REPORT ON CORONAVIRUS AND IMMIGRATION DETENTION

Professor Richard Coker MB BS, MSc, MD, FRCP, FFPH

Introduction

I have been asked by Duncan Lewis Solicitors to provide my expert opinion on the risks posed by COVID-19 to immigration detainees in the United Kingdom.

My qualifications and experience

I am Emeritus Professor of Public Health at The London School of Hygiene and Tropical Medicine (LSHTM). I trained in medicine at St. Mary's Hospital Medical School (now Imperial College London) and qualified in 1984. I gained membership, and subsequently fellowship of the Royal College of Physicians (FRCP) and subsequently was appointed fellow of the Faculty of Public Health RCP (FFPH). In 1997, after having been appointed a consultant physician to St. Mary's Hospital, London, where I was responsible for HIV and tuberculosis care, I expanded my research into communicable diseases and health systems' response to them. Through a Harkness fellowship, I analysed the social causes and response to the epidemic of drug-resistant tuberculosis in New York City. My book (From Chaos to Coercion) and my Medical Doctorate (MD) resulted from this work. I subsequently moved to LSHTM, gaining a Masters in Public Health (MSc) in 1998, and where I was later appointed professor of Public Health, and Honorary Consultant to Public Health England. I have advised UK Cabinet Office, World Health Organization, World Bank, and many governments around the world. My earlier research addressed the spread of HIV and tuberculosis within the Soviet Union including through prisons and pre-detention trial centres. Over the past 15 years my research has focused on pandemic preparedness drawing upon the following disciplines: medicine, epidemiology, mathematical modelling, sociology, policy analysis. I have published several books, multiple book chapters, and more than 250 peer-reviewed academic papers.

My instructions

I have been instructed to address the following questions:

1. What is COVID-19 and how is it spread between individuals?

2. What are the symptoms of COVID-19? Please explain what kind of symptoms a person who is severely affected by the infection would suffer, and the likely duration of such symptoms? Please also explain whether a person who is severely affected by the infection but does not die, is at risk of suffering future health complications.

3. How long may a person who has contracted COVID-19 be asymptomatic?

4. Please explain the accuracy of tests administered to screen people for COVID-19?

5. What underlying conditions (including age and pre-existing health conditions) may increase the risk of an individual (i) contracting COVID-19; (ii) suffering severe symptoms; (iii) dying?

6. What is the current rate of (i) infection; (ii) suffering severe symptoms; and (iii) mortality, within the community?

7. What is the likelihood of COVID-19 entering an immigration detention centre?

8. What is the likely rate of (i) infection; (ii) suffering severe symptoms; and (iii) mortality, within a detention centre?

9. Please explain whether the conditions in immigration detention centres described above (including for example the regular transfer of detainees in and out of detention centres; standards of hygiene; ventilation; the 'lock-in' regime; and limited space) may increase the risk of a widespread outbreak of COVID-19. Please explain whether any other factors may increase the likely infection rate in a detention centre.

10. What practical measures would be necessary within an immigration detention centre to (i) minimise the risk of individuals who are currently detained contracting COVID-19; (ii) properly isolate and contain COVID-19 if an individual (or individuals) contract the virus?

11. Please explain the concept of cluster amplification, and the impact that the spread of COVID-19 to the immigration detention estate may have on the spread of the virus amongst the UK population.

12. Please express a view on the urgency with which preventative measures should be taken to reduce the risk of COVID-19 entering the immigration detention estate or spreading amongst detainees.

<u>Source materials</u>

In formulating my opinion I draw upon my experience as set out in section 2 above. I have also relied on peer-reviewed academic papers from medical, epidemiology and public health journals including The Lancet, New England Journal of Medicine, BMC Medicine; Academic Centres' reports such as Imperial College, Johns Hopkins, London School of Hygiene and Tropical Medicine, US National Institutes of Health (NIH); World Health Organization; public health institutions such as Public Health England, US Centers for Disease Control and Prevention, European Centre for Disease Prevention and Control. Sources are referenced within the body of the report where appropriate.

