk of infection. As the dynamics of the pandemic have abated to some extent – at least in Europe – patients with cardiovascular problems are now once again increasingly seeking check-ups. In some cases, their attending cardiologists are finding extensive damage in the course of examination, leading to a retrospective diagnosis of infection with asymptomatic progression.

Covid-19 is an endothelial disease

If we take a step back and look at everything we know about the pathway of infection in the lungs and about the virus in general – and there are new findings coming along every day – the overall picture shows that Covid-19 is actually an endothelial disease. Endothelium is the name given to the cells that form the walls of blood vessels. They are present everywhere in our body where there are blood vessels. The so-called endothelium is our largest organ. And it is evidently also one of the preferred targets of viruses, because these cells are highly susceptible to infection. Sadly, we have to abandon the idea that, in people who have survived an infection, the viruses have been eliminated. This is the fascinating thing about pathology: it reveals the extent to which the walls of the blood vessels have been damaged by the disease. It resembles a battlefield, a gaping wound.

Many endothelial cells die as a result of the infection, which in turn alerts the immune system. The response is highly radical. The damaged cells are first isolated, and then the affected tissue is destroyed. The immune system effectively carpet-bombs the

area with a view to rebuilding it afterwards. But when this happens on such a large scale, it causes pandemonium, a signal storm that stokes itself up even more. We think that this accelerates the aging process. The damage can be repaired after the disease, but it will cause a massive loss of reserves. We do not think that such severe turbulence can pass through the endothelial cells without leaving a trace. This in turn affects the organs which they supply with blood. With the signal chains having been stressed, the blood can become flooded with an inordinate amount of inflammatory substances.

Why regular monitoring of the health status is so important

The signaling substance profile of the so-called "cytokine storm" (on which we have carried regular reports) resembles cell aging speeded up. For example, there are substances such as TNF-alpha and interleukin 6, which we measure epigenetically and also by means of blood count in the course of our Covid-19 Immunization Program. Increased concentrations of both have already been found in the endothelial cells of affected patients, and this concentration is many times higher during an infection. The effect of aging by many years in a few short weeks is irreversible.

The test system employed in our Salvagene Premium Program has already detected this, which is why we were able to deduce that one of our clients had in fact been infected. Only by testing our standard parameters did it become clear that there must have been an infection in the past without a corresponding PCR or antibody test having shown positive results. This is a key component of our Covid-19 Immunization Program: on the one hand, we aim to get the client into the best physical shape to ward off a possible infection, but on the other hand, we also want to detect infections that have gone unnoticed and take immediate measures to prevent long-term damage that might quickly become irreparable. Our Covid-19 Immunization Program, which will in future also be a standard component of our Premium Program, will for this reason be monitoring health status much

more closely.

With SARS-CoV-2, the occurrence of a cytokine storm in cases of complications is more or less to be expected. However, it also happens with other infections. Why does the endothelium not age in these alternative scenarios? We think that this may be the wrong way of looking at it, because there is still too little research into long-term damage resulting from influenza or other viral infections. Far too few resources have been invested in this area.

The methylation testing of the cytokine receptors, which is part of our Covid-19 Immunization Program, allows us to gauge fully for the first time the extent to which cytokine storms have occurred previously in the individual client, as this is evident from the methylation of the different receptor families. And so it is very clear what strategy has to be adopted for the minimization of long-term damage to the heart. The aging rate, which is one of the most important monitoring factors in our AI program, must continue to be kept as low as possible for our clients.

In the next Keynote in our series on the long-term damage caused by Covid-19, we will be taking a look at the kidneys.

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