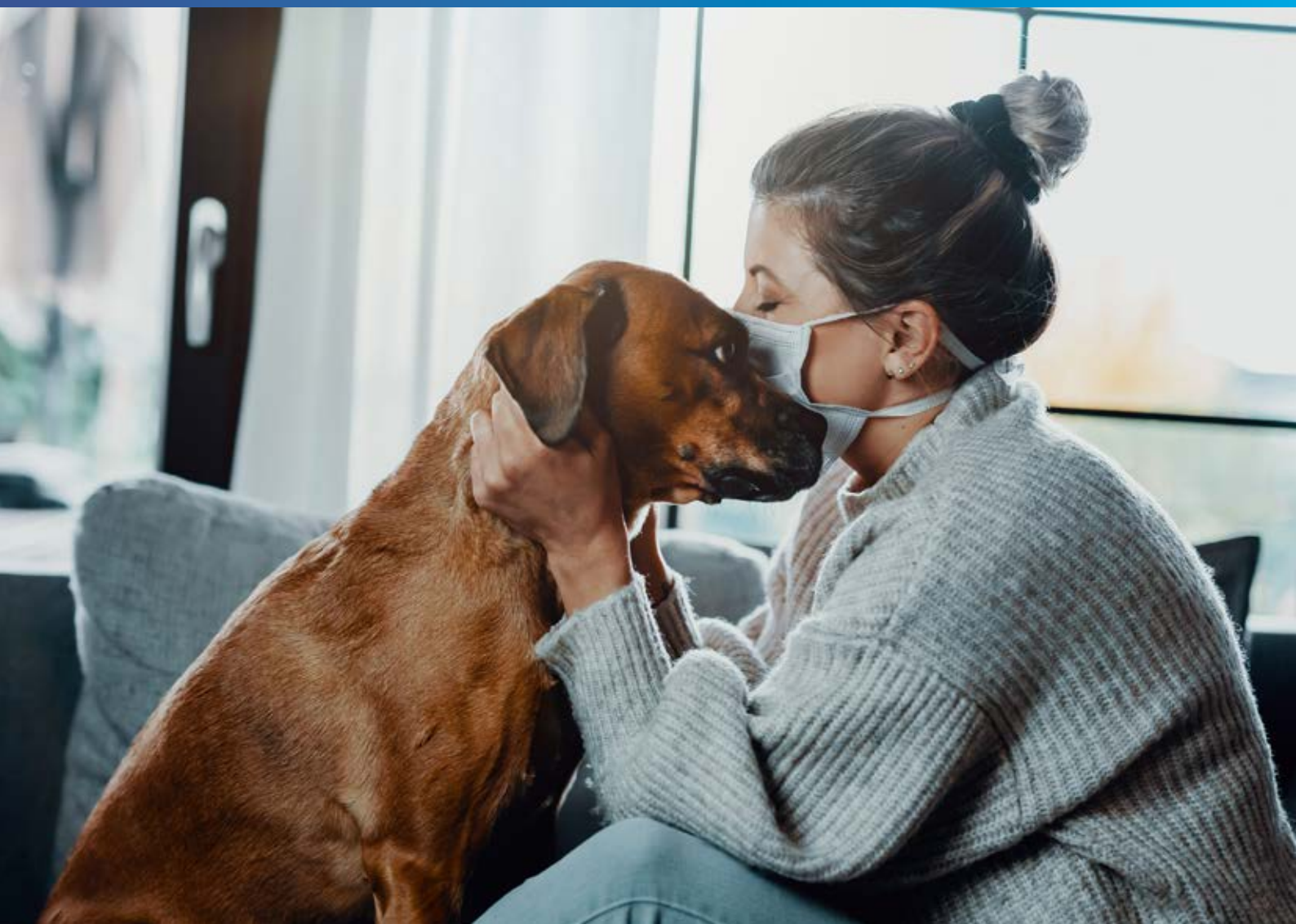
Swiss Re Institute

COVID-19 and likely long-term effects on survivors



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Executive summary

“Weird as hell,” is how Paul Garner, Professor of Infectious Diseases at the Liverpool School of Tropical Medicine, describes his personal experience of COVID-19.

The weirdness of the disease lies in the heterogeneity of its presentation. The ability to bind to the surface proteins of multiple cells allows the SARS-Cov-2 virus to infect multiple bodily organs in an unpredictable manner. This is both in terms of severity (we know some cohorts are more vulnerable, but variation between individuals is still considerable) and symptoms (dry cough being most common, but only one of many presentations).

The impact of the disease on survivor populations is likely to be equally heterogenous. The severity of the initial disease will be a significant factor. Intensive care can inflict both physical and mental trauma. Damaged organs will need time to heal or may even be permanently impaired. A severe bout of debilitating illness may leave a longer-term negative impact on mental health.

The health of the individual prior to contracting COVID-19 will affect later morbidity outcomes. Elderly cohorts, already exhibiting frailty, may become increasingly frail as a consequence of contracting the disease. Those with underlying conditions, including obesity, respiratory illness or cardiovascular conditions are likely to take longer in recovery. Lastly, the duration of COVID-19 is heterogenous. For most, it would appear, the illness is relatively brief. Some, even those with a mild initial illness, have reported multiple debilitating symptoms, the most common being fatigue, lasting many months. This paper reviews what we know so far about the effects of COVID-19 on survivor populations. At the time of writing, the disease has barely been with us nine months. Studies remain relatively small, both in number and scope, and to a limited extent we are forced back onto anecdote.

