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	**DCB**
	First to Eighth Applicants
AND	**THE MINISTER OF HEALTH**
	First Respondent
AND	**THE GROUP MANAGER OF THE NEW ZEALAND MEDICAL DEVICES SAFETY AUTHORITY (MEDSAFE)**
	Second Respondent

Continued on next page

AFFIDAVIT OF SIMON BRADLEY BROWN

Dated April 2022

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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 1993-1994, 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 Inflammation Repair Group, 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, Head of the Inflammation Repair Group and also Head of the College of Medicine and Veterinary Medicine and Chief Executive of the Medical Research Council (United Kingdom).

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.

SBB

5. 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.

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.[3] 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. In December 2021, the New Zealand Minister of Health granted provisional consent under the Medicines Act 1981 for the use of the Pfizer (Comirnaty) vaccine in 5-11 year old children.[4] I have been asked to give my expert opinion on whether the decision was reasonable and justified based on the data and science available and, in particular, whether the benefits of vaccination outweigh the risks associated with the Pfizer mRNA vaccine.

Summary of my evidence

8. Healthy children are at no discernible risk from COVID-19 (no more risk than from the seasonable flu). A healthy child's innate immune is adept at fighting opportunistic respiratory infections like COVID-19. Accordingly, a healthy child does not obtain any measurable benefit from taking the Pfizer vaccine.

9. Pfizer's clinical trials of its vaccine were wholly inadequate to establish long term safety. There are now increasing reports of negative vaccine efficacy (**VE**) in

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

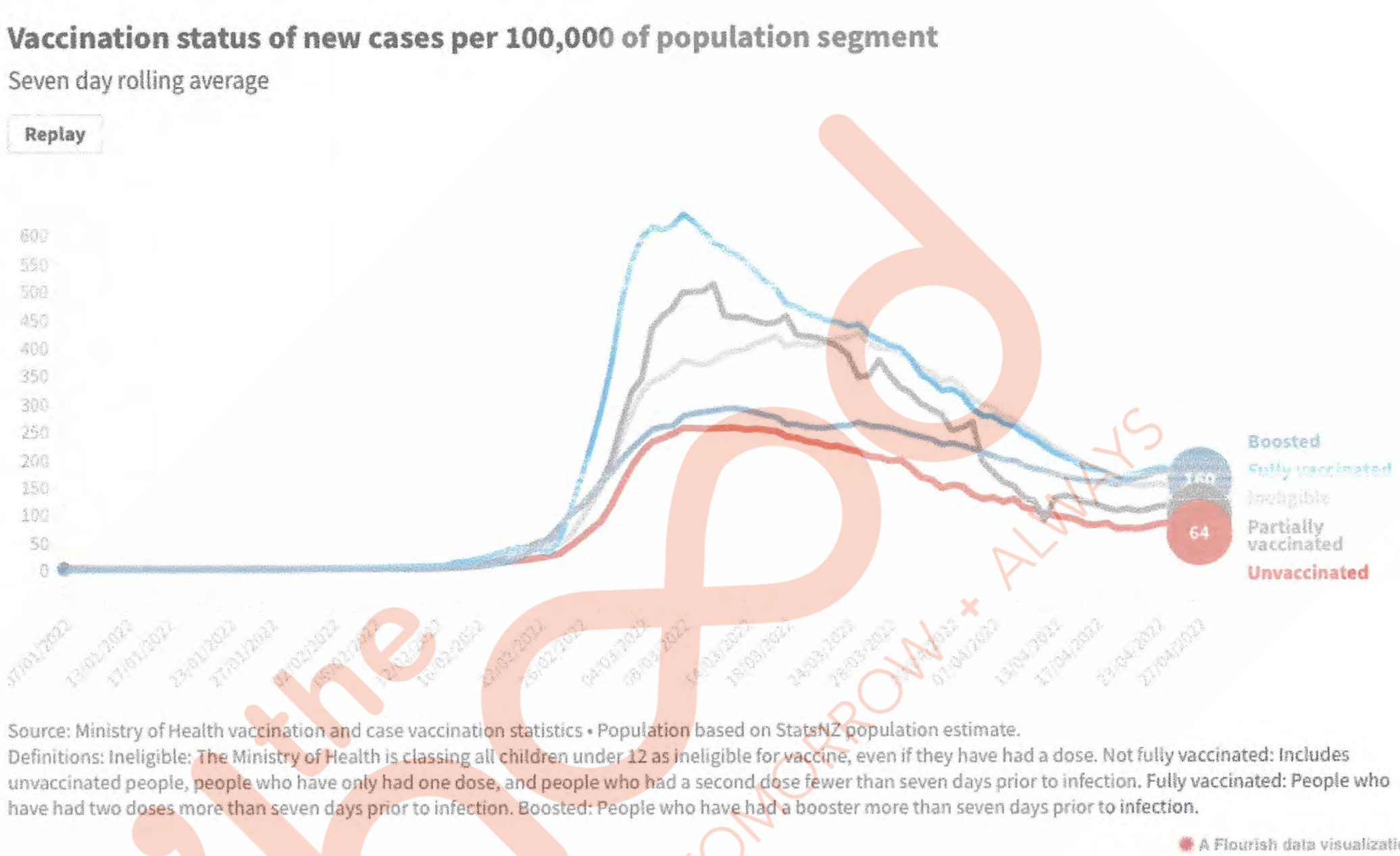
2 https://www.express.co.uk/news/uk/1547050/covid-vaccine-JCVI-omicron-delta-myocarditis

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

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

Europe, the UK and Canada. Negative VE manifests in increased infections, hospitalisations, and deaths for the vaccinated compared to the non-vaccinated.

10. Negative VE is readily apparent now from New Zealand Ministry of Health data. The RadioNZ prepared graph below illustrates that per 100,000 population segment, those who have received their booster shot are almost three times more likely to be infected with Omicron:[5]



11. This indicates that the Pfizer vaccine may actually be damaging people's immune function making them more susceptible to disease.

12. The clinical trials for the Pfizer vaccine in 5-11 year old children were underpowered and did not establish safety. Evidence is emerging that adverse events from the vaccine in children (including death) vastly exceed any benefit.

13. The Pfizer paediatric vaccine is all risk with no benefit for an otherwise healthy child.

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5 https://www.rnz.co.nz/news/in-depth/450874/covid-19-data-visualisations-nz-in-numbers (as at 27 April 2022)

14. From a public health perspective, vaccination should reduce transmission by providing "sterilizing" immunity for the individual vaccinated. Sterilizing immunity is critical for "herd" immunity to be attained and for pandemics to transition towards endemicity. Endemicity is where infection within a population is maintained at a constant, predictable and manageable baseline level.

15. However, the vaccine has failed from a public health perspective.[6] Instead of reducing or stopping infection and transmission it has enhanced both.[7] This tells us the vaccine has damaged the immune system of the vaccinated. This has potentially serious future immunological and health consequences for the vaccinated especially with the emergence of any new immune evading viral variants driven by the use of non-sterilizing "leaky" vaccines that includes Pfizer's COVID-19 vaccine.

16. It is not possible to eliminate or eradicate a respiratory virus with both human and animal disease reservoirs or attain herd immunity when using a non-sterilizing "leaky" vaccine.[8] Worse, by mass vaccinating children we lose the superior natural sterilizing impact of a child's immunity. Children will no longer be able to provide "herd" immune protection against SARS-CoV-2 (SC2) infection leaving adults, and especially the elderly, at greater risk. The exact opposite of what we had hoped to achieve by mass vaccinating everyone over the age of 5.

17. In other words, endemicity is dependent on widespread population immunity and the limited availability of susceptible hosts. The use of "leaky" vaccines has had the opposite effect of expanding the susceptible population to now include children aged 5-11.

18. In contrast, naturally acquired immunity provides broad, durable and superior "sterilizing" immunity to that of Pfizer's vaccine,[9] a finding confirmed in a recent

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6 https://doi.org/10.1101/2022.03.29.22273146

7 Worldwide Bayesian Causal Impact Analysis of Vaccine Administration on Deaths and Cases Associated with COVID-19: A BigData Analysis of 145 Countries (vector-news.github.io)

8 https://www.medrxiv.org/content/10.1101/2022.03.29.22273146v1

9 https://doi.org/10.1016/j.xcrm.2021.100354
https://doi.org/10.1016/S1473-3099(21)00676-9
https://www.israelnationalnews.com/news/312637

instalment of documents released under court order and held by the FDA as part of Pfizer's application for EUA approval of its vaccine.[10]

Scope of affidavit

19. I address the following issues in support of the above statements made in this affidavit:

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

(b) NZ population data.

(c) Inadequate safety data to properly grant provisional consent

(d) Random Clinical Trials and Obfuscation of Adverse events

(e) Vaccine benefits have been misrepresented

(f) Impact of vaccination on infection rates

(g) Impact of Vaccination on Transmission

(h) Impact of Vaccination on Hospitalization and Disease Severity

(i) The human immune system

(j) Antigenic Imprinting and Antibody Dependent Enhancement (ADE)

(k) Humoral response to Spike protein promotes infection by Antibody-Dependent Enhancement (ADE)

(l) Is the Booster masking ADE?

