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**News Coronavirus**

**Low-income economies**

**An uneven pandemic**

Coronavirus will play out very differently in the world’s poorest nations

Adam Vaughan

THE coronavirus may prove disastrous for the world’s poorest people, including those living in slums and refugee camps.

Cases were slower to appear in low-income economy countries, but as New Scientist went to press, almost nowhere had escaped the pandemic. Pakistan is one of the worst hit countries in south Asia, with more than 3000 cases as of 6 April and troops deployed across cities to enforce a national lockdown. Elsewhere, Haiti, the poorest country in the western hemisphere, has reported 21 cases.

In Africa, most cases have been in relatively affluent South Africa and Egypt, but other countries are seeing rises too. Burkina Faso now has more than 300 cases, Senegal 219 and Ghana 205. Across the continent, there are now more than 9000 cases.

The impact of the virus in many low-income economies is likely to be very different to richer ones such as the UK, says Azra Ghani at Imperial College London.

Demographics are one big difference. The world’s poorest typically live in households containing more people, with all generations living together in daily contact, in contrast to countries like the UK where older people are to some extent already socially distanced from younger ones.

"**Uganda has 0.1 intensive care unit beds per 100,000 people, versus 34.7 in the US**"

As a result, infections are likely to be spread more evenly across all age groups. "That in a sense makes everybody more at risk," says Ghani.

However, as covid-19 seems to hit older people hardest and low-income economies have much younger populations, death rates may be lower, she says. "We’d expect more infections in low-income settings but there’d be less severe cases."

Most of the data we have on the virus is coming from countries like China, Italy and the US. That means we simply don’t know how much the mitigating effect of a younger population in lower income economies will be offset by populations being more malnourished and already handling other diseases, such as malaria, HIV and TB, says Ghani.

In Latin America, countries will have to deal with other overlapping epidemics, including dengue and measles related to migration out of crisis-hit Venezuela, says Alfonso Rodriguez-Morales at the Colombian Association of Infectious Diseases.

In Africa, testing rates are rising and are now in the tens of thousands, says Kevin Marsh at the African Academy of Sciences, up from around 400 three weeks ago. But he says information is generally scarce.

**Ventilation not an option**

Treatment will also be different in much of the continent, says Marsh, because ventilation is usually not an option. Uganda has 0.1 intensive care unit beds per 100,000 people, compared with 34.7 in the US, for example.

The prospect of ventilator manufacturing being scaled up in six weeks or hospitals being rapidly built, as has been done in some countries, is unrealistic, he says, so more people, mostly older, will die at home.

Ghani is concerned that the impact of the coronavirus on healthcare in low-income economies will divert resources away from other deadly diseases. She is already aware of malaria bed nets not being delivered in some countries as a result of the crisis, for example. Previous epidemics, such as the Ebola outbreak in West Africa between 2014 and 2016, killed many people indirectly this way.

Lockdowns in low-income economies should cut transmission as they have in higher income ones. But in practical terms, shielding the oldest and most vulnerable will be “very difficult”, says Ghani, due to a lack of space in homes. Low-income economies can also ill afford such stringent shutdowns.

“Extreme population-wide social distancing and travel restrictions, if sustained over a long period, could be very harmful for fragile, export-dependent economies and stretch livelihoods beyond people’s coping ability,” said Francesco Checchi at London School of Hygiene and Tropical Medicine, writing in a blog post.

Some of those people will be the cleaners and security guards commuting on packed minibuses from informal settlements. This week, Dharavi, a slum in Mumbai, India, that is home to more than a million people, reported its first death linked to the coronavirus.

Between 900 million and a billion people are estimated to live in such informal settlements, often in high-density areas. Typically, three to five people share a room, with families sharing one toilet and, in some cases, a water tap.

“Isolation is virtually impossible in those
circumstances,” says Diana Mitlin at the University of Manchester, UK. “It’s a pretty terrifying scenario.”

A high risk of the virus spreading extremely rapidly in informal settlements is combined with the fact many people will already have persistent coughs – a key covid-19 symptom – from cooking indoors with charcoal.

