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### **Inquiry into Long COVID and Repeated COVID Infections**

As a general practitioner working in rural Australia, I am grateful to Hon Mark Butler MP for the decision to refer to the Committee this inquiry of great importance to patients and medical professionals.

Regarding the patient and medical professional experience in Australia of Long COVID and/or repeated COVID infections I offer the following perspectives.

An early account of a patient experience with Long COVID was described by Dr Paul Garner, professor of infectious diseases at Liverpool School of Tropical Medicine, and published as a blog in the British Medical Journal on May 5<sup>th</sup> 2020; after he himself developed COVID symptoms that were far more severe and prolonged than what he had anticipated<sup>1</sup>.

*He described; "The heaviness and malaise became worse, I had a tightness in the chest, and realised it could be nothing else..... My mind was a mess. My condition deteriorated. One afternoon I suddenly developed a tachycardia, tightness in the chest, and felt so unwell I thought I was dying. My mind became foggy. I tried to google fulminating myocarditis, but couldn't navigate the screen properly. There was nothing to do. I thought, if this is it so be it.*

*A few hours later I woke up, alive, and the tightness replaced by extreme fatigue. Every day, day after day. Sometimes I felt better and became optimistic; after all, the paralytic state had not recurred; but then the next day I felt as though someone had hit me around the head with a cricket bat. Staff at work criticised me for not being clear "make up your mind! Are you getting better or not?" I guess they were frightened too, but I really could not understand what was happening.*

*The illness went on and on. The symptoms changed, it was like an advent calendar, every day there was a surprise, something new. A muggy head; acutely painful calf; upset stomach; tinnitus; pins and needles; aching all over; breathlessness; dizziness; arthritis in my hands; weird sensation in the skin with synthetic materials. Gentle exercise or walking made me worse—I would feel absolutely dreadful the next day. I started talking to others. I found a marathon runner who had tried 8 km in her second week, which caused her to collapse with rigors and sleep for 24 hours. I spoke to others experiencing weird symptoms, which were often discounted by those around them as anxiety, making them doubt themselves.....Over the weeks, I have been touched by the people that have quietly stepped in to help me cope, appropriate, unobtrusive, timely. Family, friends, colleagues, and neighbours. Our local yoga studio's motto is 'a community building strength in mind, body and heart.' This love and support of gives us a direction for our future. And today the disease has lifted. For the first time, I do not feel awful. The aim of this piece is to get this message out: for some people the illness goes on for a few weeks. Symptoms come and go, are strange and frightening. The exhaustion is severe, real, and part of the illness. And we all need support and love from the community around us."*

Later a reflection paper titled 'How and why patients made Long Covid' was published in Social Science in Medicine<sup>2</sup>, further examining the very unique patient experiences with Long COVID, and describing the unfamiliar circumstances by which the entity of Long COVID was thrust from the patient experience to emerging formal recognition by the medical profession.

The authors reflected; *“Patients collectively made Long Covid – and cognate term ‘Long-haul Covid’ – in the first months of the pandemic.....Long Covid has a strong claim to be the first illness created through patients finding one another on Twitter: it moved from patients, through various media, to formal clinical and policy channels in just a few months. This initial mapping of Long Covid – by two patients with this illness – focuses on actors in the UK and USA and demonstrates how patients marshalled epistemic authority. Patient knowledge needs to be incorporated into how COVID-19 is conceptualised, researched, and treated.....While most patients initially had ‘mild’ COVID-19 and were not hospitalized, many experienced life-threatening symptoms as well as other traumatic events, often without healthcare support.*

*Thousands of patients collectively made visible heterogeneous and complexly unfolding symptoms: most were not commonly acknowledged within many healthcare and policy channels..... In a global health crisis, we need contributions made by those with wide ranges of expertise – including, crucially, patients..... Patient and lay contributions have often been ignored or underacknowledged by conventional actors, which has intensified patient suffering and societal inequalities. We need to learn from these episodes and ensure that patient contributions to the coronavirus pandemic are fully acknowledged and incorporated into policy making .....*

*Emollient descriptions of mild illness did not fit with people's often overwhelming experiences. In March, patients started sharing experiences on social media, drawing attention to possible Covid-related sequelae .....On 5 May, the British Medical Journal (BMJ) published Paul Garner's account of suffering seven weeks through a ‘roller coaster of ill health, extreme emotions and utter exhaustion’. Garner, an infectious diseases professor, also appeared in a feature, which, by 10 August, had been read over 1 million times. Garner's account travelled internationally, gathering a wider patient community around what he termed the Covid ‘long tail’. Garner reported patients took his account to medical appointments to provide evidence of the realness of their symptoms.....*