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

20. Omicron is now the predominant SC2 variant of concern (VoC) currently in global circulation where prior VoCs, including Delta, have all but been eliminated. This is no less true for NZ than it is for England or elsewhere as illustrated in the figures below:

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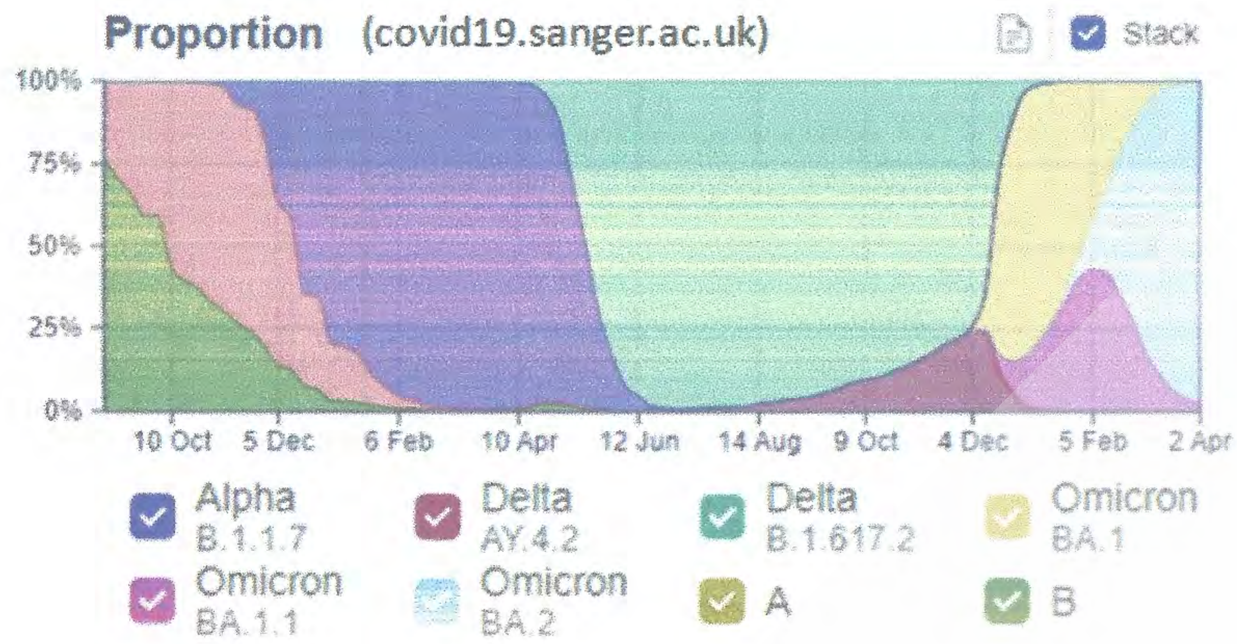
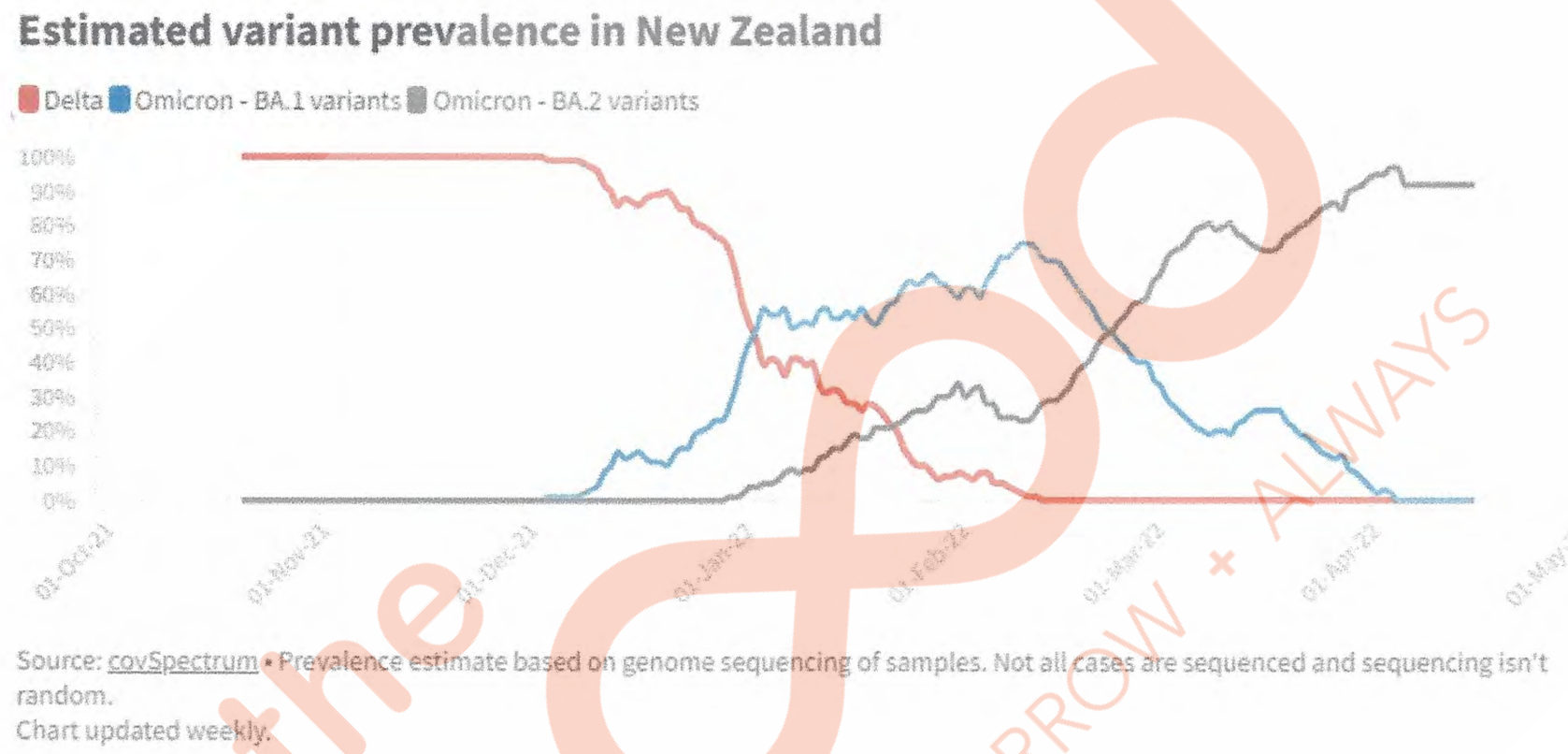
10 https://www.medrxiv.org/content/10.1101/2021.08.24.21262415v1.full.pdf
https://doi.org/10.1101/2021.11.15.21265753
https://doi.org/10.1101/2021.08.27.21262741
https://doi.org/10.1101/2022.04.18.22271936
https://phmpt.org/wp-content/uploads/2022/04/reissue_5.3.6-postmarketing-experience.pdf



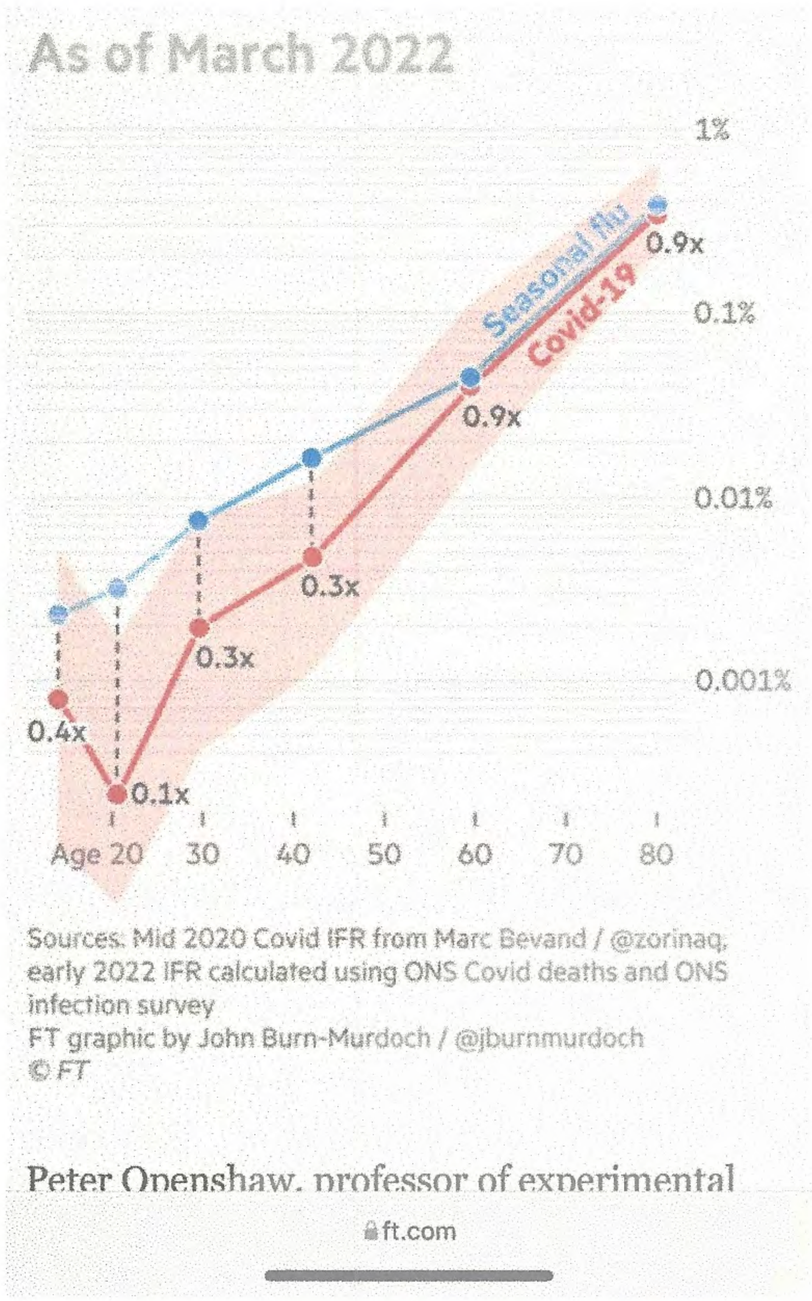
(Source: https://covid19.sanger.ac.uk/lineages/raw)



