IN THE HIGH COURT OF NEW ZEALAND
WELLINGTON REGISTRY

I TE KŌTI MATUA O AOTEAROA
TE WHANGANUI-A-TARA ROHE

CIV-2022-485-013

IN THE MATTER	of an application under the Judicial Review Procedure Act 2016
BETWEEN	**D**
	First Applicant – Eighth Applicants
AND	**THE MINISTER OF HEATLH**
	First Respondent
AND	**THE GROUP MANAGER OF THE NEW ZEALAND MEDICAL DEVICES SAFETY AUTHORITY (MEDSAFE)**
	Second Respondent

Continued overleaf

AFFIDAVIT OF SIMON BRADLEY BROWN IN SUPPORT OF NOTICE

Dated 18 January 2022

This document is filed by

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AND **THE COVID-19 RESPONSE MINISTER**

Third Respondent

Affidavit of Simon Bradley Brown

I, **Simon Bradley Brown**, retired research scientist, of Wellington, swear:

Introduction

1 I graduated from the University of Otago in 1983 with a BSc(hons) in Chemistry. I obtained a PhD in Protein Chemistry in 1989 from the University Biochemistry Group, Calgary, 1989. Between 1989-1993, I conducted Post Doctorate research at the Queensland Institute of Medical Research, Brisbane. Between 1994-1993, I conducted Post Doctorate research at the Queen's Medical Centre, Nottingham, United Kingdom. Between 1998-2011, I was a Senior Research Fellow and Principal Investigator (whereby I ran my own laboratory) within the MRC Centre for Inflammation Research, Queen's Medical Research Institute, University of Edinburgh, Scotland.

2 For 17 years between 1994 and 2011, I worked with Professor Sir John Savill who was the Divisional Head of the Inflammation Repair Group that I belonged to and was also Head of the College of Medicine and Veterinary Medicine and Chief Executive of the Medical Research Council (United Kingdom). I have worked alongside some of the United Kingdom's finest respiratory and clinician scientists.

3 Since 1989, my research focused on the role of innate immune cells in resolution of inflammation. If I was to describe my professional expertise, it would be as an innate immune cell biologist (i.e. an immunologist of the innate immune system). I have authored or co-authored numerous articles, the majority of which are in the field of, or related to, immunology. These papers are detailed in my Curriculum Vitae which is **annexed** to this affidavit and marked "**SBB-1**".

4 Although I retired in August 2011, I have a continuing interest in the field of immunology and I remain current with medical literature in the area. As a consequence, I have closely followed the unfolding COVID-19 public health crisis and the data and scientific studies that have emerged from it and, in particular, data and studies relating to the efficacy and safety of the mRNA vaccines and the impact these vaccines have, or potentially have, on the immune system.

5 For reasons recorded in this affidavit, I am deeply concerned by the Government's decision to permit (and, indeed, encourage) the vaccination of 5-11 year old children.[1] I believe healthy children are unlikely to benefit from the Pfizer vaccine and to the extent any small benefit is conferred it is easily outweighed by potential adverse events[2] including likely erosion of a child's immune system. There is not enough evidence to support the vaccination of otherwise healthy children with mRNA vaccines. This remains also the view of the Joint Committee on Vaccination and Immunisation (**JCVI**) (United Kingdom) which still recommends only those children in a clinical risk group or

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1 https://www.beehive.govt.nz/release/government-confirms-covid-19-vaccinations-protect-tamariki

2 https://www.express.co.uk/news/uk/1547050/covid-vaccine-JCVI-omicron-delta-myocarditis

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who are a household contact of someone who is immunosuppressed should consider getting vaccinated.[3] Finally, the increasing number of children testing positive for asymptomatic COVID-19 should be welcomed as this is the surest way for children to contribute towards achieving wider population herd immunity.

Expert witness code of conduct

6 I confirm that I have read the Code of Conduct for Expert Witnesses contained in Schedule 4 to the High Court Rules and that I agree to comply with it.[4] I confirm that I have considered all the material facts that I am aware of that might alter or detract from the opinions that I express, and that this evidence is within my area of expertise, except where I state otherwise.

Instructions

7 I have been asked to give my expert opinion on the Minister of Health's recent decision to grant provisional consent under the Medicines Act 1981 for the use of the Pfizer/BioNTech (Comirnaty) COVID-19 vaccine in 5-11 year old children.

8 I have been referred to a press-release issued by the Hon Chris Hipkins on 21 December 2021 in which he announced the Government's decision.[5] Minister Hipkins declared in that press release the following apparent rationale for the provisional consent:

(a) while COVID-19 generally has milder effects in children, with symptoms similar to a cold, **some** children become severely ill and require hospitalisation.

(b) if a child is infected with COVID-19 they **may** transmit the virus to other people. Immunising 5 to 11 year olds helps protect whānau members whose health makes them more vulnerable to COVID-19.

9 I address the following matters in this affidavit:

(a) Inadequate safety data to properly grant provisional consent

(b) The human immune system

(c) COVID-19 poses little risk to a child

(d) Antigenic imprinting

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3 https://www.gov.uk/government/publications/jcvi-update-on-advice-for-covid-19-vaccination-of-children-and-young-people/jcvi-statement-on-covid-19-vaccination-of-children-and-young-people-22-december-2021

4 https://www.legislation.govt.nz/regulation/public/2016/0225/latest/DLM6953324.html

5 https://www.beehive.govt.nz/release/government-confirms-covid-19-vaccinations-protect-tamariki

(e) Vaccine Associated Enhanced Disease

(f) Herd immunity

(g) Erosion of children's immunity

(h) Vaccine benefits have been misrepresented and the role of children as a disease vector exaggerated

(a) Inadequate safety data to properly grant provisional consent

10 It is not controversial to state that Pfizer's clinical trials have been wholly inadequate to establish the mid to long term safety of the vaccine. This fact is acknowledged in the risk management plan that Pfizer was required to submit to the New Zealand Government.[6] Table 1 of the risk management plan includes a list of missing information that would otherwise normally be mandatory for new medicines if proper clinical trials had been conducted – this includes no information on "Long-term safety data". Table 1 is below:

Table 1: List of important risks and missing information

Important identified risks	Anaphylaxis
Important potential risks	Vaccine-associated enhanced disease (VAED) including vaccine-associated enhanced respiratory disease (VAERD)
Missing information	Use in pregnancy and while breast feeding
	Use in immunocompromised patients
	Use in frail patients with co-morbidities (eg, chronic obstructive pulmonary disease [COPD], diabetes, chronic neurological disease, cardiovascular disorders)
	Use in patients with autoimmune or inflammatory disorders
	Interaction with other vaccines
	Long-term safety data

11 Therefore, the starting point is that the Minister's provisional consent has been given in circumstances where both the Government and Pfizer acknowledge that there is no long-term safety data available for the Pfizer vaccine.