This publication is the latest in a series by Swiss Re Institute, dedicated to reviewing what we have learned about COVID-19 since the onset of the pandemic. What we can state from the outset of this review is that there will be no swift resolution of COVID-19, either in terms of cure or vaccine, or in terms of its eventual economic, social and health legacy. As and when we do control the pandemic, there will be many suffering negative long-term health consequences. We will discuss here what those, based on our current knowledge of the illness, are likely to be.

1 What really happens in COVID-19 infections?

COVID-19: a disease of the whole body

SARS-CoV-2, a novel coronavirus causing the disease COVID-19, enters cells by using its spike protein to bind to ACE2 enzymes on the surface of cells. While the virus may be new, this is the same mechanism that SARS-CoV, a virus that caused the SARS outbreak in 2002/3, used to enter cells (Hamming et al., 2004). ACE2 plays an essential role in the regulation of blood pressure across the body.

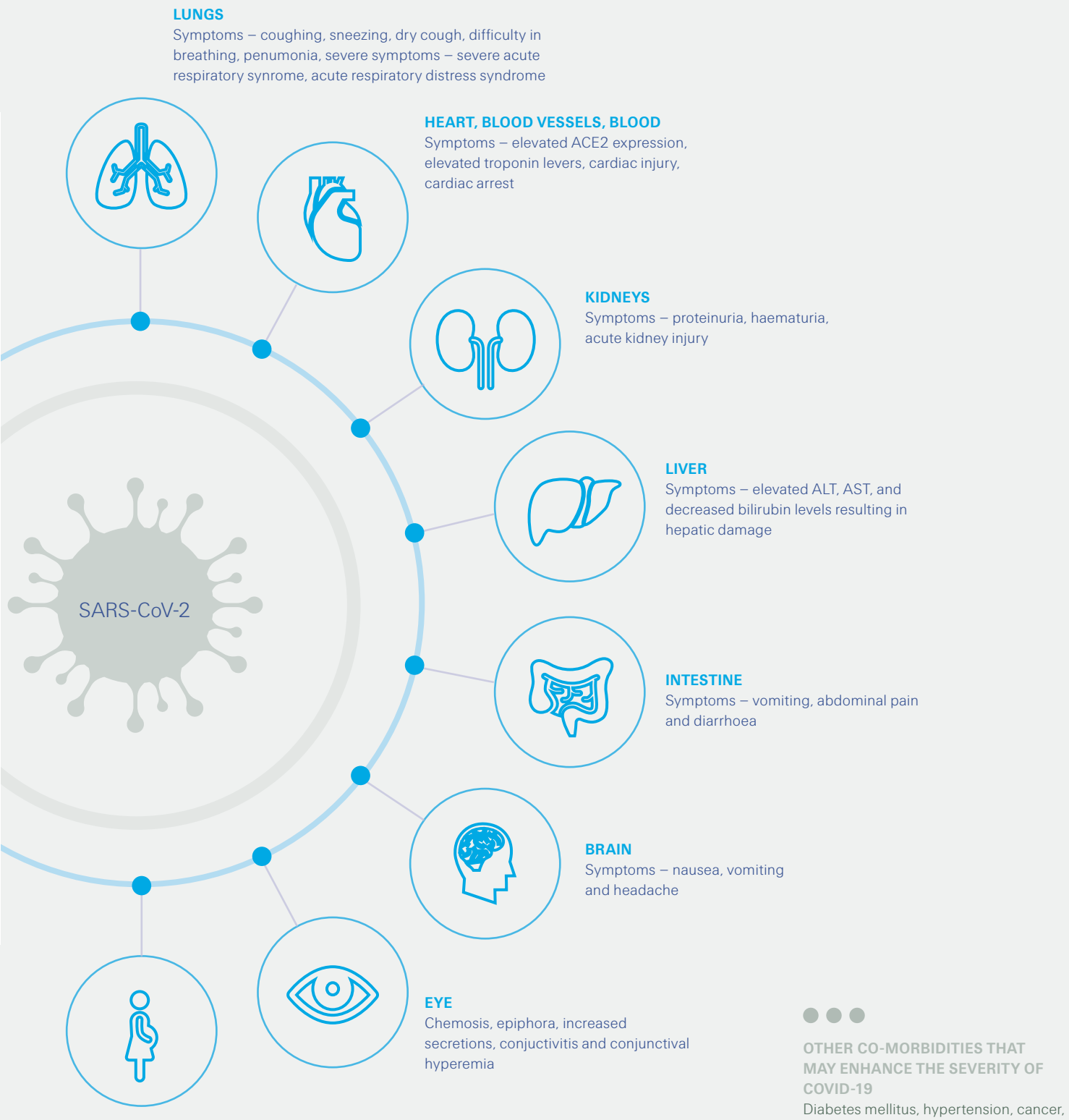
Cells with ACE2 enzymes are found in organs across the body. This helps to explain the systemic nature of COVID-19 (Renu et al., 2020). In every organ affected, the presence of SARS-CoV-2 will work to lower the organ's effectiveness, weaken it, and so produce the myriad of symptoms seen in patients with COVID-19. ACE2 enzymes are highly present in:

- **Arteries/veins** – Endothelial and smooth muscle cells that line the inside of small and large arteries and veins
- **Brain** – Olfactory lobe associated with sense of smell
- **Digestive system** – Endothelial and smooth muscle cells that line the stomach, small intestine and colon
- **Heart** – ACE2 receptors are more prevalent in heart muscle cells of patients with prior cardiovascular disease than of those without
- **Kidneys** – Particularly prevalent in the proximal tubules, which is the key area for re-absorbing water and maintaining fluid balance
- **Liver** – Lining cells in bile duct, which are involved in the regeneration of hepatocytes, and are the functional units of the liver
- **Lungs** – Lining cells in the alveoli of the lungs where gas exchange takes place
- **Nose/throat** – Lining cells of the mucosal surfaces

What really happens in COVID-19 infections?