<u>The questions asked</u>

What is COVID-19 and how is it spread between individuals?

Coronaviruses are a family of RNA viruses which typically affect the respiratory tract of birds and mammals, including humans. RNA viruses have the propensity to mutate more rapidly than DNA viruses and thus human immunity may not be lifelong even after having been infected (or, where possible, vaccinated). Seven coronaviruses are known to cause human disease, four of which are mild: viruses 229E, OC43, NL63 and HKU1. Three of the coronaviruses can have more serious outcomes in people, and those diseases are SARS (severe acute respiratory syndrome) which emerged in late 2002 and disappeared by 2004; MERS (Middle East respiratory syndrome), which emerged in 2012 and remains in circulation in camels. A novel coronavirus, probably arising from bats (similar to SARS-CoV-2 and MERS-CoV) in a wet market, COVID-19, emerged in December 2019 from China. A global effort is under way to contain its spread. COVID-19 is caused by the coronavirus known as SARS-CoV-2.

Person-to-person spread of SARS-CoV-2 is thought to occur mainly via respiratory droplets, resembling the spread of influenza. With droplet transmission, virus released in the respiratory secretions when a person with infection coughs, sneezes, or talks can infect another person if

it makes direct contact with the mucous membranes. Infection can also occur if a person touches an infected surface and then touches his or her eyes, nose, or mouth. There is evidence that SARS-CoV-2 can be spread by those without symptoms.[i] The virus can remain viable in suspended aerosols up to 3 hours post aerosolization, meaning that spread within congregate settings is a substantial risk. Spread can also occur vertically, from mother to her baby, though not through the intrauterine route.[ii] The faecal-oral route of transmission is potentially important, though less well demonstrated compared to the respiratory route of transmission. This route of transmission has implications in areas with poor sanitation.[iii] The transmission of SARS-CoV-2 as a fomite, that is, the spread of a virus through an object - door handle, door-bell, respirators, also plays a role in spreading the virus. As with the faecal-oral route, poor sanitation, again, is thus likely to play a part in transmission.[iv] The virus can remain viable on surfaces for days in the absence of appropriate cleaning.[v]

What are the symptoms of COVID-19? Please explain what kind of symptoms a person who is severely affected by the infection would suffer, and the likely duration of such symptoms? Please also explain whether a person who is severely affected by the infection but does not die, is at risk of suffering future health complications.

Most cases of COVID-19 are not severe. But many patients with COVID-19 suffer critical illness.[vi] [vii] [viii] For example, in a report from the Chinese Center for Disease Control and Prevention that included approximately 44,500 confirmed infections with an estimation of disease severity[ix]:

- ●Mild (no or mild pneumonia) was reported in 81 percent.
- ●Severe disease (eg, with dyspnoea [shortness of breath], hypoxia [low oxygen levels in the blood], or >50 percent lung involvement on imaging within 24 to 48 hours) was reported in 14 percent. In my clinical experience, a patient would feel like they were drowning.
- ●Critical disease (eg, with respiratory failure [that is breathing difficulties so severe it results in low oxygen and high carbon dioxide levels in the blood], shock [blood pressure so low that the perfusion of organs by blood begins to fail], or multiorgan dysfunction) was reported in 5 percent. In my experience, patients with critical disease are likely be unconscious or semi-conscious.

- ●The overall case fatality rate was 2.3 percent; no deaths were reported among noncritical cases.
- No deaths occurred in the group aged 9 years and younger, but cases in those aged 70 to 79 years had an 8.0% case fatality rate (CFR) and cases in those aged 80 years and older had a 14.8% CFR.
- CFR was elevated among those with pre-existing comorbidities; 10.5% for cardiovascular disease; 7.3% for diabetes; 6.3% for chronic respiratory disease; 6.0% for hypertension; and 5.6% for cancer.

