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Keeping the COVID-19 R number below 1 as mobility rises*

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- *Collapse in mobility pushed down the reproduction number to below one*
- *Mobility rising as restrictions are eased*
- *A number of developments will allow more mobility*
- *But mobility cannot fully recover to pre-lockdown level.*

In our recent research, we have highlighted how a dramatic decline in mobility was needed to push the reproduction number of COVID-19 below one and gain control of the pandemic (see here). In this note, we consider how much mobility can increase while keeping the reproduction number below one and, thus, limiting the risk of a second wave of infection. Fortunately, a number of developments can play a role in depressing the reproduction number even as mobility increases.

On the basis of some illustrative calculations, we find mobility can recover to around halfway between the pre-lockdown level and the full-lockdown level, without an extensive testing and contact tracing regime. The analysis here has been conducted for the UK, but the framework can be applied to other countries in Europe and elsewhere that do not yet have an extensive testing and contact tracing regime.

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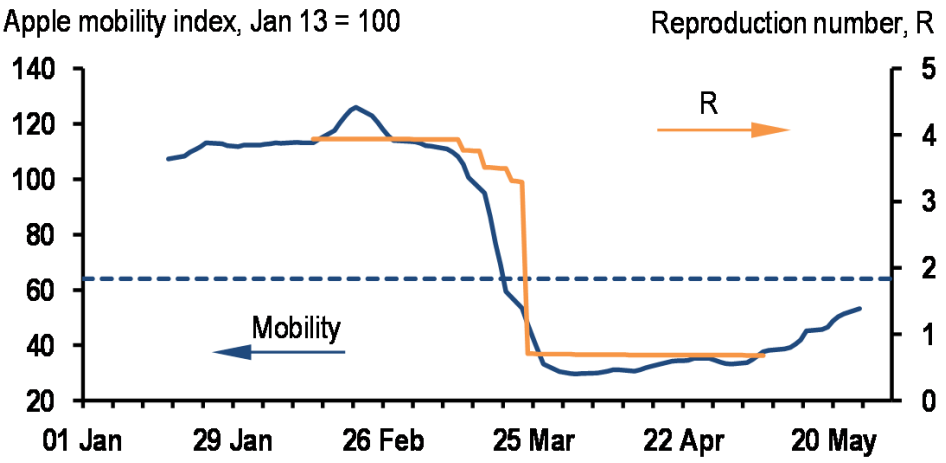
A framework for thinking about the reproduction number

It is clear that the COVID-19 pandemic has been contained by a dramatic decline in mobility, which has limited the number of contacts between infectious individuals and susceptible individuals (Figure 1). As restrictions are eased, mobility is increasing. How much can it rise without threatening a second wave of infection?

Figure 1: UK - Apple mobility vs. reproduction number



Source: J.P. Morgan; Apple, Imperial College

In addition to the dramatic decline in mobility, a number of other developments also could have weighed on the reproduction number (Table 1). We start with the basic reproduction number (R_0), which in the UK is estimated at 3.3 by Imperial College.[1] We have identified five factors that could be weighing on the effective reproduction number (R_e) in addition to the decline in mobility. While the estimates in Table 1 are reasonable, we would stress the high level of uncertainty about almost everything to do with COVID-19.

Table 1: Potential impacts of various developments

Starting value of R_0	3.3
Impact of:	
5% infection prevalence in susceptible population	-0.2
Age-related heterogeneity in susceptibility	-0.4
Self-isolation of all vulnerable individuals	-0.1
Impact of weather	-0.1
Impact of wearing masks etc.	-0.2
Final value of R_e	2.3

Source: J.P. Morgan

First, acquired immunity. R_e is affected by the acquired immunity in the population. The greater the proportion of individuals who have immunity through infection and recovery, the harder it is for infectious individuals to spread the virus. A wide variety of estimates – either antibody tests or modelling exercises – suggest that infection prevalence and, thus, acquired immunity is around 5% of the total population (Table 2). This pushes down R_e by 0.2 (R_0 - R_0 * 0.95).

[1] Imperial College estimates of R can be accessed here: https://mrc-ide.github.io/covid19estimates/#/

Table 2: Estimates of infection prevalence

Approach	Estimate of infection prevalence, % of total population	Estimate of acquired immunity in the UK, millions
Imperial College	5.4	3.6
Antibody testing - Denmark	2.7	1.8
Antibody testing - Germany	14.0	9.3
Antibody testing - Scotland	1.2	0.8
Antibody testing - Santa Clara County	3.4	2.3
Antibody testing - Los Angeles County	4.1	2.7
Vo', Italy	4.4	2.9
Antibody testing - Stockholm	7.5	5.0
Average	**5.3**	**3.5**

Source: J.P. Morgan; S. Flaxman et al., Estimating the number of infections and the impact of non-pharmaceutical interventions on COVID-19 in 11 European countries, Imperial College, 30 Mar 20; T. Spector, COVID symptom Tracker, access here: https://covid.joinzoe.com/data, Kings College London, 2020; M. Cembalest, Equity rally and herd immunity, J.P. Morgan, 13 Apr 20; E. Bendavid, et. al., Covid-19 Antibody Seroprevalence in Santa Clara County, California, 11 Apr 20; Sweden Public Health Authority, Results from investigation of the occurrence of covid-19 in the Stockholm region, 09 Apr 20; USC and LA Public Health, Preliminary results of USC-LA County Covid-19 study released, 20 Apr 20; E. Lavezzo, et. al., Suppression of Covid-19 in the municipality of Vo', Italy, 18 Apr 20

Second, age-related heterogeneity in susceptibility. Simple epidemiological models assume that everyone in the population is equally susceptible to infection at the start of an epidemic. This does not appear to be the case for COVID-19. We know that morbidity and mortality increase with age, but it also seems that susceptibility to infection increases with age. In the Italian municipality of Vo, for example, where almost the entire population (86%) was tested for the virus after the municipality was completely locked down on February 23, only 0.6% of those aged under 20 tested positive while 3.0% of those aged over 20 tested positive[2] (Table 3).