In the early stages of COVID-19, the multi-organ effects of the infection can be seen in a variety of different symptoms:

Figure 1.2
SYMPTOMS IN VARIOUS ORGANS DUE TO SARS-COV-2



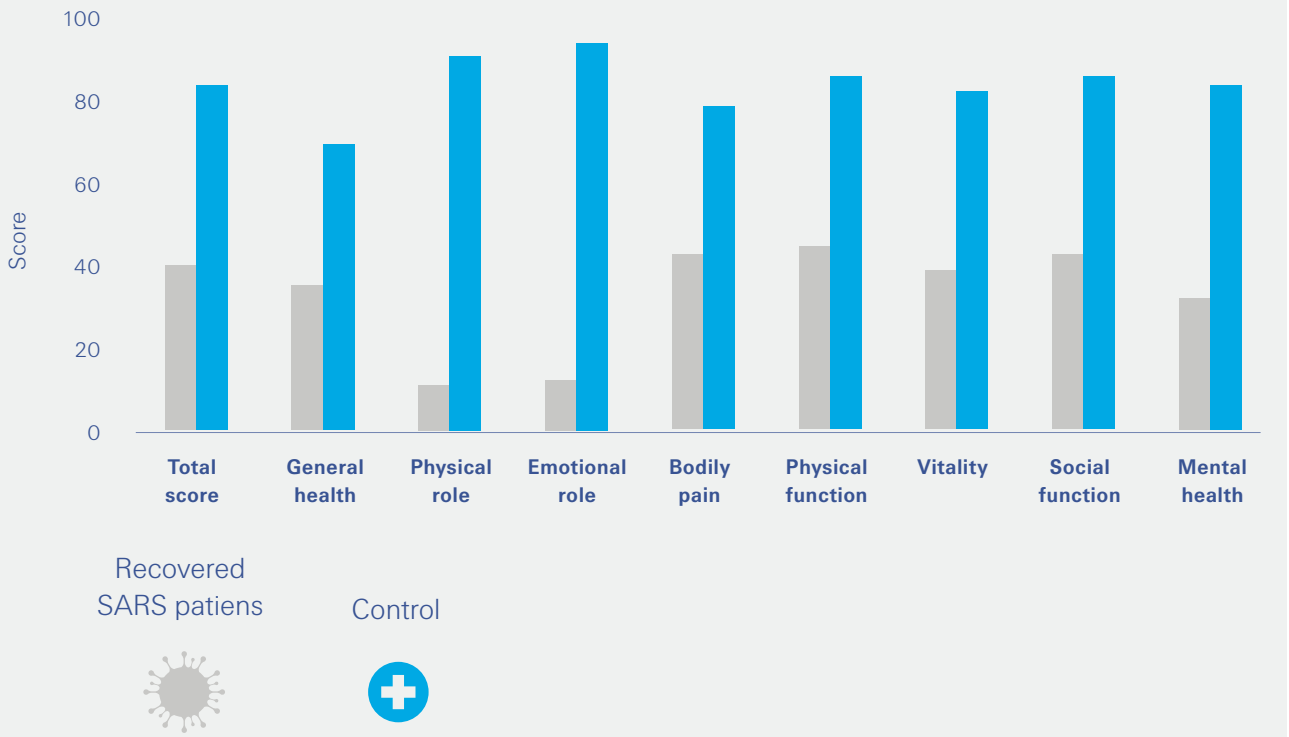
Source: Coronaviruses pathogenesis, co-morbidities and multi-organ damage

Long-term outcomes from SARS

One initial research group reported findings from 28 studies out of an initial extraction of 1 169 studies[2]. Of these, 18 focused on lung function, 6 on mental health, 5 on exercise tolerance and 5 on health-related quality of life. On the latter metric, those studies highlighted that quality of life was considerably reduced in the survivor population and remained below that recorded for those with chronic conditions in lung function, reduced exercise tolerance and psychological problems.

Figure 1.3

DIFFERENT HEALTH OUTCOMES BETWEEN SARS PATIENTS AND HEALTHY CONTROLS[3]



A group of 25 patients who had severe SARS infections were tracked over 12 years to ascertain differences in their health outcomes[3]. Approximately half had been re-admitted to hospital for a variety of reasons (including bone necrosis because of use of corticosteroids), two-thirds had had further lung infections and half had had a stroke. In addition, the survivor group appeared to have an altered metabolism to the control group as illustrated in Figure 1.3, in part as a result of treatment with methylprednisolone. As this group looked at severe infections of SARS, it is likely that the group contained a disproportionate number of patients who had severe symptoms, especially considering that SARS has a case fatality rate of approximately 10x that of COVID-19.

A further meta-analysis of 8 463 COVID-19 patients across 52 studies investigated liver function. This study analysed a broad segment of patient groups, excluding specific groups such as pregnant women; it therefore covers a wide range of disease severity. In 20% of patients, liver toxicity as a complication of COVID-19, was identified[4]. In addition, patients with severe COVID-19 symptoms were between 4 and 5 times more likely to have elevated levels of C-reactive protein, lactate dehydrogenase and the liver enzymes alanine aminotransferase and aspartate aminotransferase.

In the following sections of this report, we will explore in more depth the immediate and likely lasting impact of COVID-19 on different organ systems with the aim of developing a clearer understanding of how survivor populations will be affected and the possible impacts on future trends in mortality.