12 There is no medium term data available either.

13 And, as I discuss later in this affidavit, the data that has become available indicates that the Pfizer vaccine, in general, is giving rise to significant numbers of adverse events. From my perspective as an immunologist, there are also indications of vaccine-associated enhanced disease and erosion of immune function. This is perhaps best evidenced by COVID-19 cytokine storm[7] and by increasing reports of negative vaccine efficacy (VE) in Europe, the United

6 https://www.medsafe.govt.nz/COVID-19/Comirnaty-RMP.pdf

7 https://dx.doi.org/10.1016%2Fj.cyto.2020.155151
https://www.science.org/cms/asset/f593856f-e928-4435-8851-91c597df33bd/pap.pdf

Kingdom[8], and Canada[9] that correlate with increasing prevalence of Omicron as found in a study from the United States of America.[10] Negative VE may now be starting to also manifest in increased hospitalisation and death rates for the vaccinated as compared to the non-vaccinated in Scotland.[11]

14 The original Pfizer clinical trial has been subject to significant criticism within the scientific community, including by the British Medical Journal (one of the most prestigious journals in the world) which identified serious questions over data integrity and regulatory oversight.[12] Pfizer's clinical trial involving 12-15 yr olds has also been harshly criticised.[13] Similar criticisms could be made with Pfizer's clinical trial data involving 5-11 yr olds published 06 January 2022.[14] This latter report is based on 1517 vaccinated children observed for a period of only one month. Not only is this not sufficiently long enough to observe pathophysiological events that take time to manifest but it is not appropriately "powered" to detect less common and rare adverse events that are now known to occur such as myocarditis and pericarditis.[15]

15 In the context of explaining the "Research and data" that the Government relied upon to grant provisional consent to give the vaccine to 5-11 years olds, the Ministry of Health explains on its website that:[16]

> For the trial among 5- to 11-year-olds, participants were randomised to either receive two doses of the vaccine 21 days apart, or a placebo. 1,517 children received the vaccine, and 751 children received the placebo. **Real-world safety data is emerging quickly as the international rollout continues, and the Ministry and Medsafe are monitoring this closely as it emerges.**
>
> [My emphasis]

16 In other words, the Government is relying upon data arising from actually vaccinating children to establish whether or not the vaccine is

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8 Refer UK Health Security Agency COVID-19 vaccine surveillance reports published on https://www.gov.uk/government/publications/covid-19-vaccine-weekly-surveillance-reports

9 https://doi.org/10.1101/2021.12.20.21267966
https://doi.org/10.1101/2021.12.30.21268565
https://publichealthscotland.scot/media/11076/22-01-12-covid19-winter_publication_report.pdf
https://doi.org/10.2807/1560-7917.ES.2021.26.50.2101147

10 https://doi.org/10.1101/2022.01.11.22269045

11 https://www.heraldscotland.com/news/19843315.covid-scotland-case-rates-lowest-unvaccinated-double-jabbed-elderly-drive-rise-hospital-admissions/

12 https://www.bmj.com/content/375/bmj.n2635. See also https://blogs.bmj.com/bmj/2021/01/04/peter-doshi-pfizer-and-modernas-95-effective-vaccines-we-need-more-details-and-the-raw-data/

13 https://www.sciencedirect.com/science/article/pii/S221475002100161X?via%3Dihub

14 https://www.nejm.org/doi/full/10.1056/NEJMoa2116298

15 https://doi.org/10.1038/s41591-021-01630-0

16 Covid: Vaccine vs infection myocarditis risk - Sebastian Rushworth M.D.
https://www.health.govt.nz/our-work/diseases-and-conditions/covid-19-novel-coronavirus/covid-19-vaccines/covid-19-vaccine-health-advice/covid-19-vaccine-and-children-information-parents-and-caregivers

safe. This is grossly irresponsible; particularly in circumstances where the Government expressly acknowledges that COVID-19 generally has mild symptoms in children "with symptoms similar to a cold".[17]

17 Dr Ashley Bloomfield, six days prior to the government's announcement that the vaccine would be rolled out to 5-11 year olds, issued an urgent warning to health care professionals regarding the risk of myocarditis/pericarditis.[18] He acknowledged recent reports of myocarditis/pericarditis following vaccination with the Pfizer Comirnaty vaccine and:

> . . . the need to reiterate the importance of timely assessment and management to prevent the serious consequences of myocarditis/pericarditis.

18 He pointed out that we do not know the true rate of occurrence:

> In New Zealand, the true incidence of vaccine-associated myocarditis is unknown as the onset of symptoms occurs in the first few days after vaccination and is potentially under-reported.

19 He concludes by saying

> Finally, could we ask you to cascade the requirements across your provider network and confirm in writing that local planning and clinical leadership is in place to guide a local response to prevent the serious consequences of undiagnosed or untreated myocarditis/pericarditis.

20 The catalyst for this letter appears to have been the death of 26 year old Rory Nairn by acute myocarditis likely caused by the Pfizer vaccine.[19]

(b) The human immune system

21 The human immune system can simplistically be split into two arms:

(a) the innate; and

(b) the acquired or adaptive.

22 The latter is associated with memory to invasive or harmful pathogens (microbes) including viruses such as the emerging SARS-CoV-2(**SC2**) betacoronavirus.