The most common symptoms displayed by COVID-19 on admission to hospital are fever and cough, followed by sputum production and fatigue, and dyspnoea (shortness of breath). Less common symptoms include myalgia (muscle aches), fatigue, diarrhoea, and nausea and vomiting.[x] The time from illness onset to discharge from hospital or death averaged, in the large Wuhan cohort studied, about 20 days.

According to a joint World Health Organization (WHO)-China fact-finding mission, the CFR ranged from 5.8 percent in Wuhan to 0.7 percent in the rest of China.[xi] Considerable uncertainty, however, remains around estimates of case fatality rate and they may vary considerably by context. Some estimates suggest a CFR for COVID-19 of about 0·3–1%, which is higher than the order of 0·1% CFR for a moderate influenza A season.[xii] Estimates of up to 12% have been reported, probably associated with early rapid spread and the breakdown of, or lack of access to, health care services.[xiii]

Severe disease necessitating hospital admission is associated with comorbidity, with hypertension being the most common, followed by diabetes, and coronary heart disease, chronic obstructive airways diseases (this refers to chronic asthma, chronic bronchitis, and emphysema), carcinoma (cancer), and chronic kidney disease. Other comorbidities are also associated with severe disease and death but, presumably because the prevalence of these diseases in the population is low, the statistical association with specific diseases is lost in the epidemiological 'noise' .[xiv]

Most of the fatal cases have occurred in patients with advanced age or underlying medical comorbidities. [xv] Accepting the probability of bias, death rates in those over 80 years of age may be more than 20%, and those under 50 years old orders of magnitude at less risk, in the region of 0.2-1.0%. The background prevalence of hypertension in China is about 23%.[xvi] In

patients with severe COVID-19, the prevalence of hypertension was 30%. COVID-19 patients with hypertension are around twice as likely to die. The reasons for this are unclear. Hypertension is seldom a risk factor for death with other infectious diseases. One plausible explanation is that the association is not so much with hypertension, but an association with a family of drugs used to treat hypertension, angiotensin converting enzyme inhibitors (ACE inhibitors).[xvii] It has been proposed that these drugs facilitate viral entry into cells. Angiotensin converting enzyme 2 (ACE2) cell receptors have been shown to be the entry point into human cells for some coronaviruses, including SARS-CoV-2. ACE2 has direct effects on cardiac function and is expressed predominantly in cells lining blood vessels of the heart and kidneys. This may have implications for long-term function of these organs but this is conjecture. The long-term effects of infection of these tissues remains unknown at present.

Diabetes increases the risk of death from COVID-19 patients with severe disease about two-fold. Coronary heart disease may be associated with a much higher risk of death, in the order of 20 times. Though the order of magnitude of effect is robust, the precision of the estimate should be treated with caution. The reason for this is that the prevalence of coronary heart disease is much lower than hypertension and diabetes. Similarly, though the risk of death arising between COVID-19 and chronic obstructive airways disease is very high (in the order of a seven-fold increased risk), the precision around this number is limited.

The long-term clinical sequelae following COVID-19 remain uncertain given the emergence of the disease is so recent. There are anecdotal reports of residual lung fibrosis after severe pneumonia and, presumably, impaired lung function.

How long may a person who has contracted COVID-19 be asymptomatic?
This is an important question. It is part of a broader question: what elements of transmission dynamics are important in the spread of COVID-19? As COVID-19 is a newly identified pathogen, there is no known pre-existing immunity in humans. Based on the epidemiologic characteristics observed, everyone is assumed to be susceptible, although there may be risk factors increasing susceptibility to infection that are unknown at present.

COVID-19 has developed into a pandemic, with small chains of transmission in some countries and large chains resulting in extensive spread in many countries, such as Italy, France, Spain, the UK, Germany, Iran, South Korea, and Japan (the number of countries that can be added to

this list grows daily). This is probably the case in the US too but diagnostic capacity has been limited and so the epidemiological progression more difficult to discern.