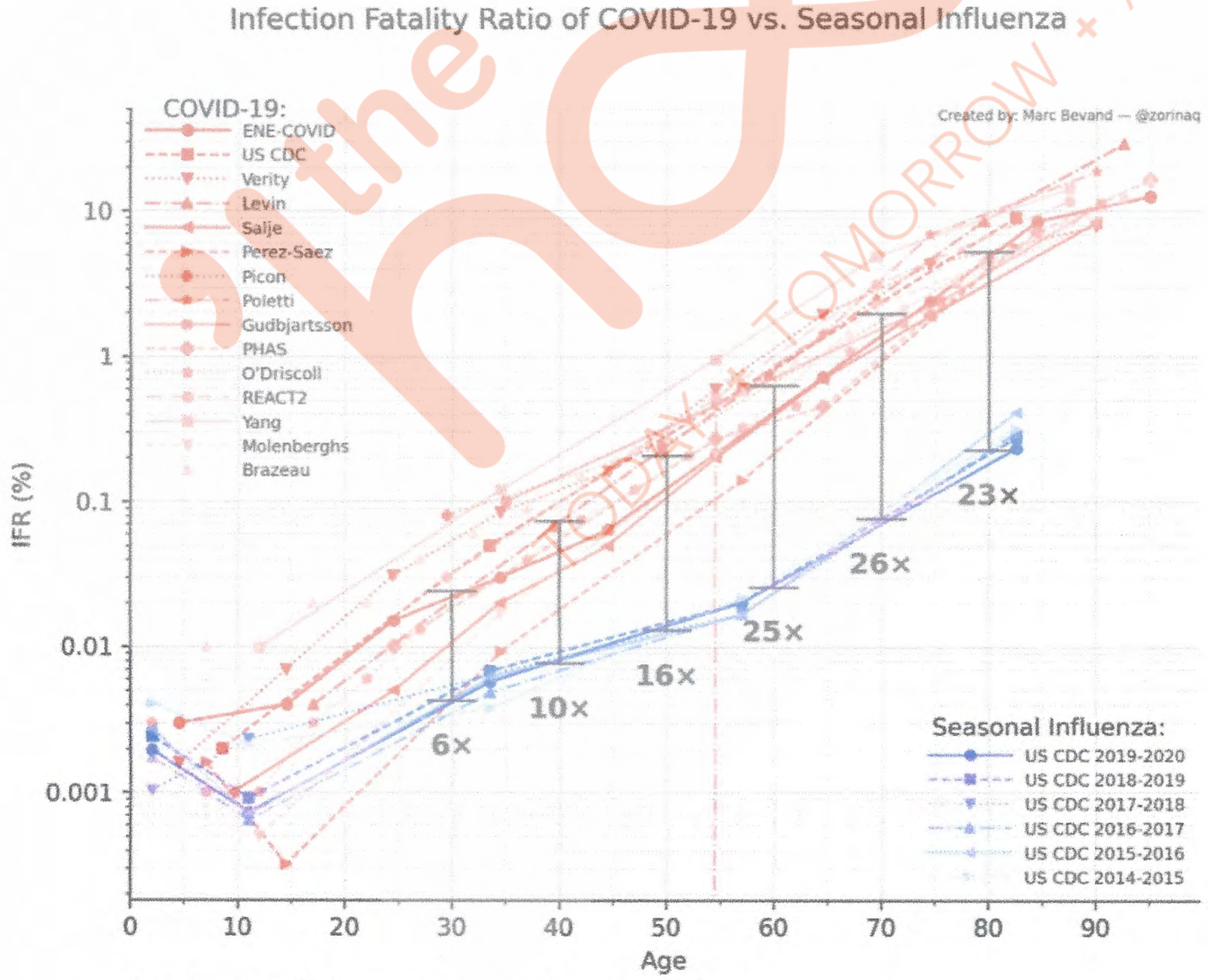
(Source: https://www.rnz.co.nz/news/in-depth/450874/covid-19-data-visualisations-nz-in-numbers)

21. There is no reason for healthy individuals (<65 yo), especially children and adolescents (<15 years old) to fear COVID-19, no more so than for any other seasonal respiratory infection.

22. The Infection Fatality Rate (**IFR**) for COVID-19 since 2019 for the overall population (i.e. all age groups) is approximately 0.4% ranging from 0.003% (ie 3 per 100,000 infections) for those aged <19, 0.27% for 50-59 year olds and 5% in those >80. These numbers have been further revised down for Omicron and are no worse than seasonal flu.



(Source: https://www.ft.com/content/e26c93a0-90e7-4dec-a796-3e25e94bc59b)



(Source: https://github.com/mbevand/covid19-age-stratified-ifr)

23. The above graphs confirm the superiority of a child's innate immune system in fighting opportunistic respiratory infections (**ORI**). The first graph represents the UK averaged over its entire COVID-19 window whereas the second graph represents global data taken at an earlier time point in the pandemic cycle when the Alpha VoC dominated. The data also demonstrates how the virulence of SC2 has attenuated while we know transmissibility increased.

24. It therefore follows that a COVID-19 vaccine is unnecessary for children aged 5-11 and there is no benefit from mass vaccination unless the child is already significantly immune-compromised or immune-suppressed and unable to rely on their own immune system. A child's doctor is best placed to assess this individual risk

(b) NZ population data.

25. The following table is taken from the Ministry of Health's (**MoH**) website: [11]

COVID-19 cases by age group

Age group	Active (confirmed and probable)	Recovered	Deceased	Total cases	Percentage of all cases
0 to 9	6187	106021	1	112209	12.6%
10 to 19	7891	154466	3	162360	18.2%
20 to 29	10337	163300	3	173640	19.5%
30 to 39	10562	149492	10	160064	18%
40 to 49	8876	116267	19	125162	14.1%
50 to 59	6998	75927	40	82965	9.3%
60 to 69	4610	41033	77	45720	5.1%
70 to 79	2219	16709	141	19069	2.1%
80 to 89	868	5992	203	7063	0.8%
90+	290	1348	147	1785	0.2%
Unknown	0	2	0	2	0%
Total	58838	830557	644	890039	100%

26. We can see that 31% (274569/890039) of all reported COVID-19 cases to date in NZ have occurred in those 18 years old or younger, the vast majority being Omicron. Of these, 958 were hospitalized with 1 death in the 0-9 and 3 in the 10-19 age group. This is a hospitalization rate of 0.3% (3 per 1000 cases) and an IFR of 0.001% (10 per million). There are 1.2 million children under the age of 18

11 https://www.health.govt.nz/covid-19-novel-coronavirus/covid-19-data-and-statistics/covid-19-case-demographics

in NZ, or ~24% of the population. While the IFR is in keeping with International data the hospitalization rate is not.

27. The US Centre for Disease Control (**CDC**) estimates that the risks of children aged 5-11 being hospitalized with COVID-19 has increased from 25 cases per 1,000,000 (<0.003%) for the Delta VoC to 40 for Omicron (0.004%) which is equivalent to what is seen for influenza.[12] This raises questions as to how NZ is attributing hospitalization data to COVID-19.

28. Children aged 5 to 11 are at extremely low risk of death from COVID-19. The foremost authority on estimating the infection fatality rate (IFR) for COVID-19 is John Ioannidis.[13] He estimates a median IFR for the Delta VoC of 0.0027% which compares with influenza in children ages 0-19. This equates to 27 deaths per million. While not wanting to sound dismissive of these deaths they predominantly occur in children with serious co-morbidities in readily identifiable risk groups.

29. A recent study has concluded that the IFR for Omicron has substantially decreased from earlier variants. The study calculated the reduction at 78.7%.[14]

30. Anecdotal data from the UK also indicates that whilst child COVID-19 hospitalisations may have increased, they are suffering from much milder symptoms. In a recent article in the Financial Times, Alasdair Munro, a paediatrics specialist at the UK's National Institute for Health Research is reported as stating that:[15]

> *Before Omicron, children [with coronavirus] came in with real respiratory distress and pneumonia. The difference is that Omicron is causing much more common respiratory diseases. Cases come in looking like other unwell children and within a day or so – under observation and ruling out other, more*

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12 Hospitalisations jump in US children aged under five with COVID-19, CDC data says - ABC News

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

14 https://doi.org/10.1016/j.ijid.2022.04.029

15 https://www.ft.com/content/28be9d3f-0b12-4c33-bda9-fbff375c0b7e

serious infections – they look a lot better", he said, adding that Covid-19 now often resembled those viruses such as croup and bronchitis.