2 COVID-19 and the lungs

The first organs usually affected

The incubation period of SARS-CoV-2 is on average 5 days, with onset of symptoms developing within 5–7 days. During Stage 1 of the disease, the virus is localised to the nose and throat and hence the use of nasopharyngeal swabs to detect the presence of antigen. After several days, the prevalence of virus reduces and inflammation increases localised to the lungs, leading to the classic ground glass appearance on CT. In the latter part of Stage 2, patients will experience hypoxia. Approximately 10–15% of those that progressed to Stage 2 will advance to Stage 3 after another 5–7 days when the inflammation becomes uncontrolled and systemic[5].

Potentially the most valuable information on the likely long-term impact of COVID-19 on lung function comes from long-term studies of the effects of SARS. Studies show that 40% of patients were found to have reduced lung capacity after six months[1]. The largest of the longer term studies tracked a cohort of 71 healthcare workers over 15 years who contracted and survived the SARS-CoV virus at the Peking University People's Hospital[6]. The number of participants decreased over time, with 15–20 participants going through various examinations as a series of case studies. These studies found that the number of imaged areas of damage in the lungs reduced significantly from 2003 to 2004, and then remained broadly constant thereafter. In addition, the lung function at 15 years of those who had a normal CT after recovery was substantially better than those with abnormalities after recovery. Although lung capacity continued to remain impaired for those who were most affected, the recovery did lead to better lung function in terms of oxygen capacity and carbon dioxide exchange.

COVID-19 starts off as a respiratory disease, but there is increasing evidence that it is more than that.

3 COVID-19 and the kidneys

ACE2 receptors and proximal tubules

COVID-19 particularly affects individuals with diabetes and kidney disease, chronic conditions with widespread effects. Studies have compared ACE2 receptors in kidney cells from healthy donors to those from patients with diabetic kidney disease (DKD)[7].

ACE2 receptors were found to be predominantly connected to the proximal tube cells, responsible for the reabsorption of water and ions – a key function of the kidneys. The proximal tubule cells of patients with DKD had significantly higher prevalence of ACE2 receptors than those of healthy donors in the proximal tube cells. This indicates that patients with diabetes and kidney disease could be particularly susceptible to damage from COVID-19.

The study concluded that viral infection was distorting an already unbalanced immune response because of DKD. Biopsies and autopsies have confirmed that the proximal tubules are damaged in both diabetic and non-diabetic patients because of COVID-19. Cells have low blood oxygen (limiting their ability to perform their energy-intensive functions) and the formation of very small blood clots are visible in the affected areas[8].

Other symptoms of COVID-19, such as fever, diarrhoea and shock, may reduce blood flow to the kidneys and cause a higher requirement for water and ion transfer across the body[9]. All these symptoms work to exacerbate the severity of the DKD and other low-functioning kidney diseases. While DKD patients have diabetes by definition, other co-morbidities such as hypertension, chronic heart and liver disease are more likely to occur as a result of secondary kidney damage.

The degree and extent of kidney damage in COVID-19 is not found in every patient. However, when identified in hospitalised groups, it can vary significantly between patients. Mild clinical signs of disorder such abnormal urine analysis to conditions such as acute kidney injury (AKI) and as severe as renal failure.

So far, short-term studies suggest that there may be long-term kidney complications.

Acute Kidney Injury
Acute kidney injury (AKI) refers to an usually sudden deterioration in kidney function as evidenced by changes in the level of creatinine in the urine and reduction in urine output. The National Institute of Healthcare and Clinical Excellence (NICE) characterises AKI into different stages, described as I>III or mild>advanced, where the advanced category can see tripling of creatinine concentrations and cessation of urine production.

Prior studies have reported AKI in 5–15% of SARS and MERS patients. AKI is more likely to occur in COVID-19 patients who were older and who had a prior history of hypertension, congestive heart failure, diabetes mellitus and chronic kidney disease.

While a comparison to SARS and MERS can be useful, there have also been short-term studies on AKI in COVID-19. A study of 3 235 hospitalised patients in New York City found AKI in just under 50% of hospitalised patients[10]. AKI is a strong indicator of more severe illness, with 20% of AKI patients requiring renal replacement therapy and AKI occurring in almost 70% of those admitted to ICU. Of all patients requiring ICU, those that had developed AKI had mortality rates of 52% as compared to 9% for those without AKI.

Return to normal kidney function
Kidney functions recovery are most relevant for our study into longer term COVID-19 outcomes. Only 60% of COVID-19 sufferers with AKI saw their renal function recover to original levels at the point of discharge. However, the discharge point for COVID-19, as defined by ICU treatment protocols, is often dependent on lung function; whilst AKI recovery can take between 7 days to 3 months[11]. The results of this initial study in the US are also seen in an equivalent Chinese cohort, which found less than half of COVID-19 patients with fully recovered kidney function, within the 12 day follow-up period[12].

4 COVID-19 – heart, blood vessels & blood

COVID-19 and cardiovascular disease

Studies have identified that those with prior history of cardiovascular disease (CVD) are more likely to have a worse outcome with COVID-19, being 3 times more likely to have a severe COVID-19 infection and 11 times more likely to die[13].

Clinical investigations, using approaches such as MRI, have shown that 78% of patients had some level of cardiac involvement after COVID-19, and that 60% were still found to have cardiac-related symptoms over time[14]. Just like many of the long-term consequences, what is not known is the length and severity of these symptoms; clearly further investigations are warranted.