The course of an epidemic is defined by a series of key factors, some of which are poorly understood at present for COVID-19. The **basic reproduction number (R0),** which defines the mean number of secondary cases generated by one primary case when the population is largely susceptible to infection, determines the overall number of people who are likely to be infected, or more precisely the area under the epidemic curve. For an epidemic to take hold, the value of R0 must be greater than 1. Estimates of R0 vary but probably lie around 2.5. At this level, left unchecked, about 60% of a population will become infected. However, uncertainty surrounds this figure for a number of reasons:

1. We are uncertain about transmission in children
2. Some communities are remote and unlikely to be exposed
3. Voluntary social distancing by individuals and communities has an impact
4. Mitigation efforts, such as the measures put in place in China, greatly reduce transmission

As an epidemic progresses, the effective reproduction number (R) declines until it falls below 1 when the epidemic peaks and then decays, either due to the exhaustion of people susceptible to infection or the impact of control measures. This has implications for the population makeup of closed community settings. For example, if children, who seldom have disease and are often asymptomatic, but are effective spreaders of infection, then communities with many children who have close contact with many people will witness rapid spread. If communities are truly closed to others, who are infected, entering, then no exposure follows. To put a closed population at risk of exposure need not be the result of a symptomatic index person entering the population. All that is needed is someone able to transmit the virus. This is where transmission from persons who are asymptomatically infected becomes important.

Knowledge of the **incubation period** of infectious diseases is crucial to our epidemiological understanding and the design of appropriate prevention and control policies. The incubation period of an infectious disease is the time interval between the exposure to a disease-causing infectious agent and the onset of symptomatic (clinical) disease. The incubation period of a disease can be very variable among individuals and a single number, such as the mean or median incubation period, does not reveal the significant heterogeneity in incubation periods

in a population. Estimating the distribution of incubation periods is important for a multitude of reasons including modelling the current and future scope of epidemics and evaluating disease control strategies. Critical to understanding the impact of incubation periods and the progress of an epidemic (or pandemic) is whether or not an infected person is contagious (i.e. is shedding virus) during the incubation period. This depends on the virus. For example, Ebola virus infected patients do not pass the virus on to others during the incubation period.

These transmission parameters reported early from Wuhan probably declined somewhat during the early weeks of the outbreak in Wuhan as social distancing measures were adopted,[xviii] but still offer important insights into the transmission dynamics of the virus.

The incubation period for COVID-19 is about 5–6 days, varying from 2 to 14 days. Outliers up to 27 days have been reported.

Epidemiological and virological analyses suggest there might be considerable **asymptomatic (or presymptomatic) infectiousness**. Viral shedding, and so presumed infectiousness, appear to be high soon after symptom onset compared with later in the illness.[xix] This raises the possibility that transmission might be more likely in the earlier stage of infection, but additional data are needed to confirm this hypothesis. Transmission of SARS-CoV-2 from asymptomatic individuals (or individuals within the incubation period) has also been described.[xx] In one study, peak viraemia seems to be at the end of the incubation period, pointing to the possibility that viraemia might be high enough to trigger transmission for 1–2 days before onset of symptoms.[xxi] However, the full extent to which this occurs is unclear. Mathematical models suggest that, the greater transmission from asymptomatic persons, the higher the R0 and the greater the challenge of containing or mitigating the disease spread.

An important uncertainty is whether there are a large number of asymptomatic cases of COVID-19. Estimates suggest that about 80% of people with COVID-19 have mild or asymptomatic disease, 14% have severe disease, and 6% are critically ill,[xxii] implying that symptom-based control (or screening before entry to unexposed communities) is unlikely to be effective.

Important questions remain around whether patients can become re-infected, what the implications for disease and further transmission are, and what immunity to SARS-CoV-2 exists, and if it does what the duration is.