31. Thus, we can conclude that a child's risk of serious illness with COVID-19 and the Omicron VoC in particular, is no worse than seasonal flu. It is against this risk that we must now assess the benefits, or lack thereof, of vaccination.

(c) Inadequate safety data to properly grant provisional consent

32. It is not controversial to state that Pfizer's clinical trials have been wholly inadequate to establish the 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.[16] 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

33. 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.

34. 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

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

integrity and regulatory oversight.[17] Pfizer's clinical trial involving 12-15 yr olds has also been harshly criticised.[18]

(d) Randomized Controlled Trials and Obfuscation of Adverse events

35. 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 year olds, the Ministry of Health explains on its website that:[19]

> *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]

36. In other words, the Government is relying upon data arising from actually vaccinating children to establish whether or not the vaccine is safe. This is extraordinary; particularly in circumstances where the Government expressly acknowledges that COVID-19 generally has mild symptoms in children "with symptoms similar to a cold".[20]

37. It is important to revisit what the Pfizer vaccine randomized controlled trials (**RCT**) actually demonstrated. In particular, Pfizer's RCT for juveniles (5-11 year olds) and its RCT for adolescents (12-15 year olds).

38. Analysis using the juvenile RCT injury report is difficult to assess as the rate of severe symptoms appeared to match those in the placebo group with a much

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17 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/

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

19 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

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

shorter follow-up period of 1 month vs 4 months for the 12-15 year olds.[21] The dose for juveniles was also 10µg compared with 30µg for the adolescents and therefore may be better tolerated.

39. Nevertheless, the juvenile RCT suggested that for every 2 juvenile non-severe COVID cases we could expect one child to have a severe grade 3 reaction to the vaccine. Grade 3 systemic reactions included fever >102.1F; vomiting that requires intravenous hydration; diarrhea; or severe fatigue, headache, muscle pain, or joint pain that prevented daily activity. Importantly, no unvaccinated child aged 5-11 in the RCT suffered a severe case of COVID-19.

40. Dr. Eric Rubin, editor of the prestigious New England Journal of Medicine (where Pfizer's RCT conclusions were published) and US Food & Drug Administration (FDA) adviser, has stated we were never going to know the safety of the 5-11 year-old vaccine "unless we start giving it".[22]

41. Many commentators have inferred a safety and efficacy for the paediatric vaccine by way of comparison with the adolescent RCT. With respect to the RCT for 12-15 year olds the trial recruited 2264 adolescents between Oct 2020 and Jan 2021 in which 1180 received one 30microgram (µg) dose before 13 Mar 2021[23]. The first thing to note is this trial was not sufficiently powered to address adverse events but designed to address tolerance of the dose (reactogenicity) and alleviation of symptomatic infection for a period <4months after dose 2.

42. On the positive side the trial showed zero COVID-19 cases in the vaccinated group and 16 mild cases (non-severe) in the placebo group giving a VE of 100%. The bad news is the predominance of harm and adverse events in the treated group. What the RCT did reveal is 7 individuals in the treated group suffered significant and serious life-threatening events with none observed in the control group. At a lesser level, lymphadenopathy was seen at 9 vs 2 and 7 vs 1 after the first and second doses between treated and control groups. Finally 51% of the treated group used anti-pyretic medication vs 9% of the control group.

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

22 https://www.politifact.com/article/2021/nov/01/context-never-going-learn-how-safe-vaccine-unless-/

23 https://www.nejm.org/doi/full/10.1056/NEJMoa2107456

43. Thus, for the adolescent trial 7 out of 1180 vaccinations (ie 0.6%) resulted in severe (grade 3) systemic reactions in the vaccinated group. Recall that the CDC reports 40 hospital admissions per million cases with COVID-19 (0.004%). Therefore the vaccine is likely to cause ~150x (0.6%/0.004%) more harm than COVID-19, or x2 if we accept NZ numbers.

44. This is not a good safety profile. It was also not powered appropriately to discover any adverse events that may occur at a rate less than 1 in 1180 such as myocarditis/pericarditis, estimated to occur at a frequency of 1 in 5000[24] although a more recent Nordic study suggests 1 in 2600[25]. That said, Pfizer's adolescent RCT did reveal one event of myocarditis/pericarditis in the treated group yet its significance was dismissed and contradicted in their report where they state "there were no vaccine-related serious adverse events and few overall severe adverse events".[26]

45. Using the Vaccine Adverse Event Reporting System (**VAERS**)[27] and the number of people aged 12-24 reported to have died from the Pfizer vaccine since 10 May to 31 Oct 2021 in the USA, Toby Rogers (PhD) estimated for every one child saved another 117 were killed by the vaccine.[28] This is based on estimates of 2-4 million doses needed to be administered to protect one child from death of COVID-19 and 128 reports of fatal side effects following coronavirus mRNA injections within this age cohort.

46. Vaccine injuries are far more common than we are led to believe. This can best be seen in the original RCT data but also from Pfizer's 12 week post-marketing experience which the FDA was recently forced to release under court order.[29] With

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24 https://doi.org/10.1093/cid/ciab989; Adverse Events Following Immunization (AEFIs) for COVID-19 in Ontario: December 13, 2020 to March 6, 2022 (publichealthontario.ca); SARS-CoV-2 mRNA Vaccination-Associated Myocarditis in Children Ages 12-17: A Stratified National Database Analysis | medRxiv

25 https://jamanetwork.com/journals/jamacardiology/fullarticle/2791253

26 https://www.nejm.org/doi/full/10.1056/NEJMoa2107456

27 VAERS is the US "early warning system" that monitors the safety of vaccines after they have been authorised or licensed for us by the FDA.

28 https://childrenshealthdefense.org/defender/fda-pfizer-covid-vaccine-risk-benefit-analysis-nntv-children/

29 https://phmpt.org/wp-content/uploads/2022/04/reissue_5.3.6-postmarketing-experience.pdf

the latest instalment we now know "The rollout of the Pfizer vaccine has led to an unprecedented number of adverse events reported — 158,000 adverse events in the first two-plus months of the rollout means that the rate of reported AE [adverse events] was approximately 1:1000, with many of the AEs graded as serious. This is based on a denominator of 125,000,000 vaccines distributed."[30] This includes 1223 deaths which is roughly the equivalent of 1 death per 100,000 people vaccinated.[31] The table below is taken directly from the Pfizer post marketing report on adverse events:

BNT162b2
5.3.6 Cumulative Analysis of Post-authorization Adverse Event Reports

Table 1 below presents the main characteristics of the overall cases.

Table 1. General Overview: Selected Characteristics of All Cases Received During the Reporting Interval

Characteristics		Relevant cases (N=42086)
Gender:	Female	29914
	Male	9182
	No Data	2990
Age range (years): 0.01 -107 years Mean = 50.9 years n = 34952	≤ 17 18-30 31-50 51-64 65-74 ≥ 75 Unknown	175[a] 4953 13886 7884 3098 5214 6876
Case outcome:	Recovered/Recovering	19582
	Recovered with sequelae	520
	Not recovered at the time of report	11361
	Fatal	1223
	Unknown	9400

a. in 46 cases reported age was <16-year-old and in 34 cases <12-year-old.

As shown in Figure 1, the System Organ Classes (SOCs) that contained the greatest number (≥2%) of events, in the overall dataset, were General disorders and administration site conditions (51,335 AEs), Nervous system disorders (25,957), Musculoskeletal and connective tissue disorders (17,283), Gastrointestinal disorders (14,096), Skin and subcutaneous tissue disorders (8,476), Respiratory, thoracic and mediastinal disorders (8,848), Infections and infestations (4,610), Injury, poisoning and procedural complications (5,590), and Investigations (3,693).

47. EudraVigilance, the European database of suspected adverse drug reactions, reports 4,100,476 injuries and 43,005 deaths through to April 09, 2022, almost half of which is due to Tozinameran (code BNT162b2, Cominarty) from

30 https://childrenshealthdefense.org/defender/pfizer-hired-600-people-vaccine-injury-reports/

31 126,212,580 doses of vaccine were shipped between 1 December 2020 and 28 February 2021. Pfizer's post marketing report records 1223 deaths.

BioNTech/Pfizer. As in the USA, this is an unprecedented level of adverse events and vaccine harm.[32]

48. To conclude, the paediatric vaccine is all risk with no benefit for the individual child, especially when no unvaccinated child aged 5-15 in the RCT suffered a severe case of COVID-19.

(e) Vaccine benefits have been misrepresented

49. Pfizer and representatives of the government have regularly advised that the adult vaccine has 95% effectiveness based upon the original Pfizer clinical trial.[33] 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.[34]

50. The important numbers are not 8 (0.04%) vs 162 (0.88%) where (0.88-0.04)/0.88=95% vaccine efficacy (RRR) 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 >83% benefit without the safety concerns.