23 Innate immunity is non adaptive and is our first line of defence against any tissue injury and/or pathogenic infection. It does not initially

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17 Refer paragraph 8(a), above.

18 https://www.pinnaclepractices.co.nz/assets/Resource-files/20211215-DHB-CEOs-Myocarditis.pdf

19 https://www.newshub.co.nz/home/new-zealand/2021/12/coronavirus-vaccine-linked-death-prompts-reminder-letter-from-ministry-of-health-to-doctors-report.html

distinguish the nature of the injury. Rather, it treats all injury as infectious and foreign in origin until it determines otherwise.[20]

24 The innate immune system comprising phagocytic cells of myeloid origin uses primitive non-specific pathogen recognition systems to sense "foreign" (non-self) by working to kill and internalize any invading micro-organisms. One of these pathogen recognition systems is the use of innate low affinity natural IgM antibodies (**nIgMs**) which have specifically evolved to provide children maximum protection against pathogens until their own adaptive immune system becomes educated by infection to specific pathogens or vaccines.

25 Part of the acquired or adaptive response is to generate high affinity antibodies [eg IgG (humoral) and IgA (mucosal)] specific to a pathogen. High affinity antibodies, by definition, outcompete the versatility and utility of lower affinity nIgMs prevalent in children. This is a vital consideration in respect to the mass vaccination of children. The high affinity IgGs driven by a vaccine must be effective and productive to provide robust and durable immunity so as to not negate (by out competing) the proven benefits of innate low affinity nIgMs.

26 It is important to stress that innate nIgMs also function in adults and when any child or adult is exposed to a new virus these nIgMs will comprise a first line of defence as it takes time for both children and adults to generate and clonally select for "sterilising" antibodies and T cells. What a vaccine does is pre-educate the immune system in preparation for encountering that specific virus in which it is then able to rapidly amplify an inducible cellular and humoral (antibody) response.

27 As we get older, our immune system begins to deteriorate (immunosenescence) and our risk of opportunistic respiratory infections (**ORIs**) increases.

(c) COVID-19 poses little risk to a child

28 There is no reason for healthy young (<65 yo) individuals to fear COVID-19, no more so than for any other seasonal respiratory infection.

29 The Infection Fatality Rate (**IFR**) for COVID-19 for the overall population (i.e. all age groups) is approximately 0.4% ranging from 0.003% for those aged <19, 0.27% for 50-59 year olds and 5% in those >80.[21] These numbers are likely to be revised down further for the Omicron SC2 Variant of Concern (**VoC**)

30 To understand why children to date have not been at risk of COVID-19 we need to appreciate that a productive SC2 infection in adults (>18 yo) is dependent on viral spike protein binding to ACE-2

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20 https://www.nature.com/articles/nature01320

21 http://dx.doi.org/10.2471/BLT.20.265892
https://www.medrxiv.org/content/10.1101/2021.07.08.21260210v2

expressed by cells of the upper nasal and lower bronchial airways.[22] In contrast, children express ACE-2 at significantly lower levels thereby providing a youth-dependent barrier to infection.[23] An important corollary is the dramatically reduced severity of COVID-19 in individuals infected with Omicron. Compared to Delta the number hospitalized and those requiring significant medical intervention are greatly reduced. The reason being is the viral Spike protein on Omicron is so radically different from Delta let alone Wuhan strains that it no longer uses ACE-2 for viral entry and therefore it can't infect the bronchi and alveoli spaces of the lower respiratory tract.[24] Thus, Omicron may yet prove to be the ideal live attenuated virus as is used in other vaccines for invoking durable and robust "naturally acquired" immunity, all without the need for the use of a non-sterilising "vaccine".[25]

31 Two key points from the above paragraph require further explanation:

(a) Firstly, SC2 (excluding Omicron VoC) is both an upper respiratory infection and following progression to a lower lung infection, a viraemia (blood borne disease). The primary benefits of the Pfizer "vaccine" have always been to reduce the severity of the viraemic sequelae and not transmission. This explains why the vaccines fail to stop initial infection and transmission of the SC2 virus despite continued government advice suggesting otherwise. It is because the "vaccines" invoke a humoral response and not the more relevant mucosal immunity required to combat and sterilise ORIs.

In contrast, a humoral response yields high circulating levels of neutralizing antibodies that opsonize the viral particles and target them for deletion by the innate immune system. Thankfully the vast majority of infected individuals (>99.5%) do not see an upper respiratory infection progress to the lungs or a viraemia that ends up hospitalizing and killing its victim. For those under the age of 65, the IFR for Delta SC2 is <0.05% rising to 0.25% if we include all those >65 yo.[26] The IFR for children under the age of 18, esp <5, is less than 0.003% or 30 per 1 million[27]; ie comparable with flu.[28] Hospitalization and more serious consequences in those <18 yo (and indeed all age groups) appear to correlate strongly with multiple co-morbidities, specifically obesity and severe immunodeficiency

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22 https://doi.org/10.1038/s41467-020-19145-6

23 https://doi.org/10.1016/j.omtm.2020.05.013

24 https://www.nytimes.com/2021/12/31/health/covid-omicron-lung-cells.html and the following preprint study from Imperial College London (*The SARS-CoV-2 variant, Omicron, shows rapid replication in human primary nasal epithelial cultures and efficiently uses the endosomal route of entry*) which can be found at https://drive.google.com/file/d/1vam2PVMWvfRBczqs_uZbnUixGja1QPZD/view

25 The Pfizer vaccine is a "non-sterilising" vaccine in that it does not confer immunity on the recipient. As such, vaccinated people continue to be a vector for infection and transmission of the virus.

26 Refer footnote 21, above.

27 Refer footnote 21, above.

28 https://www.cdc.gov/flu/about/burden/2017-2018.htm

or immunosuppression.[29] Thus we can conclude healthy children do not need protecting from SC2, even less so for Omicron. We simply need to put in measures to protect those children who are vulnerable.