The speed of the initial spread of the epidemic, its **doubling time**, or the related serial interval (the mean time it takes for an infected person to pass on the infection to others), and the likely duration of the epidemic are determined by factors such as the length of time from infection to when a person is infectious to others and the mean duration of infectiousness. For COVID-19, the doubling time is estimated at 4·4–7·5 days.

Please explain the accuracy of tests administered to screen people for COVID-19

To date the identification of individuals with COVID-19, is dependent upon a history of exposure (whether through travel where the incidence or prevalence of disease is high, or a history of contact with a person infected), an approach using clinical evaluation (an assessment of symptoms, a screening of signs such as fever), or a diagnostic test such a polymerase chain reaction (PCR). No antibody tests, which would traditionally offer a sensitive and specific approach to screening and diagnosis are yet available.

Briefly, one concept needs to be understood when considering any screening test. I say briefly because the literature on screening is lengthy and in the UK there are highly defined criteria for the introduction of screening tests systematically.[xxiii] These are not considered here because the purpose of screening generally is not applicable, on the assumption that the intention of 'screening' here is to detect cases of COVID-19 before they are introduced to, what is assumed to be, an immunologically naïve population in a detention centre.

Under these circumstances we are aiming to detect cases with accuracy before they are introduced. The **sensitivity** of a screening tool is vitally important. The sensitivity of a test (also called the true positive rate) is defined as the proportion of people with the disease who will have a positive result. In other words, a highly sensitive test is one that correctly identifies patients with a disease. Less important under my assumption is a screening tool's specificity, which is the ability of the test to correctly identify those without the disease (true negative rate).

So what is the sensitivity of each approach to screening entrants to a detention centre likely to be? That is, is it likely that entrants with infection will enter having disease that has been undetected?

1. History of exposure
    a. The number of cases being reported is increasing exponentially, and the number of countries reporting cases is changing daily. One month ago, Wuhan, China was the centre of an epidemic. Today case numbers and deaths reported daily (and per capita) from European countries are higher than Wuhan or anywhere in China. South Korea and Iran have reported very substantial numbers of cases. By next week it seems likely France, Spain and the UK will be reporting very substantial numbers of cases. Thus travel history is currently, and likely in the future, will become, a poor guide to exposure and infection. The lag between infection and infectiousness also challenges screening through this method. We are witnessing cases now that became infected several days ago. Moreover, diagnostic capacity varies in countries meaning absolute numbers of cases reported do not reflect the real incidence of infection.
    b. Almost all countries are reporting domestic community chains of transmission. And with transmission from asymptomatic and mildly symptomatic cases being widespread, the value of identifying contact with cases becomes less sensitive as a screening tool. This problem is compounded where diagnostic tests have been in limited supply.

2. Clinical evaluation
    a. Symptoms are a poor predictor of COVID-19. Patients with seasonal influenza (which is prevalent in northern hemisphere currently) and the common cold express the same symptoms. Patients with pneumonia from a whole variety of causes also present with similar symptoms.
    b. Signs such as fever also show very low sensitivity. To re-iterate, persons with COVID-19 can transmit virus when they are asymptomatic. Moreover, antipyretics such a paracetamol can mask such signs.

3. Diagnostic test

a. The gold standard screening test in the diagnosis of COVID-19 is Reverse Transcription Polymerase Chain Reaction (RT-PCR). Even in excellent laboratories this test can be challenging to operationalise, witness the challenges US CDC have had. The sensitivity of RT-PCR varies depending on the quality of the laboratory conducting the test, the severity of disease, where the sample is taken from (SARS-CoV-2 has a predilection for the lower respiratory tract; most samples are taken from the upper respiratory tract), whether the test is performed once or twice, and whether the test is combined with other diagnostic tools. A study from China, which is probably broadly representative of well-functioning laboratories, concluded that the sensitivity of a single pharyngeal swab subjected to RT-PCR had a 78% sensitivity in hospitalised patients.[xxiv] Thus, 20% of cases later shown to have COVID-19, were negative on their first pharyngeal swab. And these patients were hospitalised, and presumably had a high viraemic relative to asymptomatic and relatively well cases. (Conducting a second swab increased the sensitivity to 86%, and a combination of chest CT scan with RT-PCR resulted in a sensitivity of 92%). The sensitivity of such screening tests in the asymptomatic population is unknown but likely lower.