A review of 26 clinical studies of 11,685 patients investigated the cardiac signs in hospitalised patients. They found evidence that COVID-19 caused damage to the heart, with acute myocardial injury seen in approximately 20% of patients[15]. The review advanced a wide range of different possible explanations including the following:

- Hyperinflammation because of an immune response (cytokine storm)
- Hypoxia (low oxygen) because of lung damage, leading to heart muscle damage
- Reduction in the number of ACE2 receptors on cells, eventually leading to inflammation and higher blood pressures
- Hypercoagulability (excess blood clotting) leading to increased likelihood of capillary microthrombi
- Rupture of blood vessel constricting plaques leading to lack of blood supply and myocardial infarction

Blood & blood vessels

Whilst the initial target of the SARS-CoV-2 virus is the lining cells of the alveoli in the lungs, autopsies indicated that the virus also attacks the lining or endothelial cells of both veins and arteries[16]. The damage caused leads to further inflammation, hypoxia and increased coagulation, and increases the likelihood of multi-organ failure through interruption to the blood supply.

Damage to the lining cells in the blood vessels of the brain and the resulting lack of oxygen could be a key explanation for the high levels of delirium in older patients that are a presenting symptom of COVID-19. Any such damage to the brain is physiologically irreversible. However, as with stroke, some recovery of function is possible because of the brain's plasticity or ability to re-task functions to unaffected areas.

5 COVID-19 and the brain

Evidence of brain damage from MRI

While the cause and impacts of anosmia is currently unknown, there is the possibility that the virus is also moving through blood vessels, especially around the blood-brain barrier. Magnetic resonance imaging studies on those that had died from COVID-19 provided evidence of the endothelial damage mentioned in the previous section and damage to the olfactory bulbs[17].

Interestingly, these studies did not find any evidence of damage to the respiratory control centres in the brain, where ACE2 receptors are particularly abundant, and which, if damaged, would disrupt the automatic control of breathing. This had been put forward as a possible further complication of COVID-19 that would exacerbate the direct damage already caused to the lungs. So far, it seems that breathing difficulties reported by patients are due to the physiological impact on the lungs, as opposed to a neurological issue.

Other neurological symptoms such as headache and impaired consciousness are observed in a minority of COVID-19 patients, but these are also generic symptoms of viral infections and give little cause for alarm[18].

Mental health

At the peak of the containment measures, almost one-third of the global population was living under either lockdown or quarantine, and uncertainty remains as to the future strength of potential lockdowns[19]. Although this had a dramatic impact in reducing the spread of the virus, the effects of social isolation and longer-term economic recession are expected to have an impact on the short- and medium-term mental health of the population, in new and existing population segments[20]. Mental health remains an important topic for the insurance industry and experts from Swiss Re discussed the roles that insurance and reinsurance can play in this field in the publication on mental health and disability insurance.

Cross-sectional surveys in many countries have indicated much higher frequency of depression, anxiety and low self-rated wellbeing. Various studies have suggested that a variety of different groups could be particularly exposed to greater risk of mental ill-health, including healthcare workers, those with stressed financial situations, parents of school-aged children, and those likely to be at greater risk of both infection and severe symptoms[21].

The risk of episodes of poor mental health are not, however, exclusive in groups that have a prior medical history. Loss of employment, anxiety over infection and bereavement is likely to cause short-term mental health issues in those who have otherwise never reported a mental health condition before.

Demonstrable changes have been seen in mental health through the Avon Longitudinal Study of Parents and Children and Generation Scotland: Scottish Family Health Study cohorts[22]. These studies suggested that levels of anxiety had doubled to 24% across the population, but not depression. That said, both depression and anxiety were found to be more prevalent for younger people, women, those living

alone and those with pre-existing mental and physical health conditions. Interestingly, the studies did not report any increased risk for healthcare workers as compared to the general population.

The effects of previous epidemics on mental health has been well studied, in different countries and with different diseases. During the outbreak of Ebola in 2015, Sierra Leone reported 14 000 cases of the disease, leading to 3 900 deaths. Studies on the mental health of a cross-section of society showed that even a year after the infection's outbreak, 48% had at least one symptom of anxiety or depression, though only 6% would have met the criteria for a clinical diagnosis[23]. The clinical diagnosis rate was comparable to pre- and post-epidemic rates, suggesting that, despite the concerns over short-term effects, the long-term consequences may be less severe. The outcomes of this, as well as the impact on mental health more broadly, were discussed in another Swiss Re publication.

Similar results were seen in Asia after the swine influenza pandemic in 2009. A study in Japan reported "overwhelming fear" in the public. This was linked not only to the disease, but also the resultant economic and social disruption. In Hong Kong, 503 adults were surveyed by telephone seven times over a year during the same pandemic[24]. Some 19% reported panic, low mood or mental disturbance at the height of the pandemic, which decreased to 3% 10 months later.

Suicide and post-traumatic stress disorder

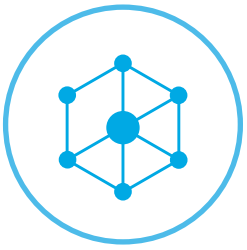
Independent increases in the rate of suicide in both Canada and USA have been predicted, as a result of both unemployment and quarantine. It has also been suggested that these increases could be mitigated by increased spending on mental healthcare, wage subsidies and work retraining, but these are long-term solutions[25]. While they may be of interest for development over the upcoming years, the impaired economic situation suggests that employment opportunities may be limited. Their prior analysis of historical trends identified that each 1% increase in unemployment rates was associated with a 1% increase in suicide rates.