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32 43,000 Deaths 4 MILLION Injuries Following COVID-19 Vaccines in European Database of Adverse Reactions - Vaccine Impact

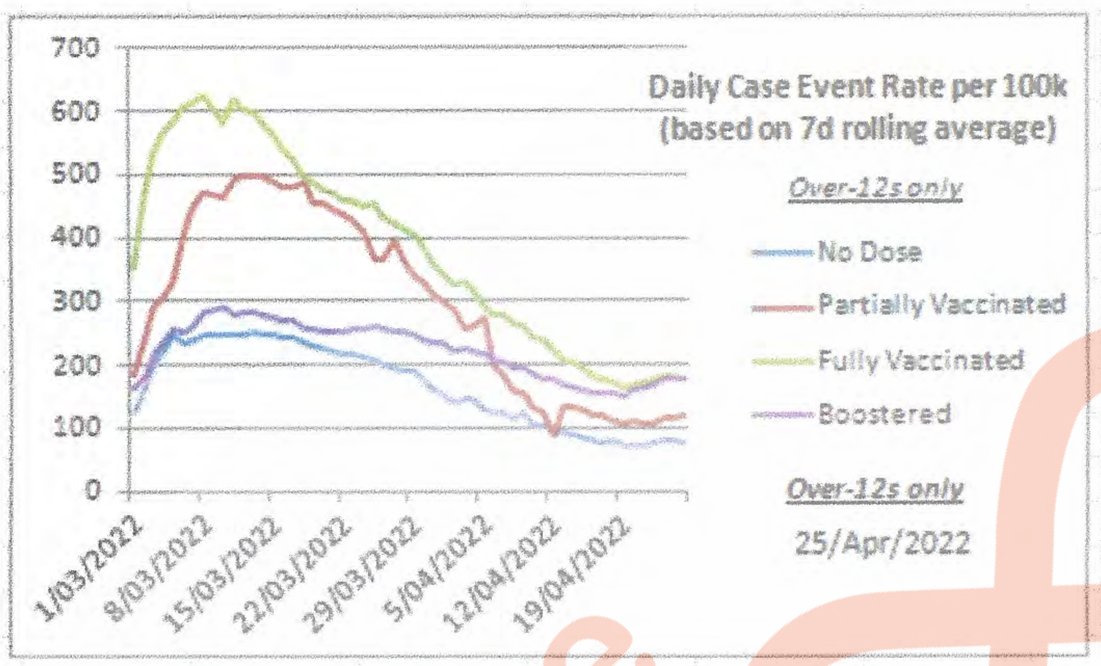
33 https://www.nejm.org/doi/pdf/10.1056/NEJMoa2034577
https://www.pfizer.com/news/press-release/press-release-detail/pfizer-and-biontech-conclude-phase-3-study-covid-19-vaccine

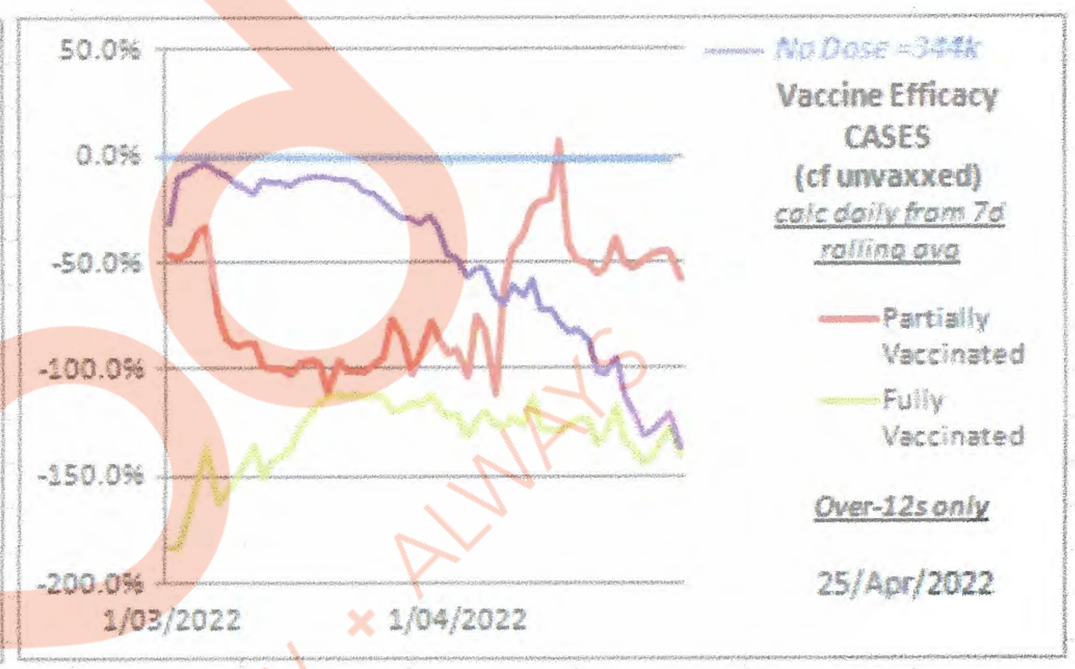
34 https://www.nejm.org/doi/pdf/10.1056/NEJMoa2034577

(f) Impact of vaccination on infection rates

52. A common MoH message to encourage vaccination was that it offered the best form of protection for children, their families, and the community against COVID-19.

53. This is demonstrably false. We now have a large cohort of the NZ population infected with the Omicron variant for which MoH data shows vaccination status. I have used the MoH data to calculate the daily case event rate per 100,000 and vaccine efficacy in the two tables below:



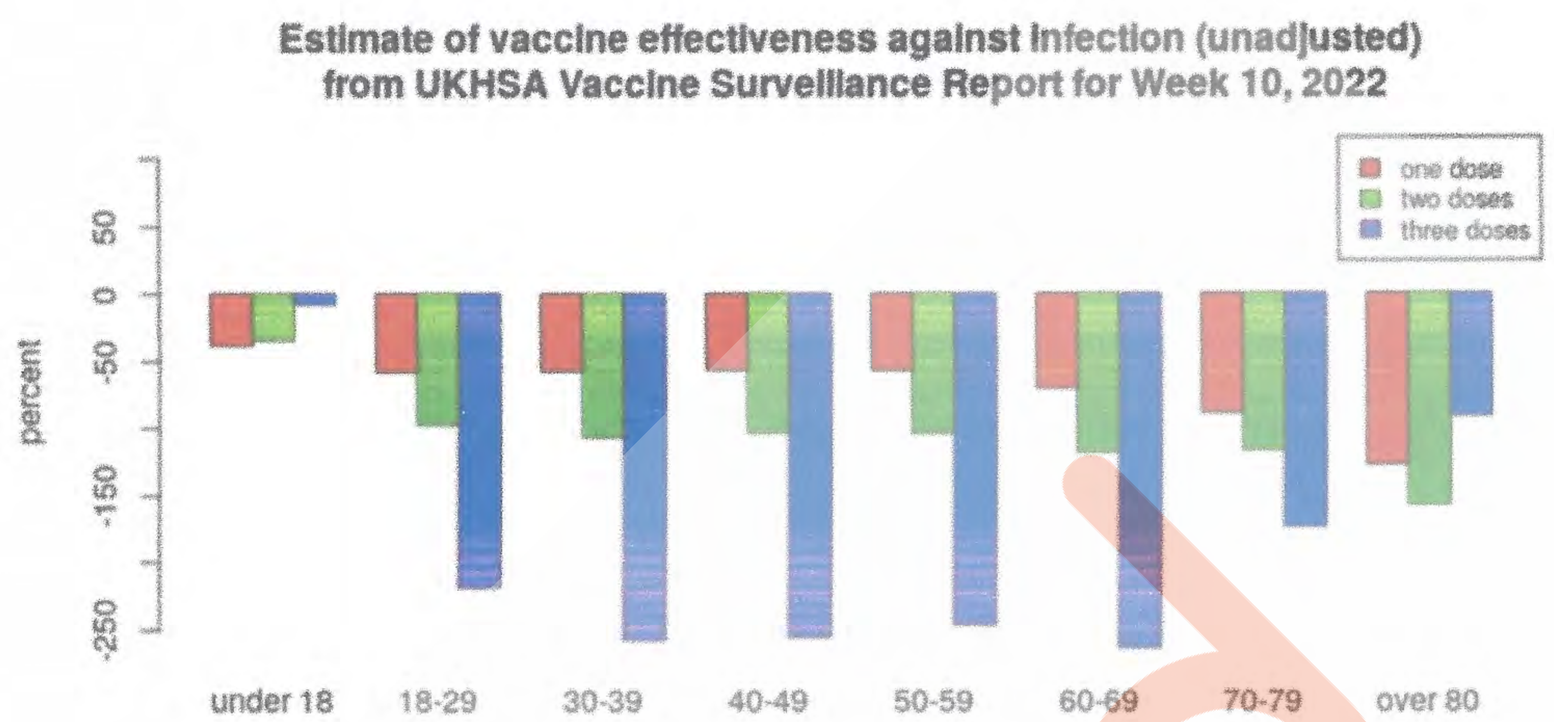


#1 https://www.health.govt.nz/covid-19-novel-coronavirus/covid-19-data-and-statistics/covid-19-case-demographics
#2 https://www.nzherald.co.nz/nz/vaccine-tracker-how-many-kiwis-have-been-vaccinated/ENMCOHM5QW6W3UN6MRMCOQKO2U/

54. Note that the numbers derived are for the eligible population as we have as yet no granularity on the status of the under-12s, nor are the data age, sex and morbidity matched. VE is estimated as per Pfizer in their original application to the FDA for Emergency Use Authorisation of their COVID-19 vaccine (BNT162b2); i.e., VE=RRR. I have assumed no bias in data collection with any potential source of error considered equivalent within the various vaccine cohorts.