In summary, there are no screening tests that offer high sensitivity to detect individuals with COVID-19. This situation may change if new screening tools are developed.

What underlying conditions (including age and pre-existing health conditions) may increase the risk of an individual (i) contracting COVID-19; (ii) suffering severe symptoms; (iii) dying?

I have previously addressed what is known about the current rate of (i) infection, (ii) suffering severe symptoms, and (iii) mortality within the community? Here, I will focus on the numbers of cases that have been reported in the UK.

The WHO's situation report dated 15th March 2020 shows that the UK to date has 1,144 confirmed cases, with 342 new cases, and 21 deaths.[xxv] The pandemic is following the same epidemiological trajectory as Italy, and is in an exponential growth curve. It is important to remember these cases are known. The underlying real epidemic which includes undiagnosed

cases is very likely to be orders of magnitude higher. Thus, the true rate of infection is unknown but substantial and will grow in coming days and weeks.

What is the likelihood of COVID-19 entering an immigration detention centre?

The risk of an index case entering an immigration detention centre is dependent on the characteristics of both the individual and their likely exposure and infection. As the pandemic evolves worldwide the risks of any individual in the wider community increase. The risk any potential index person entering a centre poses from COVID-19 can be framed by traditional epidemiological methods.

What is their **susceptibility** to infection? Have they previously been infected and recovered and are now immune? To date it appears everyone is equally susceptible to infection.

What is their likely history of **exposure**? Have they come from a community with a high prevalence of COVID-19? For example, have they been exposed because they have come from China, Iran, or Lombardy, Italy? It is important to understand two issues. First, that the risk of COVID-19 exposure depends not on the prevalence of disease now in any community visited, but the prevalence of disease at the time of exposure and infection, that is 5-6 days ago (the incubation period). Second, a history of exposure to a high prevalence community is important in any clinical assessment but may not be important as a population screening tool. The sensitivity of the question is of very limited importance.

What is the likelihood of an index case having developed **infection** upon exposure? This is unknown because community wide surveillance is challenging with the tools that exist. Those that do develop infection may express different levels of **infectiousness**. The potential to transmit, the infectiousness of individuals, remains uncertain. It is unknown whether so-called 'super-spreaders' exist.[xxvi]

What is the likely rate of (i) infection; (ii) suffering severe symptoms; and (iii) mortality, within a detention centre?

Assuming a detention centre holds a population of immunologically naïve individuals and an index case with COVID-19 who transmits the virus usually, then we expect an uninterrupted

chain of transmission and a traditional, natural, epidemic curve to emerge. As noted above, the chance of a COVID-19 case exposing a population obviously increases the more people a closed population is exposed to. If a detention centre has multiple visitors, short -term detainees, and numerous staff, all of whom have varying risks of infection reflecting their wider, outside exposure, then the risk of introduction increases substantially. Closed communities with no exposure are, in essence, quarantine centres. Detention centres are clearly not. An unfolding COVID-19 epidemic in a detention centre would, in a grim sense, represent a natural experiment. To date, wherever COVID-19 has emerged, mitigation steps such as isolation of cases, quarantine of contacts, and social distancing measures have been implemented to curtail chains of transmission. Overcrowding, unsanitary conditions, poor ventilation in a detention centre would likely increase the speed with which an epidemic unfolded even if the number of cases cumulatively remained unchanged. Poor access to health care facilities, slow procedures to diagnose, isolate, and treat patients, or quarantine contacts would further reduce the time to peak incidence. Detainees at risk of COVID-19 should, in my opinion, have access to diagnostic facilities that include haematology, biochemistry and virology laboratory services, and radiology services that include chest X-ray and CT scan capabilities. Therapeutic support for cases should include, at a minimum, appropriate pharmaceuticals and respiratory support. The full complement of support, including ventilation, available in intensive care units may well be needed for severe cases. Access to these services should be timely. We have seen the explosive transmission of COVID-19 in congregate settings like cruise ships. Hours matter if transmission is to be stopped. Without timely and effective containment steps, a detention centre would result in transmission and climbing of cases until a peak was reached in terms of new cases occurring on a daily basis. Then, because so many cases were infected, R0 falls to below 1 and the rate of new cases each day declines. '**Herd immunity'** would then be achieved such that the spread of COVID-19 would be limited within the detention centre population because a sufficiently high proportion of individuals had become immune through infection. Herd immunity is normally achieved through vaccination such that individuals' immunity is enhanced. There is no vaccine for COVID-19. Modelling suggest that approximately 60% of the population would become infected, though context is important.