(b) Secondly, the use of vaccines that induce non-sterilising immunity is precisely why Delta came to dominate globally and why it will now be displaced by Omicron. The current "leaky" (non-sterilising) vaccines are driving the infections and pandemic. This is why we are now seeing a greater number of cases globally in the later rather than earlier waves even though far greater numbers are now vaccinated. We also have a published study encompassing 68 countries and 2947 counties in the United States of America that shows no correlation whatsoever between vaccination rates and any decreased incidence of COVID-19.[30] The UK Health Security Agency provides, perhaps, the world's most transparent and granular database to track COVID-19 infections through its vaccine surveillance reports. By tracking reports 39 (2021) through to 2 (2022) it has been widely observed that as Omicron dominates the infection rate correlates with vaccination rates for age cohorts >50.[31] In other words, being vaccinated appears to correlate with a greater possibility of being infected with Omicron.

Omicron is now the predominant SC2 in global circulation and while the Pfizer "vaccine" afforded some protection against Delta it does not with Omicron. Recent in vitro data confirms that while the booster is great for re-establishing circulating levels of neutralizing antibodies against Delta it barely registers a significant response for Omicron.[32] This makes sense. Why should Pfizer's vaccine, which targets the viral Spike protein from the Wuhan strain of SC2, be effective against Omicron when the viral Spike protein has radically changed both functionally and structurally? In addition we have real population studies confirming the booster does not protect against either infection or transmission.[33] Indeed, it may well be enhancing the virus (by way of vaccine-associated enhanced disease – discussed below) as has been observed with other prior attempts to make a corona vaccine.[34] How else might we explain the recent development of negative VE (discussed at paragraph 13, above)?

32 With Omicron now the global dominant strain the current mRNA vaccinations can only do immunological harm by embedding an

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29 https://doi.org/10.1038/s41591-021-01627-9
30 https://dx.doi.org/10.1007%2Fs10654-021-00808-7
31 https://www.gov.uk/government/publications/covid-19-vaccine-weekly-surveillance-reports
32 https://www.gla.ac.uk/media/Media_829360_smxx.pdf
33 https://doi.org/10.1016/S1473-3099(21)00648-4
https://spiral.imperial.ac.uk/handle/10044/1/93038
https://coronavirus.health.ny.gov/covid-19-breakthrough-data
34 https://doi.org/10.3389/fimmu.2021.640093
https://doi.org/10.1128/mbio.01987-21

immune response to viral spike protein that will soon likely be no longer recognisable. This is called antigenic imprinting which I discuss further below.

(d) Antigenic Imprinting

33 In my opinion, vaccinating 5-11 year olds with the Pfizer vaccine is likely to erode the fidelity and efficacy of a child's immune system by "locking" it into a predefined and limited repertoire of responses that are focussed on only one constituent of the SC2 viral proteome – the Spike protein of the "Wuhan" strain. This is what we call antigenic imprinting or, more colourfully, original antigenic sin (**OAS**).[35]

34 OAS is the "acquired" bias of the body's immune system to preferentially utilize immunological memory based on a previous infection/immunogen when a second slightly different version of that antigen is encountered. What this means in practical terms, is that as COVID-19 mutates, the body's immune response continues to respond to the original unmutated version of the virus. This can have serious consequences.

35 Antigenic imprinting would be fine if the SC2 virus was not so highly mutable and undergoing significant evolution as it adapts to its new found host (i.e. us) and undoubtedly to any other potential disease reservoir with which we can exchange the virus. It is salient to record that family pets, ie cats and dogs, are a natural reservoir for other endemic and seasonal human corona viruses while more recent evidence suggests the SC2 variant of concern, Omicron, arose from mice by reverse zoonosis.[36]

36 It is important to also mention that on first encountering a previously unknown virus we all have to rely on our innate immune system to fight the infection, adults and children. This is true for the unvaccinated and is also why I believe the vaccinated will end up paying a higher price as their innate nAbs are increasingly being rendered ineffective and unproductive by the mRNA vaccines which were developed to combat early strains of SC2 that are no longer in circulation.

37 In short, we should not be "locking" our immune system to a virus that has shown itself to be adept at throwing up VoCs that have evolved rapidly to escape vaccinal control. To do so creates significant risk of the virus becoming immune evading on the basis of antigenic imprinting. In addition, I believe there is a significant risk that the current mRNA vaccines may yet prove to enhance disease (via vaccine associated enhanced disease) further contributing to viral pathogenicity and immune erosion.

38 I note that Pfizer's CEO, Albert Bourla, has already acknowledged (before Omicron was discovered) that the Pfizer vaccine would

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35 https://doi.org/10.1016/j.clicom.2021.10.001
36 https://doi.org/10.1016/j.jgg.2021.12.003

inevitably fail due to the positive selective pressure in promoting vaccinal escape variants.[37]

39 OAS has been observed following SC2 infection due to prior exposure to more seasonal endemic strains of coronavirus and this has already been proposed as a mechanism to explain more severe outcomes including death in those infected.[38] In addition, antigenic imprinting was evident in sequential immunization of mice to either SC1 (SARS) or SC2 (COVID-19), two closely related coronaviruses belonging to the same subgroup.[39] They further show that cross-reactive antigen binding of antibodies between SC1 and SC2 is common but cross neutralization of live virus was rare.[40] We can therefore extrapolate and infer from these studies that prior exposure to an earlier variant of SC2 ***may*** lead to immune evasion by subsequent or later VoCs. There are those who argue against OAS as a problem, rather it merely presents a challenge for vaccinologists.[41] The problem here is those who argue OAS is not a problem made those comments prior to Omicron's arrival with its ability to evade both vaccinal and naturally acquired immunity while also ignoring the continued selective pressure for vaccinal escape variants due to mass vaccination in the midst of a global pandemic.

40 Vaccination against the Wuhan strain of SC2 does not appear to elicit higher levels of OAS compared to infection.[42] However, it would not come as a surprise to find evidence of OAS for both initial (Wuhan) and subsequent variants of SC2 given the cross-reactivity of antibodies against SC2 and other β coronaviruses.[43] Thus it remains a distinct possibility that OAS could trigger immune evasion of emerging variants in those who have not only been infected by but also vaccinated against former versions of the pathogen.[44] The omicron VoC may not be a "natural" vaccinal escape variant despite widespread changes predominantly affecting the Spike protein but omicron is most certainly immune evading.