55. The first conclusion to draw from these two tables is that the fully vaccinated have consistently been more than twice as likely (VE=-120%) to be infected with COVID-19 than the unvaccinated throughout the period when Omicron VoC infections dominated. These negative values cannot be dismissed on grounds of base rate fallacies.

56. The UK Health Security Agency (**UKHSA**) provides, perhaps, the world's most transparent and granular database to track COVID-19 infections through its vaccine surveillance reports.[35]



57. Negative VE is also now extended to the under-18 age cohort. A recent UKHSA Vaccine Surveillance Report (10 March 2022) confirms that children under the age of 18 are now also experiencing negative VE.[36] While negative VE is somewhat negated by a booster shot it does not promote protection from infection. Unfortunately, we can expect any benefit to be short-lived[37] and likely to become progressively worse as seen with the other age cohorts. These conclusions are supported by independent studies.[38]

58. We also now have a large set of data collected by health officials in New York State showing the paediatric vaccine offered almost no protection against infection for 5-to-11 year olds, even just a month after full vaccination.[39]

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35 https://www.gov.uk/government/publications/covid-19-vaccine-weekly-surveillance-reports

36 https://dailysceptic.org/2022/03/13/infection-rates-higher-in-triple-vaccinated-than-unvaccinated-across-all-age-groups-ukhsa-data-show/

37 https://t.co/bW3H8uDRLG

38 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

39 Effectiveness of the BNT162b2 vaccine among children 5-11 and 12-17 years in New York after the Emergence of the Omicron Variant | medRxiv

59. Negative VE started to reveal itself in UKHSA reports from mid-August 2021 (Report #36) when the vaccination status of cases was first reported,[40] and was confirmed in subsequent reports.

60. The Table below is sourced from the UKHSA report for Week 41, 2022:[41]

Table 2. COVID-19 cases by vaccination status between week 37 and week 40 2021

Cases reported by specimen date between week 37 and week 40 2021	Total	Unlinked*	Not vaccinated	Received one dose (1-20 days before specimen date)	Received one dose, ≥21 days before specimen date	Second dose ≥14 days before specimen date	Rates among persons vaccinated with 2 doses (per 100,000)	Rates among persons not vaccinated (per 100,000)	Vaccine Effectiveness
30-39	83,007	7,138	20,532	626	6,479	48,232	823.9	709.8	**- 16%**
40-49	111,896	6,778	11,729	292	3,551	89,546	1,455.8	696.2	**- 109.1%**
50-59	74,981	4,506	4,998	85	1,463	63,929	903.1	489.3	**- 84.6%**
60-69	38,184	2,455	1,694	24	525	33,486	589.0	314.1	**- 87.5%**
70-79	23,109	1,363	622	7	201	20,916	451.5	253.0	**- 78.5%**
80+	10,770	839	375	7	184	9,365	364.6	298.5	**- 22.1%**

61. The final column contains my own calculations of VE. It illustrates negative VE in all age groups above 30 years old.

62. Negative VE means the vaccine has damaged the immune system of those who have been vaccinated and/or their vaccinal response is giving the virus a helping hand (i.e via Antibody Dependent Enhancement (**ADE**)).

63. There are also indications that the immune function of vaccinated individuals is dysfunctional. This is perhaps best evidenced by increasing reports of negative vaccine efficacy (**VE**) in Europe,[42] the United Kingdom[43], and Canada[44] that correlate with increasing prevalence of Omicron as found in a study from the United States of America.[45] Negative VE now manifests in increased

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40 https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1016465/Vaccine_surveillance_report_-_week_36.pdf

41 https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1025358/Vaccine-surveillance-report-week-41.pdf

42 https://t.co/vc6B39HYZf

43 https://www.gov.uk/government/publications/covid-19-vaccine-weekly-surveillance-reports

44 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

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

hospitalisation and death rates for the vaccinated as compared to the non-vaccinated in Scotland.[46]

(g) Impact of Vaccination on Transmission

64. Studies have shown that the secondary attack rates (SAR) and viral loads for the transmission of COVID-19 are similar for those vaccinated or unvaccinated.[47] Most of these studies looked at the Delta VoC although it has also been confirmed for Omicron.[48]

65. Perhaps the more notorious example of vaccine failure in preventing transmission is the large Omicron outbreak that occurred at a Norwegian Christmas party with an attack rate of 74% among participants in which 98% were fully vaccinated.[49] There are numerous other examples.

66. Our MoH acknowledged in a position statement posted on their website 18 Nov 2021 (only to delete the very next day):[50]

> *"When there is high COVID-19 vaccine coverage (i.e., above 80 percent of eligible people are fully vaccinated), transmission is more likely to occur from a vaccinated than an unvaccinated individual"* and *"As our vaccination numbers increase, we will see fewer cases but more of those cases will be in fully vaccinated people,* ***meaning it is more likely transmission will occur from a vaccinated individual than an unvaccinated individual"*** (my emphasis)

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

47 https://www.thelancet.com/journals/laninf/article/PIIS1473-3099(21)00648-4/fulltext
https://www.medrxiv.org/content/10.1101/2021.11.12.21265796v1.full
https://www.thelancet.com/journals/lanwpc/article/PIIS2666-6065(21)00208-X/fulltext
https://www.medrxiv.org/content/10.1101/2021.07.31.21261387v6

48 https://doi.org/10.1101/2022.02.07.22270437; https://www.news-medical.net/news/20220210/Study-suggests-SARS-CoV-2-Omicron-showed-increased-household-transmission-and-immune-evasion-in-Norway.aspx

49 https://doi.org/10.2807/1560-7917.ES.2021.26.50.2101147

50 https://twitter.com/SinbSinbad/status/1461407067000827908?s=19; https://t.co/lV35xNaQWN

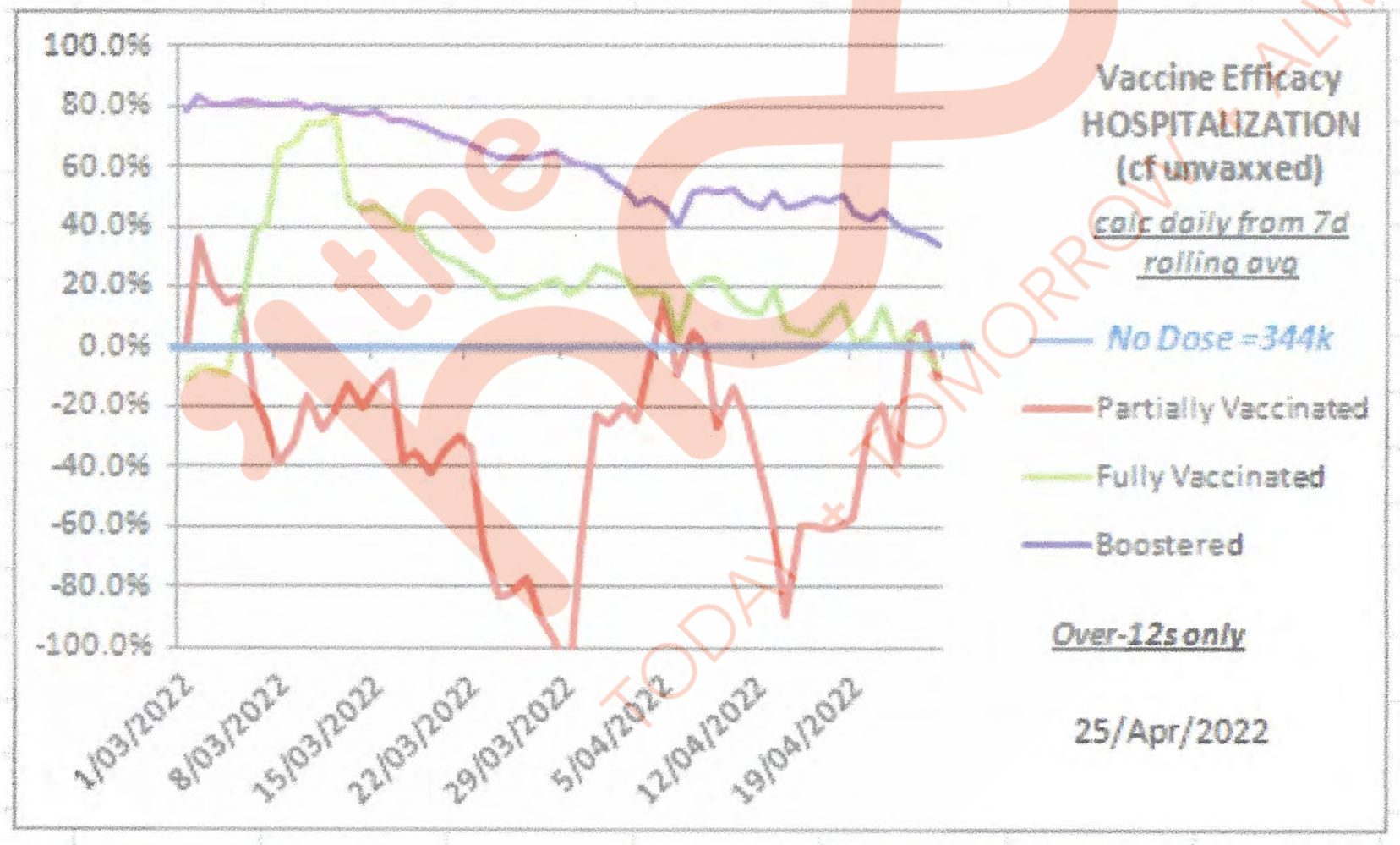
67. This position statement was updated on 19 November 2021 to state:[51]

> "... the difference in the risk of transmission between vaccinated and unvaccinated people will be negligible."
>
> "Asymptomatic infection is the issue, not the vaccination status of the patient."