The overwhelming nature of the COVID-19 pandemic for both those that experienced or witnessed its impact is expected to lead to an increased prevalence of post-traumatic stress disorder (PTSD). This could affect survivors, family members, healthcare professionals and even the general public. A systematic review of previous infectious disease outbreaks where the prevalence of PTSD in healthcare professionals was approximately 20%; and of these 40% were reporting high PTSD symptoms 3 years after the event[26]. An equivalent review for patients reported that almost 50% had PTSD at outset and that 50% of these continued to report symptoms over 2 years later.

Other studies show a similar message, with economic pressure leading to short-term stresses which could be aggravated[27]. Repeated stresses over a long period of time, especially with an excessive consumption of negative media could lead to PTSD developing. It is also too early to know the psychosocial effects of having suffered from COVID-19. Previous epidemics suggest an increased long-term risk of PTSD and mood disorders in those who were ill enough to require admission to hospital[28]. There is also the potential additional burden for social isolations as a result of distancing and quarantine measures. This topic is covered in greater depth in our paper on mental health, published at the height of the lockdown in Europe.

Post-traumatic stress disorder (PTSD) was the only mental health condition to have a sustained increase after previous epidemics finished. Throughout the first six months, reported cases of anxiety, stress and depression were on the rise.

6 COVID-19 – causing frailty in the population



Concept of frailty

As we have seen in previous sections, COVID-19 damages multiple organs through its interactions with the ACE2 receptors and knock-on effects. As such, the disease progressively degrades vital functional capacity, perhaps permanently in the most severe cases. This means that the disease is most destructive to those whose functional capacity was already most degraded whether through the effects of age or prior existing conditions. However, it is important to remember that some proportion, perhaps a very large proportion, of the population will have either mild symptoms or be asymptomatic.

This is particularly apparent in nursing/care homes where most residents will typically have a large number of prior existing conditions, and where up to 50% of excess deaths have been seen in many countries. It is therefore worthwhile to explore in more detail the assessment of vital functional capacity and the reverse concept, namely frailty.

Frailty measures how the accumulation of health problems makes older persons more vulnerable to new threats[29]. The Clinical Frailty Scale, for example, is a widely used tool for either healthcare professionals or patients/caregivers to rate individual state of health between 1 – very fit and 9 – terminally ill. It is through understanding frailty that individuals can be better protected and shielded against threats such as COVID-19.

While a coronavirus global pandemic has not previously been reported, there are lessons to learn from previous influenza pandemics for the long-term recovery of patients. Indeed, influenza recovery patterns were initially modelled to predict recovery in SARS, and have since been taken full circle to predict for COVID-19[30]. Other conditions that are known to cause long-term health issues and rapid loss of quality of life, such as sepsis, have also been considered[31].

Measures of frailty

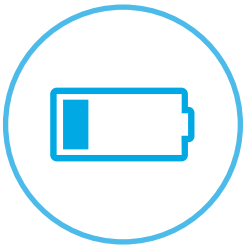
A further proposed phenotype of frailty which was associated with the presence of three of more of the following indicators[32]:

- Weight loss – unintentional loss of more than 5% of body weight in the previous year
- Weakness – grip strength in the lowest quintile adjusted for gender and BMI
- Poor endurance – based on self-reports of exhaustion during exercise testing
- Slowness – time to walk 15 feet in the lowest quintile adjusted for gender and height
- Low physical activity – self-reported calories expended per week in the lowest quintile adjusted for gender

A modified version of this phenotype was used for a study of frailty in the UK Biobank cohort. Participants were divided into three groups – 1) those who tested positive for COVID-19, 2) those who tested negative for COVID-19, and 3) those who had not been tested for COVID-19[33]. The study concluded that there was no evidence that frail individuals were more or less likely to become infected with COVID-19 after allowing for multi-morbidities. The study provided a marker and baseline for further research that should evaluate whether frail populations were more likely to die and have poorer health outcomes going forward.

Further factors underlying the heterogeneity between different countries will relate to the extent to which adequate personal protective equipment, widespread testing, zoning within care homes and limitations on discharge from hospitals limited the spread of the virus to and within care homes.

7 COVID-19 – Long haulers and post-viral fatigue



Long haul COVID-19 sufferers

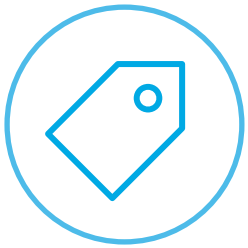
The cause of the longevity of the illness remains unclear. It could be a result of immunological response; viral reservoirs promoting a recurrence of the disease, as with herpes or dengue fever; or the result of longer-term organ damage.

Post-viral fatigue is a recognized condition, with many similarities to illnesses COVID-19 long-haulers report. Alternatively, COVID-19 may trigger the onset of myalgic encephalomyelitis (ME)/chronic fatigue syndrome (CFS). The biological pathways of ME/CFS are themselves not fully understood, with no diagnostic laboratory test and the illness is often not included in medical training courses[34].

One key factor that must be considered is the length of time that symptoms are being reported for, after the initial infection. While severity is not mentioned, the individuals investigated here still report secondary symptoms an average of 60.3 days after onset of the first COVID-19 symptom. While data on the likelihood of long-term symptoms is still being gathered, 60% of people experiencing fatigue of dyspnoea (shortness of breath) for 2 months is certainly a cause for concern. The multifaceted nature of chronic fatigue, with both physical and mental aspects, has also been reported as a potential feature for COVID-19[35].

For some, the disease continues long after the coughing and fever stops.

8 Size and characteristics of survivors



Different experiences across populations

In the previous sections we have highlighted the various ways in which COVID-19 can affect different organ systems. Whilst COVID-19 is a novel disease, it is clear that different individuals have different levels of cross-reacting immunity given prior exposures to other coronaviruses, and differing patterns of exposure and susceptibility given age, gender, occupation, place of residence, travel patterns and prior history of medical conditions.