41 My key concern with mass vaccination in the middle of a global pandemic is the vaccines are placing significant and continuous selective pressure on the virus to promote escape variants. Thus, not

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37 https://www.insider.com/pfizer-ceo-vaccine-resistant-coronavius-variant-likely-2021-8

38 https://doi.org/10.1038/s41467-021-23977-1
https://www.biorxiv.org/content/10.1101/2021.05.21.445201v2
https://www.cell.com/cell/fulltext/S0092-8674(21)00160-4?_returnURL=https%3A%2F%2Flinkinghub.elsevier.com%2Fretrieve%2Fpii%2FS0092867421001604%3Fshowall%3Dtrue
https://doi.org/10.1101/2021.05.04.21256571

39 https://doi.org/10.1101/2020.10.14.339465

40 https://doi.org/10.1016/j.celrep.2020.107725

41 https://www.statnews.com/2021/04/16/next-generation-covid-19-vaccines-are-supposed-to-be-better-some-experts-worry-they-could-be-worse/
https://doi.org/10.1016/j.clicom.2021.10.001

42 https://doi.org/10.1101/2021.09.30.21264363

43 https://doi.org/10.1126/science.abd4250

44 https://www.statnews.com/2021/04/16/next-generation-covid-19-vaccines-are-supposed-to-be-better-some-experts-worry-they-could-be-worse/
https://doi.org/10.1016/j.clicom.2021.10.001
https://doi.org/10.1017/ice.2021.199

only do prior endemic strains of corona exert antigenic imprinting but so will the vaccine. I therefore found it vexing that in response to my Official Information Request regarding "leaky" vaccines, Medsafe replied: "The stated concerns have not been identified through clinical trials or through the large number of real-world studies of the Pfizer vaccine, including before and after formal approval, at a sufficiently robust level that would trigger an assessment from COVID-19 Vaccine Technical Advisory Group".[45] Not only do I consider the response false but it would seem apparent that Medsafe is not looking to front foot potential (and predictable) problems with the Pfizer vaccine. As I also raised with MedSafe, this includes the risk of driving an adverse autoimmune response as a consequence of repeated vaccination in order to maintain semi-persistent high levels of circulating antibodies.

42 We may not need to wait long to find out whether OAS is a problem with next generation formulations of the mRNA vaccines. Apparently Moderna and Pfizer are working on mRNA vaccines that target spike protein of VoCs other than Wuhan including Omicron and Beta.[46] The beta variant trial is now over 12 months in gestation yet I am unaware of any Phase 1 clinical trial data having as yet been made public. For the sake of public and child safety it would be wise to await these results in order to obviate any concerns regarding OAS negatively impacting the ability of a vaccinated child's immune system to mount a proper response to future VoCs. Another serious consideration, also summarily dismissed by Medsafe, is vaccine associated enhanced disease.

(e) Vaccine Associated Enhanced Disease

43 Vaccine Associated Enhanced Disease **(VAED)** is another potentially serious consequence of rolling out a "leaky vaccine" to a large portion of the population.[47] The phenomenon of VAED is an event that occurs with some viruses, where pre-existing non-neutralizing or sub-neutralizing antibodies to viral surface proteins that were generated during a previous infection can promote the subsequent entry of related viruses or VoCs into the cell. VAED can lead to increased pathogenicity by intensifying the inflammatory process during a secondary infection with any antigenic-related virus.[48] Most important, VAED has been observed in prior attempts to develop coronaviral vaccines[49] and may have been seen in patients diagnosed with COVID-19.[50]

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45 https://fyi.org.nz/request/16617-cv-tag-discussion-papers-regarding-adverse-effects-of-using-a-leaky-covid-vaccine#incoming-64244

46 https://dx.doi.org/10.1016%2Fj.eng.2021.04.005

47 https://doi.org/10.1038/s41564-020-00789-5
https://dx.doi.org/10.1016%2Fbs.ai.2021.08.003

48 https://doi.org/10.1080/21645515.2020.1796425
https://www.nature.com/articles/s41586-020-2538-8

49 https://doi.org/10.3389/fimmu.2021.640093
https://link.springer.com/article/10.1007/s40259-021-00495-6#ref-CR11
https://link.springer.com/article/10.1007/s40259-021-00495-6#ref-CR12

50 Refer footnote 9, above.

44 Table 1 of the Pfizer risk management plan (referred to at para 10, above) acknowledges the risks of VAED arising from its vaccine.[51] Table 3 of the same risk management plan discusses the evidence for linking the risk to the Pfizer vaccine in the following terms:

> VAED is considered a potential risk because it has not been seen in human studies with this or other COVID-19 vaccines being studied. It has not been seen in vaccine studies in animal models of the SARS-CoV-2 virus either. However, in selected vaccine studies in animal models as well as in some laboratory studies in animal cells infected with 2 other related coronaviruses (SARS- CoV-1 and MERS-CoV), abnormalities in immune responses or cellular responses indicative of VAED were observed. Because of this, VAED is considered a potential risk. In the past, there have been other examples of particularly respiratory viruses where VAED has been observed. For example, some children who received an inactivated respiratory syncytial virus vaccine (a different type of virus), had worse signs of disease when they were subsequently infected with respiratory syncytial virus.

45 VAED is thought to occur by several mechanisms where the immune response is not fully protective and actually either causes the body to have an inflammatory reaction due to the type of immune response with specific types of T-cells, or the body does not produce enough strong antibodies to prevent SC2 infection of cells or produces weak antibodies that actually bind to the virus and help it to enter cells more easily, leading to worse signs of disease.

46 While VAED has not yet been demonstrated definitively to exist with these vaccines we cannot continue to ignore data out of Israel, the United Kingdom, the European Union and North America that clearly shows both Delta and Omicron VoCs have a growing propensity to preferentially spread within the vaccinated and boostered.[52] This is a canary in the immunological coal mine and needs to be addressed and explained before we subject children to untold potential harm and not just from widely reported adverse events but also auto/immune dysregulation which may not become apparent for some time and is a potential ticking timebomb.