*Acknowledgement of prolonged Covid illness by the wider scientific community often occurred subsequent to patient efforts. In May, Van Kerkhove responded to a question about ‘people suffering from symptoms for many weeks’ stating, ‘Thus far there is very little evidence to suggest there are people who are persistently suffering from COVID-19’ .....patient-made material from informal channels was used as evidence prior to data being formally available from scientific studies.*

*On 9 July, Fauci stated ‘if you look anecdotally, there is no question that there are a considerable number of individuals who have a post-viral syndrome’. Fauci mentioned as evidence ‘chat groups that you just click on and see people who recovered that really do not get back to normal’.....Long Covid challenges common assumptions that were in place in the early pandemic and which often persisted despite patient testimony. In the making of Long Covid, conventional hierarchies of evidence, and normative routes for scientific dissemination were frequently disrupted. A patient-led survey released on a collective's website; the self-appellation of a community after a trucker hat; a single case study authored by a patient, and taken by others to clinical appointments; the circulation of a hashtag first used by a patient to refine the model of COVID-19 in published articles.....We end by calling for the work of more actors to be documented, and for patients’ ongoing contributions to be recognised and used to combat the suffering of multitudes.”*

Recognition of this debilitating and frustrating condition has prompted a number of reviews and research efforts internationally, notable the National Institutes for Health and Research review ‘Living with Covid’, authored by Dr Elaine Maxwell<sup>3</sup>, and the ‘COVID-19 rapid guideline: managing the long term effects of COVID-19’, Authored collaboratively by National Institute for Health and Care Excellence (NICE), the Scottish Intercollegiate Guidelines Network (SIGN) and the Royal College of General Practitioners (RCGP)<sup>4</sup>.

I would respectfully request that the Senate Committee for this inquiry consider in detail the findings, expert opinions, evidence reviews and the conclusions or recommendations of these and other comprehensive guidelines; and in consultation with Australian based experts with broad experience in treating patients with Long COVID, consider utilising this large body of existing work to facilitate the production of guidelines suitable for use in the Australian health care context.

Research into the pathophysiology of Long COVID is also of great interest and this condition unfortunately remains poorly understood. Proposals include a role for anti-idiotypic antibodies, discussed in this research article titled, *"A Possible Role for Anti-idiotypic Antibodies in SARS-CoV-2 Infection and Vaccination"*<sup>5</sup>. Another review article titled *"Long COVID- mechanisms, risk factors, and management"*<sup>6</sup> describes the multi organ involvement and the multitude of symptoms, and this research article titled *"The neuroinvasiveness, neurotropism, and neurovirulence of SARS-CoV-2"*<sup>7</sup> provides some insights into the neuroinflammatory effects of SARS-CoV-2 via spread across the blood brain barrier and subsequent neuroinflammation.

In the review by Crook et al<sup>6</sup>, some potential aetiologies for Long COVID are suggested, including the action of pro inflammatory cytokines causing impacts due to neuro toxicity and neuroinflammation, endothelial damage, damage to cardiomyocytes, thrombotic events and blood-brain barrier damage and dysregulation resulting in chronic inflammation within the brainstem, brain and other organs. These proposed impacts leading to fatigue, cognitive and mental health impacts, myocarditis, autonomic dysfunction and chronic fatigue respectively and the authors note that *"clinical characterization of patients with long covid is essential to provide appropriate treatment options. Gaining an understanding of why certain disease phenotypes arise in different individuals is an important piece of the puzzle. A review, which included perspectives from patients with long covid, suggested that the condition may actually be four different syndromes. Recognizing which patients belong to which subgroup of long covid, and understanding the pathophysiology, will be important in deciding the treatment they receive."*

A range of potential treatment options are also described, with the authors concluding that currently Long COVID remains enigmatic, and that *"greater understanding of the pathogenesis, risk factors, symptoms, and methods of treating long covid is required"*.