In the UK, Public Health England (PHE) provides a daily summary of the number of individuals who are currently in hospital with COVID-19 and the number admitted each day. Until 23 June, just over 100 000 patients had been admitted to NHS England hospitals with COVID-19. A separate organisation, the Intensive Care National Audit & Research Centre (ICNARC), keeps track of the number of admissions to intensive care units (ICU). Its most recent report on 24 July reported that 13 308 patients had been admitted to ICU. Combining this information with data from the ONS on number of registered deaths leads us to the following summary of transitions:

- 8.4% of general population infected
- 2.1% of those infected (symptomatic and asymptomatic) admitted to hospital
- 10% of hospital admissions moved to ICU
- 30% COVID-19 mortality rate for those admitted to hospital
- 40% COVID-19 mortality rate for those admitted to ICU

This means that there are already a number of different survivor groups in the population whose experience of COVID-19 has been different and whose future longevity would be affected to different extents. Following from that NHS England survey, we can see that there are:

- 6 000 survivors from ICU
- 74 000 survivors from hospital who did not require ICU
- 4.6 million previously infected individuals who didn't require hospital – this group will be very heterogeneous, ranging from those with no symptoms to those with mild symptoms to those in nursing homes with serious symptoms but who were not admitted to hospital.

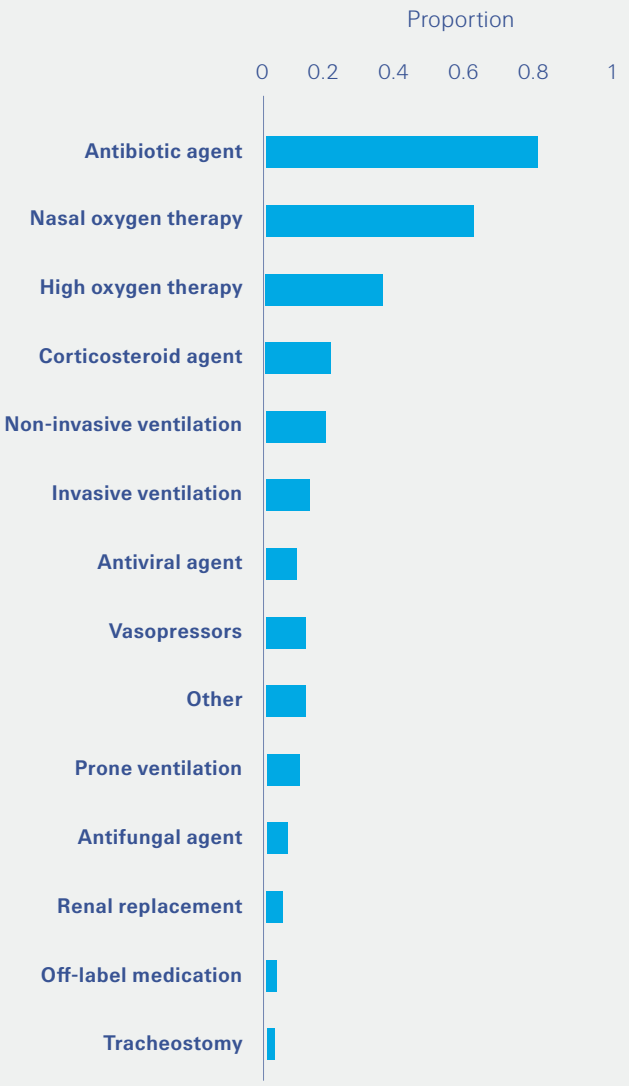
A retrospective cohort of 1 368 COVID-19 patients had been admitted to hospital in Spain[36]. Of these, 61 were re-admitted or 5% of the cohort, which is perhaps lower than expected given the mortality rates in hospital. Those who were re-admitted were more likely to have hypertension, to be immunocompromised, had shorter hospital stays and were more likely to have had a fever during the initial presentation.

A much larger international prospective cohort of hospitalised patients is being currently tracked through the International Severe Acute Respiratory Infection Consortium Clinical Characterisation (ISARIC 4C) Protocol. The cohort is predominantly based in the UK and the most recent report (8 June) from ISARIC tracks outcomes on 34 608 patients. A prior report on the UK cohort summarised the baseline features of 20 133 UK patients who had been admitted to hospital with co-morbidities[37].

The most recent ISARIC report also provided information on the treatments that had been received by patients whilst in hospital as set out in Figure 7.2. Eighty percent of patients were prescribed antibiotics and 15% of patients were prescribed corticosteroids.

SARS-CoV-2 infections could affect the progression and health outcomes of pre-existing medical conditions, in a similar way to the impact that influenza has on chronic respiratory diseases such as asthma and chronic obstructive pulmonary disease[38]. Moreover, quarantine measures and lockdown controls are likely to decrease activity levels which will exacerbate the effect of pre-existing medical conditions for COVID-19 survivors and mean that future survival rates are likely to deteriorate compared to current experience. While clinical approaches such as antibiotics, corticosteroids and oxygen treatment were commonly used, other techniques such as extracorporeal oxygenation, nitric oxide or covalence plasma therapy were far less common[37]. For future treatments, however, the knowledge gained around successful treatments could lead to a more successful treatment pathway for subsequent waves of infection. Potential towards the long term, novel drug treatments are likely to decrease infection rates, limit the use of invasive ventilation and ultimately improve outcomes.