47 As noted, the vaccine "locks" our immune response to deal with the original version of the SC2 virus. However, the virus keeps evolving to evade vaccinal control. The real possibility now exists for the virus to hijack the vaccinal response to enhance disease (i.e. VAED). We are seeing enhancement from overseas data following mass vaccination programs but whether it is VAED has yet to be proven as the vaccine also causes immunosuppression which would itself render the vaccinees more susceptible.[53]

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51 A copy of the plan is located on the Medsafe website (https://www.medsafe.govt.nz/COVID-19/Comirnaty-RMP.pdf)

52 Refer footnote 9.

53 For example, see https://www.gov.uk/government/publications/covid-19-vaccine-weekly-surveillance-reports and https://publichealthscotland.scot/media/11076/22-01-12-covid19-winter_publication_report.pdf which indicate greater numbers of Omicron infections and deaths in the vaccinated compared to the unvaccinated.

(f) Herd Immunity

48 Herd immunity is the phenomena where those who have acquired "sterilising" immunity through either infection or vaccines act as "sumps" to remove transmissible pathogens in circulation.

49 Three key points should be made with respect to herd immunity:

(a) it was recognized long before modern vaccines were first rolled out; and

(b) it can have an impact on pathogenic transmission within homes and wider community well below the so called Herd Immunity Threshold, ie as low as 15% of the population having acquired immunity; and

(c) it is wholly dependent on "sterilising" immunity that the COVID-19 vaccines fail to provide. The use of the Pfizer vaccine (Cominarty) is of no benefit in contributing to herd immunity because it fails to stop infections and transmissions. Vaccinating children is not going to protect vulnerable family members.

50 In my opinion, SC2 will never be contained or herd immunity attained while we mandate mass vaccination with a non-sterilising vaccine. Based on numerous precedents within both human and animal populations we know transitioning to herd immunity is typically attained following 3 waves of infection.[54] The first typically impacts the elderly, the second is broad by age demographic in which children largely remain unaffected and typically act as a "sump" to sterilise circulating levels of virus. The third wave is seen as a mopping up exercise in which the virus finally spreads to those who escaped the first 2 waves. It is clear to me that the vaccines are not working as they should and that they have destroyed the "3-wave" paradigm for the transitioning of viral epidemics to endemicity.[55] It is deeply concerning that while data out of the United Kingdom and South Africa confirm >98% of their population have now been exposed to SC2 they are experiencing 4th and 5th waves that no longer resolve. SC2 has been turned into a perpetual cycle of infections, a manmade crisis which, in my opinion, has been borne of mass vaccination with non-sterilising vaccines.

51 The simple facts are you cannot eliminate a virus or generate any meaningful "herd" immunity with a leaky vaccine. It is impossible. But what you can do is render the vaccinee a disease reservoir, especially if severely immunocompromised such as an AIDS patient, allowing the virus to evolve and escape vaccinal pressure thereby raising the viral burden in the wider community.[56] We now have scientific reports submitted for publication that reveal antibody-resistant SC2 variants in

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54 For example, see https://www.cdc.gov/flu/pandemic-resources/1918-commemoration/three-waves.htm

55 https://doi.org/10.1101/2021.12.20.21267966

56 Why scientists think the new variant may have emerged in an HIV patient (telegraph.co.uk)

vaccine breakthrough cases.[57] This is an explosive development. These breakthroughs are not rare events. The problem is our medical agencies charged with public health fail to monitor breakthrough cases unless it leads to hospitalization and death (as per the Centers for Disease Control and Prevention (**CDC**) directives in the United States of America). These escape variants are a harbinger of potentially worse disease with a concomitant increased health burden. If these variants make it to New Zealand then the vaccinated may be rendered defenceless and may exhibit enhanced infection and disease. As mentioned above (para 40), the Omicron VoC may not be a natural escape variant but it is nevertheless immune evading which presents us with the same risk. The concern for many global health experts is that this has not fed into global vaccination strategies regarding COVID-19.

52 There are those who will make the argument that breakthrough cases are misunderstood and that the vaccines are working to reduce hospitalization and death. While the reduction in severe symptoms requiring hospitalization etc would be correct this does not mean the vaccine works to stop infection or transmission. This comes back to the viraemia vs respiratory infection argument above [para 31(a)]. The problem is the virus still circulates resulting in ever increasing viral loads within the wider community. This then impacts the innate immune systems of both children and the unvaccinated. The problem is the vaccinated may then become incubators to throw off escape variants that may end up finding a competitive advantage and crowd out the prevailing VoC. This is precisely what Delta did to Alpha and what Omicron is doing to delta. Where does it stop? The fear is that by mass vaccinating we will end up with something more pathogenic/severe. It is also why we are failing to achieve endemicity. Crucially, while children are increasingly testing positive they are not ending up in hospital because of COVID-19 unless they have significant co-morbidities. This would be true and no worse than for any other ORI.

(g) Erosion of children's innate immunity

53 The immune sterilising effect of children is due to their innate immune system and repertoire of low affinity nIgM Abs. These innate Abs are low affinity and broad specificity, they have evolved precisely to give children an immune advantage until their acquired immune system is educated by real-world exposures and continues to develop until post-puberty. These innate Abs make no distinction as to what the virus is, just that it is "foreign". I believe the mRNA vaccines will erode if not destroy this advantage by promoting unproductive (weak or non-neutralizing) high affinity IgG Abs that out compete the innate nIgM Abs and therefore leave a child's immune system compromised in its ability to fight SC2 as the virus evolves under vaccinal pressure. These arguments are not novel or controversial but are firmly rooted in long standing immunological principles.

54 In other words, by mass vaccinating children with a non-sterilising "vaccine" we lose the natural sterilising impact of a child's immunity.

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57 https://www.nature.com/articles/s41564-021-01041-4

Children will no longer be able to provide "herd" immune protection against SC2 leaving adults, and especially the elderly, at greater risk, the exact opposite of what we wish to achieve by mass vaccinating everyone over the age of 5.