Another research article published in January 2022 titled *"Persistence of SARS CoV-2 S1 Protein in CD16+ Monocytes in Post-Acute Sequelae of COVID-19 (PASC) up to 15 Months Post-Infection"*<sup>8</sup> provides valuable insights into several immunological findings in Long COVID. The researchers examined T-cell and B-cell and monocyte subsets in severe COVID patients compared to Post-Acute Sequelae of COVID-19 (PASC), (this being the term used for Long COVID patients in that article). The researchers identified characteristic immune cell subset abnormalities that accompanied the unique cytokine/ chemokine profile, noting elevations in some monocyte subsets. The group examined the monocyte subsets for severe SARS-CoV-2 infection, and observed that whilst 36% of severe COVID-19 patients had SARS-CoV-2 RNA detected in the peripheral mononuclear cells, only 4% of the Long COVID subset of patients were SARS-CoV-2 RNA positive. The researchers concluded, *"In summary, the mechanism of PASC proposed in this report suggests that intermediate monocytes remain in circulation due to low CCL4 levels extending their time to differentiate leading to an accumulation of non-classical monocytes. Further, our data suggests that interruption of the CX3CR1/fractalkine pathway could be a potential therapeutic target to reduce the survival of S1-containing non-classical monocytes and the associated vascular inflammation previously discussed."*

*It is important to note that the S1 protein detected in these patients appears to be retained from prior infection or phagocytosis of infected cells undergoing apoptosis and is not the result of persistent viral replication. Full length sequencing of the five cases submitted for genomic analysis failed to identify any full-length sequence in the spike protein gene, or any other gene, that could account for the observed spike protein detected by proteomic analysis. In contrast, fragmented SARS-CoV-2 sequence was identified in all five of the cases. We have observed a pattern of high Ct value or negativity by PCR, accompanied by scant, fragmented viral sequence identified by whole viral genome sequencing over the past several months, a major shift from the low Ct value, full length viral sequences identified throughout most of 2020. The reasons for this shift are unclear, but as seen in these cases, it is unlikely these patients are producing any replication competent viral genomes, and are thus unlikely to transmit the infection. In contrast, the data reported here supports the hypothesis that*

*an immune response to persistent viral antigens, specifically the S1 fragment of the spike protein eliciting the PASC immune response previously published and marked by elevated inflammatory markers including IFN- $\gamma$ , IL-6, IL-10, and IL-2, among others."*

This unusual finding of an immune response to persisting viral antigens, in the absence of any S1 or other viral genome in patients with Long COVID and the shift in the years following 2020 to negative PCR findings and fragmented SARS-CoV-2 sequences on viral genome sequences might be explained by the elephant in the room. By this I refer to the possibility of Long COVID occurring as a consequence of COVID -19 vaccination, in at least some patients.

In regards to this possibility- that of potential overlap in aetiology between adverse events following vaccination and the Long COVID syndrome- the following points are relevant for the Committee consideration;

Prior to the vaccination roll-out, in March 2020, a Consensus Conference of Experts was convened; and following peer review by the Brighton Collaboration Network and by selected Expert Reviewers, a case definition for the assessment of Vaccine-associated Enhanced Disease (VAED) was authored and published<sup>9</sup>. VAED is defined as *"an illness that occurs in persons who receive a vaccine and who are subsequently infected with the pathogen that the vaccine is meant to protect against. This definition assumes previously antigen-naïve vaccine recipients, which can be assessed by determining seronegative status prior to vaccination, when feasible. The need for documentation of seronegativity prior to vaccination, which can be done retrospectively, is particularly relevant in Phase II-III clinical trials. In the context of such trials, the working group acknowledged the difficulty in distinguishing between vaccine failure and VAED. Thus, all cases of vaccine failure should be evaluated for VAED."*

The Guideline further described the factors to consider in evaluating for VAED:

*"A- Recognizing VAED in an individual patient is particularly challenging. VAED might be identified first as a vaccine failure. The clinical presentation may be variable within a spectrum of disease that ranges from mild to severe, life threatening, with or without long term sequelae, to fatal.*

*B- Identification of VAED requires the recognition of a clinical presentation that is different, atypical, modified or more severe in comparison to the natural or known (typical) disease presentation, or that occurs at a higher frequency from the control group or expected background rates in the specific target population.*

*No clinical presentation is pathognomonic for VAED."*

These are of particular note in the Australian context, in which a very high percentage of the population has been vaccinated, and large numbers who experienced technically 'vaccine failure', in that many thousands of patients contracted COVID-19 despite vaccination.

