Until there's a Covid vaccine, we need to focus on treating longer-term health consequences | Elizabeth Hartland

As Covid-19 infection numbers show a welcome downward trend in Melbourne and the city's residents look forward to some easing of restrictions, it's time to consider the longer-term health consequences of the pandemic.

More than 27,000 Australians – including some 20,000 Victorians – have been infected with the virus, with almost 900 deaths to date. Many countries are now in the grip of a second wave as the pandemic continues to take a toll on millions of lives around the globe – not only in terms of death, but also in the lingering, debilitating symptoms arising from severe, damaging inflammation.

The magnitude, severity and duration of Covid-19 suggests we will see a wave of longer-term health consequences emerge in survivors, and these will be many and varied; immunological, haematological, gastrointestinal and neurological.

Sars-Cov-2 infection not only causes lasting lung injury, but can also result in complications of the heart, liver, brain and kidney. Survivors report a disturbing array of ongoing symptoms and disabilities, including confusion, heart palpitations, extreme fatigue, joint pain, loss of taste or smell and shortness of breath. How many of these will translate to chronic ill health in the future? We simply don't know yet, and that's worrying.

Even before Covid-19, damaging inflammation was recognised as the root cause of death for many diseases, including influenza, sepsis, heart disease, cancer, diabetes and chronic kidney, liver and autoimmune diseases. However, emerging recognition of the chronic health impact of the Covid-19 pandemic brings renewed concern.

It is now clear that survivors will need long-term follow-up to document and understand the full course of recovery and range of complications. As the state most affected, a Victoria-led but nationally and internationally linked inflammation research initiative is the best chance to inform and improve their ongoing care.

Inflammation

Inflammation is the result of a complex and interconnected system of immune sentinels and messengers, called cytokines, that ideally act in a coordinated way to respond to injury and infection. When needed, this frontline system puts out a call to arms for the immune system to fight a virus or other threat, and once the crisis is over, launches a new wave to start repairing the damage. When these events are not coordinated, inflammation can become misdirected, overblown or chronic, leading to lasting, damaging effects on the body.

Corticosteroid treatment

In the case of Covid-19, the dangerous "cytokine storm" resulting from uncontrolled hyperinflammation (when too many messengers are sent in) leads

to widespread tissue damage, organ failure and a high risk of death. Alongside supportive respiratory intensive care, corticosteroids such as dexamethasone have been trialled to stem the rampant inflammation in severely affected people. Some studies have reported a 10% reduction in death with low-dose corticosteroids in critically ill patients.

While this is promising, corticosteroids have their limitations. These drugs have a broad anti-inflammatory effect which may also delay the body's ability to clear the virus.

What else can be done?

There is hope. Multiple research studies are under way at Hudson Institute of Medical Research and elsewhere to improve the way acute hyperinflammation is managed, including testing drugs with more targeted actions. These are designed to allow parts of the immune system to continue clearing the virus while blocking the most damaging inflammatory effects.

A fuller understanding of the genetic and environmental factors that predispose individuals to severe Covid-19 infection will help manage acute cases and longer-term complications. Advanced age, male gender and the existence of underlying chronic disease all heighten a person's risk of developing life-threatening Sars-Cov-2.

The scale of the pandemic has also unearthed some rare conditions, including a severe hyperinflammatory syndrome in children. Additionally, research published recently in Science showed that impairments in the body's frontline antiviral response, the interferons, can genetically predispose some people to life-threatening forms of Covid-19, while an autoimmune condition more common in men can produce antibodies that block interferon's protective actions. This knowledge opens the way for personalised therapies tailored to these patients, rather than a one-size-fits-all approach.

So how can we prevent further suffering from this and future viral pandemics? Overwhelmingly, we need access to a better range of therapies that target damaging inflammation. It's now time for more research focus and public discussion around treatments for patients who are still being affected by Covid-19, while vaccine and antiviral development are under way.

• Professor Elizabeth Hartland is director of the Hudson Institute of Medical Research in Melbourne, which houses one of the largest groups of inflammation researchers in Australia

Since you're here ...

... we have a small favour to ask. You've read in the last year. And you're not alone; millions are flocking to the Guardian for open, independent, quality news every day, and readers in 180 countries around the world now support us financially.

We believe everyone deserves access to information that's grounded in science and truth, and analysis rooted in authority and integrity. That's why we made a different choice: to keep our reporting open for all readers, regardless of where they live or what they can afford to pay.

The Guardian has no shareholders or billionaire owner, meaning our journalism