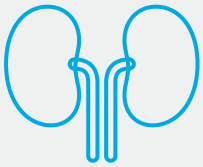
Figure 7.2
PREVALENCE OF TREATMENTS IN HOSPITAL ACROSS ISARIC COHORT AS OF 8 JUNE



Co-morbidities with cancer, diabetes and high blood pressure are common, all raising the risk of poor outcomes. But the survivor population is a broad one, as are their treatment pathways – there is still a lot to learn about the ideal treatment regime.

9 Long-term effects and life & health insurance

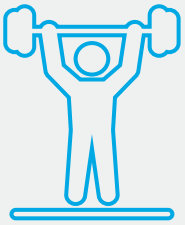
Overall, it is likely that there will be a number of individuals who will have some degree of impaired lung function. This is likely to disproportionately affect individuals who received invasive treatment in hospitals, such as ventilation, and those with significant comorbidities or older groups. This may lead to shortened lifespans and insurers could see excess claims in their life insurance portfolios and may need to consider underwriting loadings for new policies sold to survivors for some years to come after the pandemic.



Individuals with diabetic kidney disease will already be known to their insurer. Currently, there is not enough information available to discuss the implications for acute kidney injures on the insurance industry. Short-term studies suggest that there could be long-term damage to kidney function, but that this damage may not be severe enough to require dialysis. Further research and observations are needed, but from this it appears the impact on life insurers would likely be muted.

The significant implication here is that individuals with previously identified cardiovascular damage are currently at a greater risk. It is likely that these individuals would have identified as a high risk through the normal underwriting process so no material impact will be observed, but there could potentially be a significant segment of individuals with a mild cardiovascular condition that would not have raised any flags during the underwriting process whose conditions would be exacerbated by the virus. Life insurers should look for more information as the experience develops to assess strategies to address this risk.

There is a potential strong short-term risk for mental health related claims, especially in regions and markets most affected by COVID-19. There is also strong supporting evidence for a marked increase in PTSD after the pandemic subsides, which could have long-term consequences. Insurers should expand their criteria for individuals at risk of poor mental health and consider proactive or protective measures to prevent long-term illness.



One of the biggest unknowns is the long-term but non-specific impacts of the virus, which is hard to quantify. With the most significant spread of the virus only occurring in 2020, and the long-term effects only starting to be investigated, there is a lot that is currently unknown. However, some evidence is available, and studies have already identified a range of potential specific risk. The implications for the long-haul group, as loosely defined as it is, are significant. If there is recurrence of the initial viral infection, the repercussions for disease control and the infectiousness of individuals is significant, and there may be consequences for the eventual development of vaccines and boosters:

- A significant number of individuals may require extensive rehabilitation.
- Employee absenteeism as a result of post-COVID-19 conditions for the most severely affected may be significant and could generate insurance claims.

Conclusion

The initial wave of COVID-19 at the beginning of 2020 was dreadful. As of September 2020, the world approached the sombre milestone of 1 million COVID-19 deaths and almost 30 million confirmed cases. Those in recovery are estimated at 20 million worldwide.

For the survivor population, there is good news. The long-term repercussions of the then-novel coronaviruses SARS and MERS were not severe at a population level. Both had much higher mortality rates than COVID-19; but much less prevalence. Full recovery for most of the population, particularly younger and healthier cohorts, was by far the most likely outcome for survivors. We already know that most COVID-19 sufferers experience a mild bout of illness, followed by full recovery.

However, there are survivor groups, for whom COVID-19 will leave a more damaging legacy. While small in relative terms, the scale of the pandemic suggests we will still be talking of hundreds of thousands, perhaps millions of individuals. These groups include:

- Sufferers requiring intensive care in the first wave of the disease, particularly those who needed forced intubation, will require physical and mental rehabilitation.
- Organ damage, notably to the lungs, kidneys and heart, will require ongoing treatments, may inhibit quality of life, could increase vulnerability to further infections, and may even result in early deaths.
- There will be increased frailty in sufferers within elderly cohorts, requiring increased care, a decrease in life quality and a potential mortality impact.
- Mental health will be impacted by COVID-19. This includes those suffering a severe initial illness; those who mentally struggled to cope with lockdown or the socio-economic consequences of the pandemic; and those who experienced nervous conditions as a result of infection.
- Long-haul COVID-19, with a multiple of symptoms, most notably ME/CFS-like fatigue, are reported by many sufferers, with illness dragging into months. Studies are ongoing to ascertain the frequency and severity of long-haul COVID-19.

Finally, there remains the possibility that COVID-19 could be recurrent in sufferers. Malaria can lay dormant for months or even years, before individuals relapse into illness. SARS and MERS suggest this will not be the case; and we have no current evidence of recurrence, at least not over a longer time period. Long-haul COVID-19 sufferers report illness coming in waves.

Swiss Re Institute is committed over a series of publications to better understand COVID-19, and to make findings available to clients and other stakeholders. As the payee within most healthcare systems, insurers have a direct interest in the increased health cost burden of sufferers with the legacy of COVID-19. The long-term health effects of COVID-19 affects both our mortality and morbidity modelling; as well as our future underwriting criteria.

More particularly, our concern must lie with those whose long-term health has deteriorated as a result of COVID-19. They may be a minority, but still a substantial number, for some of whom the disease will have been life changing. Those individuals will need help with their physical and mental rehabilitation; and will require our compassion, understanding and empathy. As Hippocrates wrote, “cure sometimes, treat often, comfort always.“

Although likely to be few in number, there is still so much to learn about the implications for the “long haul” survivors. Swiss Re Institute is committed to publicising the latest developments in this ever-changing landscape.

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