As noted above, the clinical consequences of the introduction of COVID-19 depend upon the characteristics of detainees, their age profile, and the presence of co-morbidities.

Please explain whether the conditions in immigration detention centres described above (including for example the regular transfer of detainees in and out of detention centres; standards of hygiene; ventilation; the 'lock-in' regime; and limited space) may increase the risk of a widespread outbreak of COVID-19. Please explain whether any other factors may increase the likely infection rate in a detention centre.

The experience of COVID-19 on cruise ships suggests that a scenario where 60% of detainees become infected is plausible and credible. In my expert judgement, this would occur rapidly, within the order of four weeks, if the conditions referred to in the Shaw report set out in my letter of instruction remain the case. Many of the elements that facilitate spread on cruise ships which have had transmission of COVID-19, such as poor ventilation, challenging sanitation conditions, limited space and passengers being confined to their cabins for lengthy periods are the same as exist in immigration detention centres.

What practical measures would be necessary within an immigration detention centre to (i) minimise the risk of individuals who are currently detained contracting COVID-19; (ii) properly isolate and contain COVID-19 if an individual (or individuals) contract the virus?

Returning to epidemiological and public health basics, I would advise the following measures be taken:

1. To reduce the risk of detainees being exposed to a case of COVID-19

    a. Ensure any detainee with symptoms suggestive of COVID-19 receives urgent medical attention, and isolation until determined to be free of the disease. It is critically important to ensure people already detained do not have COVID-19 and thus expose others who are immunologically naïve.

    b. Reduce the number and frequency of transfers in to the detention, reduce the number of visitors, reduce the changeover of staff.

2. To contain the COVID-19 should transmission result in a case in a detention centre emerge:

    a. If a case of COVID-19 enters the detention centre they should be isolated effectively, their contacts in the centre be quarantined immediately.

b. The case should receive immediate care, and those charged with his/her care should be trained in the use of personal protective equipment (PPE), such as fitting of masks, gloves, gowns, and eye protection.

c. Those charged with looking after contacts who have been quarantined should also be trained in the use of PPE.

d. As noted, poor ventilation and unsanitary conditions facilitate the spread of the virus. Such conditions should be improved if they exist. The following list is from WHO's guidance on sanitation and COVID-19[xxvii]:

    i. managing excreta (faeces and urine) safely, including ensuring that no one comes into contact with it and that it is treated and disposed of correctly;

    ii. engaging in frequent hand hygiene using appropriate techniques;

    iii. implementing regular cleaning and disinfection practices. (As a matter of common sense, in my expert opinion, I would expect cleaning and disinfection practices to be conducted at a minimum of every two days);

    iv. safely managing health care waste;

    v. regularly laundering bedsheets and clothing. (In my opinion, at least once a week);

    vi. providing adequate and accessible toilets (including separate facilities for confirmed and suspected cases of COVID-19 infection).

e. Overcrowding in congregate settings should be avoided if possible. The virus spreads in congregate settings and, where poor sanitation, poor ventilation, and overcrowding exist the virus can overwhelm a population, particularly a population with co-morbidities or that is elderly. Thus, where feasible, congregate settings should be limited to only individuals for whom there is no alternative. This is especially important for those at increased risk of developing severe disease or death. If detention is unnecessary it should be relaxed. This should be done *before* the virus has a chance to enter a detention centre. Preventing an outbreak is much easier than controlling an outbreak. The risk to any individual is likely to be much lower in the wider community, where social distancing is feasible, than in a place that is, by definition, a congregate setting.