Table 3: COVID-19 susceptibility and the age distribution

Age group, years	Number of individuals	Individuals positive for virus	% positive
0-10	217	0	0
11-20	250	3	1.2
21-30	240	4	1.7
31-40	286	7	2.4
41-50	439	5	1.1
51-60	496	16	3.2
61-70	384	15	3.9
71-80	318	19	6.0
81+	182	4	2.2
Aggregation			
Age group: 0-20	467	3	0.6
Age group: > 20	2345	70	3.0

Source: J.P. Morgan; E. Lavezzo, Suppression of Covid-19 outbreak in the municipality of Vo' Italy, 2020

A strong impression of age-related susceptibility is also evident in an Icelandic study of 9,199 individuals (2.5% of the total population).[3] Of the 564 children aged under 10 years, only 38 (6.7%) tested positive. Meanwhile, of

[2] E. Lavezzo, Suppression of Covid-19 outbreak in the municipality of Vo' Italy, 2020

[3] D. Gudbjartsson, Spread of SARS-CoV-2 in the Icelandic population, 2020

the 8,635 individuals aged over 10 years, 1,183 (13.7%) tested positive. In Table 1, we assume that only 50% of individuals under the age of 20 are susceptible. This represents 12% of the UK population. This pushes down R_e by 0.4 ($R_0 - R_0 * 0.88$).

Third, self-isolation of older individuals. The UK government is not inclined to introduce any formal age-related restrictions. But, nevertheless many older individuals who feel vulnerable may decide to continue to shield themselves from contact with the rest of the population. In Table 1, we assume that 25% of individuals above the age of 70 decide to continue shielding themselves. This would represent 3% of the UK population, and would push R_e down by 0.1 ($R_0 - R_0 * 0.97$).

Fourth, the impact of the weather. Generally, respiratory viruses thrive better in colder, drier conditions, for a number of reasons: first, viruses are more stable in cold weather with low levels of ultraviolet light; second, respiratory droplets remain in the air for longer in drier conditions; third, individuals have more contacts when staying indoors during winter; and fourth, immune systems can be run down in winter by reduced vitamin D due to less exposure to sunlight.

Empirical work suggests a very modest impact of temperature and relative humidity on the transmission of COVID-19. For example, Wang et al.[4] estimate that a 1°C increase in temperature reduces Re by 0.023, while a 1% increase in relative humidity reduces R_e by 0.0078. In Table 1, we apply these coefficients to changes in UK weather since March and find that R_e is reduced by 0.1.

Fifth, the impact of wearing masks, etc. During the COVID-19 pandemic, individuals have been encouraged to wear masks, wash their hands regularly, use hand sanitizer, and conduct more frequent cleaning operations to reduce the likelihood of infection given contact between an infectious individual and a susceptible individual. Unfortunately, there is very little empirical work on the efficacy of these actions. A meta-analysis of 14 trials showed no impact of wearing masks on the spread of influenza type infections.[5] In Table 1, we assume that all of these increased hygiene measures reduce the reproduction number by 0.2. But, this estimate is particularly uncertain.

Putting it all together

On the basis of the calibration that we have assumed, the five developments that we have identified could push the reproduction number down by 1.0pt (from 3.3 to 2.3). If mobility were to quickly return to pre-lockdown levels, R_e would move back towards 2.3, which would guarantee a second wave of infection. This suggests that, absent an extensive testing and contact tracing regime, mobility will need to remain subdued relative to pre-lockdown levels. The experience in recent months suggests that 10pts on the Apple mobility index equals 0.39pt on the reproduction number. If we need to keep R_e at 0.9, and prevent it from moving up to 2.3, then the Apple mobility index needs to stay around 36pts below the pre-lockdown level. This is just under halfway back from the full-lockdown level, as illustrated in the horizontal line in Figure 1. The only way for mobility to increase further than this is to introduce an extensive testing and contact tracing regime, which for the UK, at least, is only beginning to take shape. The efficacy of contact tracing is also influenced by the level of new infections at the start of lockdown easing. These were still relatively high in the UK when lockdown easing began.

[4] J. Wang, et al., High Temperature and High Humidity Reduce the Transmission of Covid-19, 2020

[5] T. Jefferson, Physical interventions to interrupt or reduce the spread of respiratory viruses, 2020

About the author

David Mackie *is a Managing Director and Senior Advisor for European and Global Thematic Research. He has been at JPMorgan for 30 years, analyzing a number of different European economies and various regional and global issues. From 2000 to 2018 he was the Head of Economic Research for Western Europe, managing a small group of economists, but he is now in a new role focusing on thematic research. Prior to joining JPMorgan he spent 5 years at the Bank of England, both as an economist and as a manager of the official foreign exchange reserves. David completed his undergraduate studies at Cambridge University in 1981 and his postgraduate studies at Oxford University in 1984.*

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