55 For these reasons, vaccine development is traditionally undertaken over a lengthy period of time in order to ensure that these risks are properly investigated before the vaccine is made available to the public. Pfizer acknowledges that it has not properly investigated this risk and that it remains an "important potential risk" that needs to be closely monitored. It is unclear to me how the Government could encourage mass vaccination of children with a leaky mRNA virus in these circumstances. In contrast, the advice of the JCVI would seem more prudent (see 5 above).

(e) Vaccine benefits have been misrepresented

56 Pfizer and representatives of the government have regularly advised that the vaccine has 95% efficiency based upon the original Pfizer clinical trial.[58] This is misleading. The 95% refers to a relative risk reduction (**RRR**), not the more relevant absolute risk reduction (**ARR**). Many in the public have been misled to believe the latter, ie 95% of the vaccinated are protected. This is false, Pfizer's own trial data showed 8 out of 18,198 vaccinated while 162 out of 18325 in the placebo developed COVID-19.[59] The important numbers here are not 8 (0.04%) vs 162 (0.88%) where 0.04/0.88=95% efficiency but 0.88-0.04=0.84% (ARR), ie the vaccine has less than a 1% benefit without considering adverse risk events. Another way to look at this is to consider the number you need to vaccinate (**NNTV**) before you see any benefit of the vaccine. The NNTV for the Pfizer vaccine is 1/0.0084 = 119. The Pfizer trial only looked at symptoms, not mortality or hospitalization or indeed reinfection. The NNTV for the Moderna vaccine is 176. This compares to 1.2 for measles vaccines. I do not believe children should be exposed to the adverse events of a COVID-19 vaccine for less than a 1% benefit particularly in comparison to, for example, the measles vaccine which offers almost 100% benefit without the safety concerns.

Conclusions

57 In my opinion, the data increasingly points to mRNA vaccines (including the Pfizer vaccine) as being a Trojan horse that will compromise our innate and adaptive immune system to fight future waves of coronavirus. It is not possible to eliminate or eradicate a respiratory virus with both human and animal disease reservoirs. From an immunological perspective, the Government's decision to roll out the Pfizer vaccine to otherwise healthy 5-11 years olds will confer no material benefit on them and may well give rise to serious adverse events in the mid to long term. These adverse effects include decreased immunity to future variants of the virus (via antigenic imprinting) and immune deterioration from the need for ongoing

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58 https://www.nejm.org/doi/pdf/10.1056/NEJMoa2034577. See also: https://www.pfizer.com/news/press-release/press-release-detail/pfizer-and-biontech-conclude-phase-3-study-covid-19-vaccine.

59 https://www.nejm.org/doi/pdf/10.1056/NEJMoa2034577

multiple vaccinations and booster shots. There is also a significant risk of VAED. There is no long term data to support vaccination of otherwise healthy children.

Sworn at Upper Hutt)

this 18th day of January)

2022 before me)

Simon Brown

BRADLEY

Patricia Margaret Yee-Nagy
Solicitor
Upper Hutt

This is the exhibit marked **"SBB-1"** referred to in the annexed affidavit of **SIMON BRADLEY BROWN** and sworn at this 18th day of January 2022.

A Solicitor of the High Court of New Zealand

"SBB-1"

Curriculum vitae

Patricia Margaret Yee-Nagy
Solicitor
Upper Hutt

Senior Research Fellow (SFC funded) & Principal Investigator
MRC Centre for Inflammation Research (CIR)
Queen's Medical Research Institute
University of Edinburgh, UK
August 1998 – July 2011

My primary function was to lead a team and develop a successful and independent vein of research that was internationally recognized within the field of inflammatory cell biology, supported 100% by funds won in open competition from either UK Research Councils or medical research charities. My secondary function was to teach and train young scientists, predominantly as researchers (MSc, PhD and Clinical Training Fellows) in pursuit of their own independent careers. As one of the few senior scientists and the only trained chemist in a clinical training environment I was also heavily involved on Health and Safety committees. I also sat on the Postgraduate Committee that formally assessed the annual progress and development of all MSc, PhD and Clinical Training Fellows enrolled within the CIR.

Prior to my one and only permanent position I obtained my post-doctoral research training as:

Senior Research Officer
Queens Medical Centre, University of Nottingham, UK. **1994-1998**

Research Officer
Queensland Institute of Medical Research, Brisbane, Australia. **1990-1994**

It is a measure of my post-doctoral success that I was interviewed for a permanent position with the then Department of Medicine at the University of Edinburgh in 1998.

I append an abbreviated copy of my professional cv for your appraisal.

Tertiary Education:
BSc (Hons) in Chemistry, University of Otago, Dunedin, New Zealand (1980-1983)
PhD in Protein Chemistry & Biospectroscopy, University of Calgary, Calgary, Canada (1985-1989)

Independent Grant Funding (post 2000):

1. The Wellcome Trust (064487): £1,190,306 (4 posts); 01 Jan. 2001 - 31 Dec. 2006 (time only extension to 31 Dec 2007). Co-applicant with Prof John Savill. Macrophage interaction with cells dying by apoptosis and the regulation of glomerulonephritis.
2. The Salvesen Trust (195CIR R36931): £41,173; 01 November 2002 - 30 October 2003. Lead applicant with Dr Nathalie Franc (UCL). CD31-dependent clearance of effete leukocytes in man and drosophila: a model for resolution of inflammation.
3. MRC/EPSRC/BBSRC Discipline Hopping Award (63933): £50,054; 01 October 2003 -30 September 2004. Co-applicant with Dr's V Koutsos, B Noble, and Prof H Simpson. The use of Atomic Force Microscopy to measure cell-cell interactions.
4. Leukaemia Research Fund (LRF 0391): £150,398; 01 December 2003- 31 November 2006. Lead applicant with Dr John Davies, Department of Haematology, Western General Hospital. The role of SHP-1 in myeloid leukaemias.
5. MRC Clinical Research Training Fellowship for Dr Nishrin Spencer. £181,818 (G84/6718); 01 December 2005-31 November 2008. Compartmentalized apoptosis in platelet formation by megakaryocytes.
6. COLT Foundation: £38,696; 01 Aug 2007 – 31 July 2008. Lead Applicant with Profs Bill MacNee & Ken Donaldson. A pilot study into the use of zebrafish as a model for nanoparticle toxicology.
7. Leukaemia Research Fund (LRF 07044): £117,894; 01 July 2007- 30 June 2009. Lead applicant with Prof Christopher D. Gregory, Queen's Medical Research Institute. Role of ERG1, a specific voltage-gated potassium channel, in haematopoietic malignancies.
8. MRC Project grant (G0900550); £700,870; 01 Oct 2009 – 30 Aug 2012. Co-applicant with SG Hillier (PI), HOD Critchley, JI Mason, JP Iredale, CR Harlow. Postovulatory ovarian repair: a role for LOX in scare-free adult healing