68. Thus, we can conclude that vaccination has no significant impact on transmission.

(h) Impact of Vaccination on Hospitalization and Disease Severity

69. The Pfizer vaccine does appear to have a benefit with respect to disease severity and hospitalization in those 12 years and older which improves with a third booster dose. However, the effect is known to wane significantly by 6 months.[52] This is illustrated by the following table that I prepared from MoH data:



#1 https://www.health.govt.nz/covid-19-novel-coronavirus/covid-19-data-and-statistics/

#2 https://www.nzherald.co.nz/nz/vaccine-tracker-how-many-kiwis-have-been-vaccinat

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51 Ministry of Health position statement, https://web.archive.org/web/20211119020805/https://www.health.govt.nz/system/files/documents/pages/ministry_of_health_position_statement_on_the_management_of_unvaccinated_individuals_in_healthcare_settings.pdf

52 COVID-19 vaccine surveillance report: week 15 (publishing.service.gov.uk) https://doi.org/10.1016/S2213-2600(22)00101-1

70. What the NZ data suggests is that while the vaccinated population is more than twice as likely to get infected, the booster vaccinated are now, at best, half as likely to be admitted to hospital with COVID-19 and, therefore, any benefit of the vaccine has been lost.

71. There is concern that the hospitalisation benefit of vaccination will also deteriorate here in NZ to the point where there is a dis-benefit (negative VE). This is again best seen with UKHSA data where they are experiencing their 4th or 5th wave and >98% of the population has now likely been exposed to COVID-19 (based on serology). What is important to note is the increasingly worse rates of hospitalisation across age groups for the vaccinated relative to unvaccinated (Table below is a montage of Table 12 from reports #3, #7, #13, 2022). Again, and as observed for case rates, this is evidence of vaccine harm.

	Cases reported by specimen date between week 51 2021 (w/e 26/12/21) and week 02 2022 (w/e 16/01/22)		Cases reported by specimen date between week 3 2022 (w/e 23 January 2022) and week 6 2022 (w/e 13 February 2022)		Cases reported by specimen date between week 9 2022 (w/e 6 March 2022) and week 12 2022 (w/e 27 March 2022)	
	Unadjusted rates among persons vaccinated with at least 3 doses (per 100,000)	Unadjusted rates among persons not vaccinated (per 100,000)[1,2]	Unadjusted rates among persons vaccinated with at least 3 doses (per 100,000)	Unadjusted rates among persons not vaccinated (per 100,000)[1,2]	Unadjusted rates among persons vaccinated with at least 3 doses (per 100,000)	Unadjusted rates among persons not vaccinated (per 100,000)[1,2]
Under 18	2,295.7	3,990.1	1,637.8	4,529.9	1,454.0	1,711.7
18-29	3,460.5	3,853.3	3,294.6	1,495.1	3,118.8	941.6
30-39	3,857.1	3,251.7	4,579.1	1,652.1	4,324.7	1,085.6
40-49	4,012.4	2,573.9	4,416.0	1,442.9	3,957.8	955.3
50-59	3,995.9	2,133.3	2,458.4	937.3	3,303.4	779.8
60-69	3,070.0	1,499.8	1,685.2	652.3	2,814.9	572.8
70-79	2,062.8	1,129.7	1,129.6	520.0	2,161.5	532.1
≥80	1,842.6	1,374.8	1,268.0	831.7	2,023.7	775.6

72. There are several possible explanations for negative VE. To understand how negative VE can arise following COVID-19 infection we need to understand a few fundamental principles of immunology.

(i) The human immune system

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

(a) the innate; and

(b) the acquired or adaptive.

74. The latter is associated with memory to invasive or harmful pathogens (microbes) including viruses.

75. Innate immunity is non adaptive and is our first line of defence against any tissue injury and/or pathogenic infection. It does not initially distinguish the nature of the injury. Rather, it treats all injury as infectious and foreign in origin until it determines otherwise[53].

76. 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.

77. 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 (neutralizing) to provide robust and durable "sterilizing" immunity so as to not negate (by outcompeting) the proven benefits of innate low affinity nIgMs.

78. 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 "sterilizing" 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.

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53 https://doi.org/10.1038/nature01320

(j) Antigenic Imprinting and Antibody Dependent Enhancement (ADE)

79. In my opinion, vaccinating 5-11 year olds with the Pfizer vaccine will erode the plasticity and effectiveness of a child's adaptive 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**).[54]

80. 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 version of the virus encountered.

81. Antigenic imprinting would be fine if the SC2 virus was not so highly mutable and undergoing significant evolution as it adapts under vaccinal pressure to its new found host (i.e. us) and undoubtedly to any other potential disease reservoir with which we can exchange the virus.

82. I note that Pfizer's CEO, Albert Bourla, has already acknowledged (before Omicron was discovered) that the Pfizer vaccine would inevitably fail due to the positive selective pressure in promoting vaccinal escape variants because the vaccine fails to provide sterilizing immunity.[55]

83. Immune evasion cannot explain negative VE, it simply describes vaccine failure where VE will trend to zero.

84. OAS has been observed following 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

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54 https://doi.org/10.1016/j.clicom.2021.10.001

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

with SC2.[56] 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 sarbecovirus subgroup.[57] They further show that cross-reactive antigen binding of antibodies between SC1 and SC2 is common but cross neutralization of live virus was rare.[58] 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.

85. In the past two months three papers clearly demonstrate the immune response to new VoCs is driven by specificities induced through prior vaccination. Two of these are in humans with the Pfizer vaccine[59] while the third uses a replicating mRNA vaccine in mice and hamsters.[60] All studies observed the immune response failed to redirect against new epitopes within the receptor binding domain of the Spike protein (S-RBD) mutated in the Omicron VoC. This is the essence of OAS and explains why Omicron BA.1 and BA.2 are immune evading. In short, vaccinated individuals will increasingly mount an unproductive (non-neutralizing) immune response in which vaccinal antibodies that were originally neutralizing for the Wuhan strain of SC2 are increasingly non-neutralizing or worse, enhancing.

86. There are multiple possible explanations as to how negative VE can arise but where I weight one or more of the following to now operate within the vaccinated population.

(a) The first relates to how high levels of unproductive non-neutralizing vaccinal antibodies will out-compete innate nIgMs for the recognition of SC2. That is, the vaccine has rendered an important component of our innate immune system ineffective when compared to the unvaccinated.

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56 https://doi.org/10.1038/s41467-021-23977-1
https://www.biorxiv.org/content/10.1101/2021.05.21.445201v2
https://doi.org/10.1016/j.cell.2021.02.010
https://doi.org/10.3390/life11040298

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

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

59 https://doi.org/10.1101/2022.04.01.486695;
https://doi.org/10.1016/j.cell.2022.01.018

60 https://doi.org/10.1101/2022.01.31.478520;

The importance of nIgMs in contributing to sterilizing immunity and preventing disease following infection is established.[61]

(b) The second is a shift in either the balance between or the concentration of neutralizing and non-neutralizing antibodies, a mechanism known to enhance infection with coronaviruses in an Fc-Receptor and C1q-dependent manner. This form of ADE has now been shown with SC2 [62] and adds to observations seen with the closely related SARS and MERS sarbecoviruses.[63] Thus, ADE should have always been anticipated as flagged by others.[64]

(c) The third is generation of antibodies in response to the vaccine or prior infection that target the spike protein of SC2 and enhance binding to its human host receptor ACE2 to promote infection of target cells in an Fc-Receptor independent manner.