Please explain the concept of cluster amplification, and the impact that the spread of COVID-19 to the immigration detention estate may have on the spread of the virus amongst the UK population.

Prisons and centres of detention are well-recognised 'epidemiological pumps'. For example, prisons in the former Soviet Union and the United States in the 1990s were settings for explosive outbreaks of multidrug resistant tuberculosis and HIV that spread beyond prisons to transmit to non-prison populations. Congregate settings, notably in hospitals, were the driving context for the spread of SARS in 2003.[xxviii] The outbreak on the Diamond Princess cruise ship showed the potential for transmission and disease in a closed setting. The authors of an analysis concluded 'Unless strict infection management and control are taken, our findings indicate the potential of COVID-19 to cause greater outbreak on the ship.' [xxix] Analysis of COVID-19 outbreaks seeding wider community transmission supports this assertion.[xxx] For example, the ski chalet-associated cluster in France and the church- and hospital-associated clusters in South Korea were preludes to widespread transmission. Moreover, since gathering in closed environments was prohibited in the wake of the rapid spread of the disease in China, the incidence of disease has declined. Reduction of unnecessary close contact in closed environments may help prevent large case clusters and superspreading events that seed beyond those confines.

Please express a view on the urgency with which preventative measures should be taken to reduce the risk of COVID-19 entering the immigration detention estate or spreading amongst detainees.

This pandemic is the most serious public health crisis the world has faced in more than a generation. Case numbers are climbing exponentially around the world. Health systems will likely be stretched close to, or beyond, breaking point. Mitigation matters for overall public health. Reducing the potential for settings such as detention centres to act as epidemiological pumps is an urgent consideration. Beyond the public health imperative, there is an imperative to prevent any detainees being exposed, reduce the risk of spread from detainees developing COVID-19 to other detainees (and others), and ensuring those that who do succumb receive medical care in a timely and appropriate manner to reduce suffering and offer them the best chance of survival. Action to address these issues in detention centres is needed extremely urgently.

Declaration

The contents of this report are true to the best of my knowledge and belief. I understand that in preparing this report I have an overriding duty to the Court, as defined in the *Ikarian Reefer* case and I confirm that I have complied with this duty. Although based in Bangkok, I would be prepared to attend the Court via videoconference to give evidence if required.

17th March 2020

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[i] https://jamanetwork.com/journals/jama/article-abstract/2762028

[ii] https://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736(20)30360-3.pdf

[iii] https://www.thelancet.com/journals/langas/article/PIIS2468-1253(20)30048-0/fulltext

[iv] https://www.preprints.org/manuscript/202002.0283/v1

[v] https://www.medrxiv.org/content/10.1101/2020.03.09.20033217v1.full.pdf

[vi] https://www.ncbi.nlm.nih.gov/pubmed?term=31986261

[vii] https://www.ncbi.nlm.nih.gov/pubmed?term=32053579

[viii] https://www.ncbi.nlm.nih.gov/pubmed?term=31986264

[ix] https://jamanetwork.com/journals/jama/fullarticle/2762130

[x] https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(20)30566-3/fulltext#seccestitle150

[xi] https://www.who.int/docs/default-source/coronaviruse/who-china-joint-mission-on-covid-19-final-report.pdf

[xii] https://www.thelancet.com/action/showPdf?pii=S0140-6736%2820%2930567-5

[xiii] https://wwwnc.cdc.gov/eid/article/26/6/20-0233_article

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