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- Brown SB. 2006. Class III anti-arrhythmic methanesulfonanilides in the treatment of Inflammatory Diseases. Patent PCT/GB2006/050345

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2. Hunter C, Kadakia TB, Cooper D, Perretti M, Schwartz RC, Brown SB. 2010. Selective inhibitors of Kv11.1 regulate IL-6 expression by macrophages in response to TLR/IL-1R ligands. **ScientificWorldJournal.** 10:1580-96.
3. Sboros V, Glynos E, Ross JA, Moran CM, Pye SD, Butler M, McDicken WN, Brown SB, Koutsos V. 2010. Probing microbubble targeting with atomic force microscopy. **Colloids Surf B Biointerfaces.** 80(1):12-7.
4. McLeish JA, Chico TJA, Taylor HB, Tucker C, Donaldson K, Brown SB. 2010. Skin exposure to micro- and nano-particles can cause haemostasis in zebrafish larvae.

Thrombosis Haemostasis. 103(4):797-807.
5. Zhang J, Shipston MJ, Brown SB. 2010. A role for potassium permeability in the recognition, clearance and anti-inflammatory effects of apoptotic cells. ***Mol. Neurobiology.*** 42(1):17-24
6. Bournazou I, et al 2009. Apoptotic human cells inhibit migration of granulocytes via release of lactoferrin. ***J. Clin. Invest.*** 119(1):20-32.
7. Poland CA, Duffin R, Kinloch I, Maynard A, Wallace WA, Seaton A, Stone V, Brown S, Macnee W, Donaldson K. 2008. Carbon nanotubes introduced into the abdominal cavity of mice show asbestos-like pathogenicity in a pilot study. ***Nature Nanotechnol.*** 3:423-8.
8. Vernon-Wilson EF, Auradé F, Tian L, Rowe I, Shipston MJ, Savill J, & Brown SB. 2007. CD31 delays phagocyte membrane repolarization to promote efficient binding of apoptotic cells. ***J. Leuk. Biol.*** 82:1278.
9. Brown SB, Tucker C, Ford C, Lee Y, Dunbar D, Mullins J. (2007) Class III anti-arrhythmic methanesulfonanilides inhibit leukocyte recruitment in Zebrafish. ***J. Leuk. Biol.*** 82:79-84.
10. Hartley PS, Savill J, Brown SB. (2007) Hypoglycaemia predisposes platelets to death by affecting calcium homeostasis. ***Platelets*** 18:103-12.
11. Vernon-Wilson EF, Auradé F, Brown SB. (2006) CD31 promotes beta-1 integrin-dependent binding of apoptotic leukocytes opsonized for phagocytosis by fibronectin. ***J. Leuk. Biol.*** 79:1260-67.
12. Donnelly S, Roake W, Brown SB, Naik H, Wordsworth P, Isenberg DA, Reid KBM, Eggleton P. 2006. Calreticulin, CD91 and C1q recognition and clearance pathway of apoptotic neutrophils is impaired in systemic lupus erythematosus. ***Arthritis & Rheumatism*** 54:1543-56.
13. Hartley PS, Savill J, Brown SB. 2006. Platelet death leads to self-agglutination and loss of CD42b. ***Thrombosis & Haemostasis*** 95(1):100-6.
14. Ward JR, Bingle L, Judge HM, Brown SB, Storey RF, Whyte MKB, Dower SK, Buttle DJ, Sabroe I. 2005. Agonists of Toll-like Receptor (TLR)2 and TLR4 are Unable to ModulatePlatelet Activation by Adenosine Diphosphate and Platelet Activating Factor. ***Thrombosis & Haemostasis*** 94(4):831-8.
15. Dransfield I, Rossi AG, Brown SB, Hart SP. 2005. Neutrophils: dead or effete? Cell surface phenotype and implications for phagocytic clearance. ***Cell Death & Differentiation*** 12:1363-7.
16. Clarke MC, Savill J, Jones DB, Noble BS, Brown SB. 2003. Compartmentalized megakaryocyte death generates functional platelets committed to caspase-independent death. ***J. Cell Biol.*** 160:577-87.
17. Jersmann HP, Ross KA, Vivers S, Brown SB, Haslett C, Dransfield I. 2003. Phagocytosis of apoptotic cells by human macrophages: Analysis by multiparameter flow cytometry. ***Cytometry*** 51A:7-15.
18. Brown SB, Heinisch I, Ross E, Shaw K, Buckley CD, Savill J. 2002. Apoptosis disables CD31-mediated cell detachment from phagocytes promoting binding and engulfment. ***Nature*** 418:200-3.
19. Brown SB, Clarke MCH*, Magowan L, Sanderson H, Savill J. 2000. Constitutive death of platelets leading to scavenger receptor-mediated phagocytosis. A caspase-independent cell clearance program ***J. Biol. Chem.*** 275: 5987-96.

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- Brown SB, Dransfield I (2008). Electric fields and inflammation: may the force be with you. ***The Scientific World Journal.*** 8:1280-94.
- Gregory CD, Brown SB. (2005). Apoptosis: eating sensibly. ***Nature Cell Biol.*** 7:1061-3.

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