(k) Humoral response to Spike protein promotes infection by Antibody-Dependent Enhancement (ADE)

87. The vast majority of the neutralizing antibodies generated by infection or the Spike-based vaccines are targeted against the S-RBD.[65] Modern medicine has taken advantage of this to clonally select plasma B cells from convalescent patients and harvest the monoclonal antibodies (**mAbs**) produced for therapeutic purposes, ie to neutralize infection. While many of these mAbs were of tremendous benefit in the treatment of patients infected with previous VoCs they are no longer effective or administered for Omicron, such are the immune

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61 https://doi.org/10.3389/fimmu.2020.610300

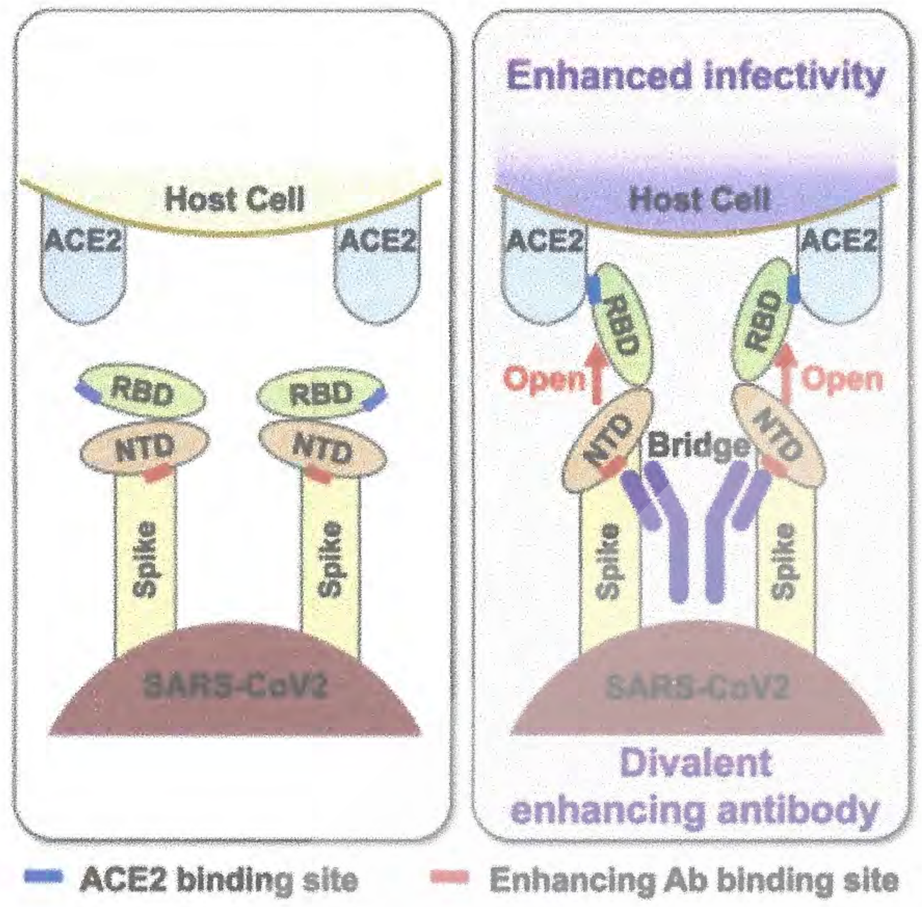
62 https://doi.org/10.1128/spectrum.01553-21
https://doi.org/10.1016/j.cell.2021.06.021
https://doi.org/10.1016/j.isci.2021.103720

63 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
Molecular Mechanism for Antibody-Dependent Enhancement of Coronavirus Entry(asm.org)

64 https://doi.org/10.1016/j.bbrc.2014.07.090
https://doi.org/10.3389/fimmu.2021.640093
https://doi.org/10.1016/bs.ai.2021.08.003
https://doi.org/10.1016/j.vaccine.2021.01.055

65 https://doi.org/10.1016/j.cell.2020.09.037

evading properties of Omicron's S-protein mutations. This alone tells us the vaccine is ineffective.



88. These studies also identified mAbs that augmented coronaviral infection, ie ADE, and were also detectable at high concentrations in severe and acute patients.[66] These "enhancing" mAbs were found to bind a specific region of the N-terminal domain of the Spike protein (S-NTD) known as the N2 loop. The binding of enhancing mAbs promotes infection of target epithelial cells in an Fc-Receptor independent manner (as per 85.c above).

89. I am not a viral immunologist or molecular epidemiologist (not to be confused with a public health epidemiologist) but I hold the view that SC2 gave up virulence and pathogenicity for increased transmissibility. Omicron represents that evolutionary advance. The concern now is that SC2 will take advantage of this increased transmissibility to re-find virulence. In this respect we now have a paper describing the infectivity and virulence of the Delta Omicron recombinant XD (Deltacron).[67] This paper states "This recombinant exhibits immune escape properties similar to Omicron, while its behaviour in mice expressing the human ACE2 receptor is more similar to Delta." That is the disease was worse requiring the mice to be euthanized. Deltacron is essentially Delta that has swapped out its

66 https://doi.org/10.1016/j.cell.2021.05.032
https://doi.org/10.1101/2020.12.31.424729

67 https://doi.org/10.21203/rs.3.rs-1502293/v1

spike protein for Omicron's while ominously preserving the near N-terminal of Delta's S-NTD containing the N1 and N2 loops.

90. Delta was unquestionably a VoC that came with a higher disease burden, especially for the unvaccinated, while the vaccinated were relatively better protected. That advantage will be lost, if not reversed, if Deltacron takes off and displaces Omicron as the predominant VoC.

(l) Is the Booster masking ADE?

91. Waning of humoral responses is normal and necessary to prevent problems arising from immune complex deposition resulting in organ damage but also from causing possible autoimmune attack of our own body. The fact that the vaccine has imprinted an immune response is apparent from numerous global studies showing the rapid "recall" of a humoral response following a booster shot. We can therefore conclude a booster is not needed as the virus should now be that booster. This also suggests that by boosting Ab levels the vaccine is being used as a therapeutic (recall) and not for affinity maturation and immunological memory, the primary purpose of a vaccine. This is, in my opinion, an abuse of our immune system that will lead to long term damage.

92. Pertinent for ADE is the observation that non-neutralizing antibodies or antibodies at sub-neutralizing levels can enhance infection and disease as already observed with closely related sarbecoviruses.[68] This phenomenon is often observed when antibody concentrations decrease as a result of waning immunity; an antibody may neutralize potently at high concentrations but cause enhancement of infection at sub-neutralizing concentrations. This now raises the distinct possibility that the booster is not required for immune maturation but to re-establish levels of circulating antibodies directed at neutralizing SC2 above a threshold where they no longer enhance infection. I consider this explanation a better fit for the population data.

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68 https://doi.org/10.1038/s41564-020-00789-5
https://dx.doi.org/10.1016%2Fbs.ai.2021.08.003
https://doi.org/10.1016/j.cyto.2020.155256
https://doi.org/10.1016/j.bbrc.2014.07.090
https://doi:10.1128/spectrum.01553-21

93. It was also observed that the balance between neutralizing and ADE-facilitating antibodies in COVID-19 vaccinated people favoured neutralization of the original Wuhan strain, but not the Delta strain due their reduced spike protein binding affinity, whereas ADE facilitating NTD-antibodies displayed increased binding affinity.[69] The ADE effect was concentration dependent as expected for FcR-dependent ADE.[70] This balance phenomenon between neutralizing and ADE-facilitating antibodies in vaccinated people could explain UKHSA data between Reports 36 and >42 (Delta) where 2-dose vaccinated case rates increased as the population's immunity waned.

94. To understand how misguided our approach has been and the immune damage we have caused we need only look at the increased rates of infection (and hospitalization) in the vaccinated vs those of the unvaccinated[71] and that Northern Hemisphere nations are failing to resolve viral infections. In short, mass vaccination with a non-sterilizing vaccine has destroyed the 3-wave paradigm by which viral respiratory infections transition to endemicity.[72]

95. In my opinion, the reckless and indiscriminate use of a non-sterilizing vaccine has driven viral evolution to give rise to immune evading variants. We also now have population evidence that SC2 antibody specificity, breadth and maturation are affected by antigenic imprinting skewing the serological response toward the Wuhan strain of rather than variant antigens (OAS). We also have a confirmed mechanism for FcR-independent ADE which could act independently or in concert with FcR-dependent mechanisms to enhance infection and explain negative VE that may lead to enhanced disease as new VoCs arise (VAED). In short, the virus in a vaccinated host now finds itself in a supportive non-hostile immune environment. This explains why we are seeing non-resolving waves of continuous viral infection in other countries.

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69 https://doi.org/10.1016/j.jinf.2021.08.010

70 https://doi:10.1128/spectrum.01553-21

71 Worldwide Bayesian Causal Impact Analysis of Vaccine Administration on Deaths and Cases Associated with COVID-19: A BigData Analysis of 145 Countries (vector-news.github.io)

72 https://www.cdc.gov/flu/pandemic-resources/1918-commemoration/three-waves.htm
https://doi.org/10.1101/2021.12.20.21267966

Conclusions

96. My conclusions are at paragraphs 8 to 18, above.

Sworn at) Upper Hutt

this 29th day of April)

2022 before me)

Michael John Fokker, JP
#4114
UPPER HUTT
Justice of the Peace for New Zealand

Simon Brown

This is the exhibit marked "**SBB-1**" referred to in the annexed affidavit of **Simon Bradley Brown** sworn at Upper Hutt this 29th day of April 2022 before me:

"SBB-1"

Curriculum vitae

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. I was also active in providing study options for second year medical students. 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

Patent:

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

Peer-reviewed publications (post 2000):

1. Taylor HB, Liepe J, Barthen C, Bugeon L, Huvet M, Kirk PD, Brown SB, Lamb JR, Stumpf MP, Dallman MJ. 213. P38 and JNK have opposing effects on persistence of in vivo leukocyte migration in zebrafish. **Immunol Cell Biol.** 91(1):60-9.
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.

Commentary & Opinion:

- 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.
- Brown SB, Vernon-Wilson EF. (2005). Promoting apoptosis in disease management: A Panacea or Trojan horse? ***Curr. Opin. Pharmacol.*** 5:444-8.