Then there is the alarming prospect of the virus entering refugee camps, which house between 8 and 9 million people globally. Paul Spiegel and Shaun Truelove at Johns Hopkins University have modelled what impact that would have on the 600,000 Rohingya people living in a camp in Bangladesh. They found that up to 544,000 could be infected in a year, with potentially more than 2100 deaths.

The youthful population explains the relatively low mortality rate given the high case numbers, but Truelove says this is a best-case scenario. People in refugee camps may already be malnourished and may not be allowed into intensive care units at nearby hospitals, so death rates could be higher.

Social distancing efforts are under way in this camp, says Spiegel, including reducing queues for food distribution. But with high densities and uneven access to water, he fears for refugees and warns that camps aren’t impervious to the virus.

No reports of the virus in camps have reached Spiegel, but he says he wouldn’t be surprised if refugees had already been infected.

“The one positive thing is often refugees are blamed falsely for bringing in diseases, and it’s clear here no one can be blaming refugees and migrants for this particular disease,” he says.

Risk factors

Does a cell protein explain covid-19 severity?

Jessica Hamzelou

DOES a protein on the surface of some of our cells explain why certain people are more at risk from covid-19?

Studies of confirmed cases so far show that people with coronary heart disease or diabetes are more likely to die than other people if they catch the coronavirus. Individuals with lung disorders, such as chronic obstructive pulmonary disease (COPD), and smokers are also at greater risk, says Janice Leung at the University of British Columbia in Canada.

As we begin to understand the virus better, focus is turning to a protein called ACE2. The coronavirus attaches to this receptor protein on the surface of our cells to gain entry to them. The protein is carried by cells in the nose, lungs and gut.

It is possible that variation in how much of this protein people have may help explain why some are more likely to die from covid-19. When Leung and her colleagues looked at lung tissue samples from volunteers, they found that the cells of smokers had much more ACE2 (medRxiv, doi.org/dqx2).

This may explain why these people are more likely to have severe covid-19 infections, says Leung. “If you ever needed another reason to stop smoking, this would be it,” she says.

People with diabetes also seem to produce more ACE2. But we don’t know yet if ACE2 levels really do have an effect on coronavirus infections.

“People with coronary heart disease or diabetes are more likely to die of the coronavirus”

The link between covid-19 deaths and diabetes and cardiovascular and heart conditions has led to some concern over ACE inhibitor drugs. These drugs, which are used to treat high blood pressure, coronary artery disease, heart failure and diabetes, work by targeting the ACE enzyme – a different protein, but one that works alongside ACE2 to regulate blood pressure.

A study of 106 people with covid-19 by Yong Xiong at Zhongnan Hospital of Wuhan University, China, and his colleagues found that having hypertension, diabetes or cardiovascular disease was linked to patients clearing the virus from their bodies more slowly.

The team suggests that ACE inhibitor drugs taken by some of these patients may have increased their levels of ACE2, providing the virus with more opportunities to dock onto their cells, although the study didn’t show whether this was the case (medRxiv, doi.org/dqx3).

Just as we don’t yet know if ACE2 levels contribute to symptom severity, we also don’t know if taking ACE inhibitors has a negative effect. All we can really say so far is that people with certain conditions are at higher risk of death from covid-19, and that these people are likely to have higher ACE2 levels and also be taking ACE inhibitors.

Because no clinical studies have so far shown that ACE inhibitors raise the risk of covid-19, many organisations, including the American Heart Association and the European Medicines Agency, recommend that those prescribed these drugs continue to take them.

Ultimately, ACE2 might be a good target for drugs to block infection by the virus, but we don’t yet know if interfering with the protein would be safe. Several studies in mice suggest that ACE2 plays an important role in responding to injury in the lungs, so blocking its function might prevent such injuries from healing.

“We know a little bit about ACE2, but we clearly don’t know enough to actually say anything intelligent yet,” says Jose Ordovas-Montanes at Boston Children’s Hospital in Massachusetts.