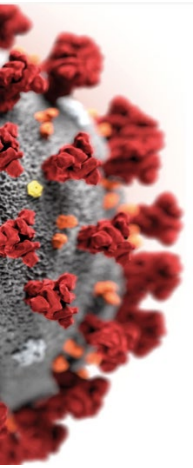

Combating COVID-19 webinar, October 23, 2020

Q&A

Question	Answer
High-throughout and high-content serology testing and research, *Sophia Hober, KTH*	
Were the people carrying antibodies tested again after some days (2d, 7d, 2weeks) to see if the immunity stays in the same people?	Yes, we tested all individuals that were shown to have antibodies against SARS-CoV-2 after four months and about 20% showed such low levels that they were counted as negative in our assay. However, we could still measure specific antibodies, but the levels were too close to the negative controls to be reported out as positive.
Hopefully we will soon have vaccines being rolled out. Besides standard monitoring of clinical outcomes, what research should be performed to monitor biological/molecular/cellular response to the different types of vaccines?	Beside careful monitoring of adverse effects, I think that there are four different analyses that should be made in order to confirm that a vaccine is effective: 1. Most importantly: analysis of the antibody production post vaccination. This should be done quite frequently to assess the antibody development. 2. Analysis of the effectiveness of the antibodies to neutralize the virus by inhibiting the cell entrance. 3. Evaluation of the T-cell response after vaccination, with a panel of carefully selected peptides. 4. If there is a fast decrease in antibody titers within, a further B-cell response study would be warranted for this specific individuals
Environmental biobanking and virus profiling, *Anna Székely, Uppsala University*	
Is drinking water in Sweden/in the world being tested on the virus?	When SARS-CoV-2 is inoculated to wastewater and drinking water, it can survive for short time. There are only 2 reported cases of successful virus cultivation from fecal samples. Here is a paper about infectivity by survival: https://pubs.acs.org/doi/10.1021/acs.estlett.0c00730#.X34x3cfzHSA.twitter
Are the clades we heard in the first talk picked up in the environmental samples? Different clades on surface vs. sewage?	We are conducting our first sequencing soon and then we will be able to answer these questions

It might be a bit of speculation. How likely is it to have a bigger virus load in the wastewater, which is then not filtered out in the wastewater treatment and more resistant particles end up in the environment which triggers mutation rate and can become more damaging to human or starts the human to animal transition?	Viruses are very efficiently filtered out during regular treatment process (10^4 decrease) However one our group members (Zeynep Cetecioglu) has reported persistence of the virus in fermenters. However, they are probably not infectious. So, in general in my opinion mutations happen mainly during replication and environmental triggered mutagenesis during viral particle stages is unlikely/unimportant.
The virus in the waste water is not infectious anymore, right?	It has been tested many times and according to my last literature search there are only 2 reports of infectious virus found in fecal samples
National COVID-19 data portal and data-driven efforts towards COVID-19, *Johan Rung, SciLifeLab Data Centre*	
Are the Swedish and the European portals being used in parallel by Swedish researchers? How exactly do they complement each other in terms of practical use?	The European COVID-19 data portal is the hub, presenting the COVID-19 data stored in the archives at the European Bioinformatics Institute (EBI). National nodes, such as the Swedish one, aggregate COVID-19 related data and services for the national users. They are most often not databases in their own respect, but may connect users to national data resources and services. One important part of this is helping Swedish users prepare data for submissions to the EBI archives, which should be the prioritized way to submit biological data. In the Swedish portal, we also make projects and data from Swedish universities findable, highlight data sharing aspects of new research, provide information about national testing, and so on. We also have some services, such as the SciLifeLab Data Repository, for data publishing for data where no EBI archives is suitable.
National biobanking for COVID-19 research, *Mia Phillipson, Uppsala University*	
How are severe cases categorized? Is it cases that require intensive care, cases that report severe symptoms, or can it be measured by viral load or similar?	On the level of respiratory support and care required