Some of the listed relevant clinical parameters for the assessment of VAED included: *"Fatigue, Myalgia/myositis/myonecrosis, Convulsions/seizures, Arthralgia/arthritis, Acute cardiac injury, Myocarditis, Multiorgan failure and Death"*

Notable also the breadth of clinical presentations considered under the case definition for VAED and variable clinical course including severity. The Guideline further details:

*"The following outcomes would be concerning for VAED or VAERD in a person with confirmed infection:*

*a. Death. This would be particularly concerning if death occurs in person without other risk factors for mortality (note phase I-II trials with selected healthy population) or if it occurs at higher rates than expected.*

*b. Hospitalization, including hospitalization above expected rates.*

*c. Worsening or clinical deterioration over time, particularly, although not exclusively, if differing from the anticipated natural course of the disease.*

*d. Prolonged clinical course compared to natural disease.*

*e. Complications of acute disease, new morbidities or new diagnoses subsequent to natural infection post-vaccination (for example higher rate of MIS-C or MIS-A)”*

Of note, all of these events have been reported following vaccination to the TGA adverse event database (DAEN) in quite astonishing numbers, particularly compared with traditional vaccines. Some of these severe events were even noted in the early clinical trials for the vaccines. For example, death due to cardiac arrest was reported in 7 of the patients in the BNT162b2 vaccine trial group (compared with 2 in the placebo group)<sup>10</sup> and several of the passive post marketing reports to the TGA of cardiac arrests have been considered by the Fatal Adverse Event Meeting of the TGA to be suspected as ‘causal’, including a cardiac arrest causing death in a 7 year old child. Several of the causality assessment reports as released by the TGA for FOI 3727 are included following this letter as Appendix 1. The possibility that such adverse events may have been a manifestation of VAED must be considered and evaluated by this Committee, particularly considering the high numbers of COVID-19 cases in Australia despite our high vaccination coverage; and the possibility of a vaccinated patient having a subclinical or undiagnosed COVID-19 infection after vaccination and then suffering from one of the many clinical presentations that characterise VAED.

Reports of deaths and serious adverse events on the TGA database, including those included as Appendix 1 must be reviewed, in my opinion, as part of this inquiry.

In addition, excess death rates for the population of more than 17%, without other explanation, requires urgent evaluation. If these deaths followed COVID-19 infection in a previously healthy and vaccinated individual then this provides very strong evidence for a risk of VAED following vaccination. If these deaths occurred in the absence of confirmed COVID-19 infection, but were unexpected deaths in vaccinated individuals, this also requires urgent review to investigate other underlying pathological events associated with the vaccine, for example sub clinical myocarditis resulting in cardiac injury and sudden death, or some other as yet unknown, pathological impact of the COVID-19 vaccines, but could also represent VAED following subclinical or undiagnosed COVID-19 disease.

**Given that Long COVID is, by definition, a ‘prolonged clinical course compared to natural disease’, each case of Long COVID, according to this international expert case definition, requires thorough evaluation to determine the possibility of VAED.**

There are in fact a number of potential aetiologies in addition to VAED for complex or prolonged multiorgan adverse events following the COVID-19 vaccines. For example, the synthetic nano lipid carriers utilised in the mRNA vaccines are not registered on the Australian Register of Therapeutic Goods or evaluated as an independent ingredient for therapeutic safety, and represent first use in the population for vaccination. For the BNT162b2 vaccine the non-clinical or animal study data demonstrated minimal metabolism of these lipid products over the investigated study period (48 hours) with no clear evidence of clearance from the body following vaccination<sup>11</sup>. These lipids also crossed the blood brain barrier and were found in a variety of other organs<sup>12</sup>. Given the robust immune response demonstrated in the non-clinical data; including cytokine release, increases in eosinophils and other white blood cells, lymphadenopathy and other findings; it is at least theoretically possibly that these products might, for example after crossing over the blood brain barrier and into neuro tissues, induce the very neuroimmune impacts discussed as the likely aetiology for Long COVID.

Incidentally the concern regarding the inability to effectively metabolise synthetic lipids is not new, noting for example details provided for the novel vaccine adjuvant in US Patent 3149036, September 15 1964<sup>13</sup>, “*The need therefore exists for an adjuvant which is relatively nontoxic to the host and*

*which will potentiate the antibody response to all antigens and additionally will maintain the titer over a long period of time thus endowing the host with a long period of immunity. In an attempt to satisfy the current needs, it had been proposed to use a mineral oil emulsion in which the antigen was incorporated in the aqueous phase. While this seemed to present some promise of providing an adjuvant type composition, it was found that it was not in fact a suitable solution because the mineral oil was not metabolized by the animal host and therefore could be a carcinogen."*

The potential inflammatory impacts of persisting S1 protein or protein fragments and the potential immunological responses to the nano lipids or nucleotide sequences contained in the vaccines have not been fully evaluated.

Any inquiry traversing the subject of the patient and healthcare provider experience of Long COVID and the research into the aetiology and best practice responses, without specifically evaluating the relationship this condition might have with COVID-19 vaccines; is disingenuous at best, and near negligently disempowering to these patients at worst.

Further publications discussing the syndrome of prolonged adverse events following vaccination include a case report from the medical author who uses the term 'Long post-COVID vaccination syndrome'<sup>14</sup>. The clinical features of this case mirror the described symptoms of Long COVID to an extent that cannot be overlooked by this Committee. Another published study details the experiences of long-term adverse events in 498 vaccinated physicians and dentists and reported long-term adverse events in 16% of this group of professionals<sup>15</sup>.

To demonstrate the impact of this disempowerment for patients suffering Long COVID type symptoms following vaccination, I respectfully request the committee members (and anyone reading this submission)- please now go back to the first page, and read again, but this time from the perspective of the writer describing adverse events following vaccination.

What is your first 'gut feeling' or emotional response when you read of Dr Gardner describing the chest tightness, heaviness, fatigue and fear that he was going to die, when viewed through the lens that he was describing his symptoms following vaccination?

What is your subconscious response when you read of patients coming up with their own name for what they have suffered, and bringing stories of other patients to show their doctors in order to appeal for belief in the legitimacy of their complaints?

When you read of patients finding each other by sharing their stories, this time sharing their side effects after vaccination, on Twitter or social media; what is your visceral response to such narratives?

I suspect that many reading this, if honestly reflecting, might recognise in themselves the presumptions, reactions and subconscious prejudices held.

How many of you have read such stories of adverse events after vaccination on online platforms or in the media and viewed such accounts with contempt or derision? How many have called out such suffering patients as 'anti vaxers' – by some logic that can only be considered bizarre given that these patients have of course all been vaccinated.

How many of you have heard stories or seen videos with patients describing unusual muscle tremors, seizure like movements or debilitating fatigue and thought this must be psychological or somehow otherwise feigned?

How many have read of stories in the media of a grieving parent or spouse who lost their loved ones after taking the vaccine, and if any empathy was felt at all, this tinged with the justification that this death was somehow 'for the greater good' of society?

These reactions are the very real consequence of unconscious bias.

The systemic discrimination against patients experiencing these debilitating symptoms must be frankly evaluated as a key priority area for the patient experience aspect of this inquiry. This inquiry must seek the perspectives of all patients with these as yet not fully explained symptoms, and must do so without bias or political agenda, and with an unimpeded focus on determining the truth through listening to all of these perspectives. The patient history has always been and remains the greatest source of medical information and of the highest value in solving any diagnostic dilemma.

Finally, I note that on page 41 of the NICE 'COVID-19 rapid guideline' is the recommendation to *"provide all information in accessible and age-appropriate formats so that people can understand and take part in decisions about their care. Follow relevant national guidance on communication, providing information (including different formats and languages) and shared decision making"*. The Medical Board of Australia Code of Conduct for Doctors in Australia<sup>16</sup> similarly states: *"Providing good patient care involves Recognising and respecting patients' rights to make their own decisions (3.1.5), Ensuring your personal views do not adversely affect the care of your patient or the referrals you make (3.2.14) That, Your decisions about patients' access to medical care must be free from bias and discrimination (3.4) and that Informed consent is a person's voluntary decision about medical care that is made with knowledge and understanding of the benefits and risks involved (4.5)"*.

No review of the patient and medical provider experience on Long COVID can be considered at all complete without an inquiry into the impact of mandatory vaccination policies and of the position statement from AHPRA and the National Boards on 9<sup>th</sup> March 2021.

It is my observation that mandatory vaccination policies have caused immense suffering to patients, have caused community division, resulted in seriously negative impacts on businesses and vulnerable employees, have perhaps irreparably fractured the doctor patient relationship and have likely directly resulted in serious harm or death in patients who experienced severe adverse events following vaccination.

The experiences of medical professionals facing patients who were suffering due to the impacts of vaccination mandates, against the threat of regulatory action for providing medical advice relevant to their individual patient circumstances, (should such advice be less favourable than the enthusiastic recommendation for vaccination in all patients); are also highly relevant to this inquiry.

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