Host response to COVID-19 infection, *Petter Brodin, Karolinska Institutet*	
What are the treatment options available for MIS-C?	IVIG, Steroids and immunomodulatory biologics like IL1RA, Anti-TNF and anti-IL6R
Autoimmune responses such as Kawasaki disease are known as rare outcomes from some virus infections. There are also suggestions regarding risks of onset of such side effects from vaccines against viruses. How can you distinguish if these very unfortunate outcomes are a result of a virus infection and not a result of vaccination? Thinking of the hopefully soon-to-appear vaccines.	It is possible that MIS-C is induced by vaccination and we need to monitor for this and many other adverse reactions to vaccination once the programs are rolled out.
Cellular targets and biology of COVID-19 infection, *Emma Lundberg, KTH*	
How would you explain the change in subcellular localization? Is it because of a general bad state of the cells or change of the target signals in the proteins?	Since all these host proteins are known to interact with the virus, we believe that they are relocated through interactions with the viral proteins and in this way hi-jacked to optimize the cellular machinery to gain the virus. We have no evidence that target signals in the proteins change or that these are for example stress response proteins that signal a general bad cellular state.
Drug Discovery and studies of drug targets for COVID-19, *Kristian Sandberg, Uppsala University*	
Why is drug discovery important for university scientists when big pharma invest so much in drug development	Drug Discovery is a process of how to apply various technologies within different research areas to develop a prototype drug. After the drug discovery stage comes preclinical development when the final result of the drug discovery phase, the nomination of a candidate drug, is further developed into an experimental drug that can be tested in clinical studies. Big pharmaceutical companies are very skilled in how to drive projects through the preclinical development phase and to perform clinical studies in collaboration with clinicians in the health care sector. However, it is important to understand that health care and big pharma are **increasingly** dependent on academic research and spin-outs from universities as a source for new innovative drugs through discovery of prototype drugs (see *Kneller* DOI: 10.1038/*nrd3251 and Lipton*, DOI: 10.1016/*j.cell.2016.04.021*). A healthy academic drug discovery branch is therefore essential for a functional life science sector that can attract and work with the pharma industry to develop new therapies for patients.

The invisible patients: a broader approach to COVID-19 is needed in data collection and research, *Åsa Kristoferson Hedlund, Chair of Svenska Covidföreningen (the Swedish Covid organisation)*	
Have you seen an increased number of COVID infected people developing ME (kroniskt utmattningssyndrom)?	We have members that experience severe fatigue and some of them experience what could be post-exertional malaise. However, since longcovid patients experience fluctuations in their symptoms it is often hard to determine what is post exertional and what isn't. ME/CFS is a diagnosis of exclusion based on symptoms. Since we don't have enough data and knowledge about longcovid it would be irresponsible to give the longhaulers that diagnosis. We must first establish if longcovid is a disease in itself, and how to examine it and what tests to take. Without that knowledge we do not know what to exclude before going forward with an ME/CFS diagnosis. We are however concerned that some doctors don't seem to realize that. For example, in research we start to see organ damage, and decreases in oxygen saturation during a six minutes walking test, which is not typical for ME/CFS. But most of our members have not gotten those examinations, at the point where the doctors start talking about ME/CFS. However, we haven't seen any alarming increase in ME-CFS diagnoses yet.
How is longcovid currently distinguished from differentially diagnoses?	I would say that it is important to keep in mind ***when*** the patient got sick and ***how*** the disease started and developed. In most of our members' stories we see that the disease starts as a fever, similar to a flu or a stomach flu, when the infection rates were high, and then goes from there to multi systemic symptoms, often (but not always) via pulmonary symptoms. If there is a cardinal symptom I would say that it probably is the fluctuation of symptoms accompanied by at least some degree of fatigue (mental and physical). But breathing problems, cardiac distress and muscle weakness is also very common. Unfortunately, when it comes to testing, the common/old tests frequently come back "normal".