A workable strategy for COVID-19 testing: stratified periodic testing rather than universal random testing

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Abstract: This paper argues for the regular testing of people in groups that are more likely to be exposed to SARS-CoV-2, to reduce the spread of COVID-19 and resume economic activity. We call this ‘stratified periodic testing’. It is ‘stratified’ as it is based on at-risk groups, and ‘periodic’ as everyone in the group is tested at regular intervals. We argue that this is a better use of scarce testing resources than ‘universal random testing’, as has been recently discussed globally. We find that, under reasonable assumptions and allowing for false negative results 30 per cent of the time, 17 per cent of a subgroup would need to be tested each day to lower the effective reproduction number $R$ from 2.5 to 0.75, under stratified periodic testing. Using the same assumptions the universal random testing rate would need to be 27 per cent (as opposed to 7 per cent as argued by Romer (2020b)). We obtain this rate of testing using a corrected method for calculating the impact of an infectious person on others, and allowing for asymptomatic cases. We also find that the effect of one day’s delay between testing positive and self-isolating is similar to having a test that is 30 per cent less accurate.

Keywords: coronavirus, COVID-19, epidemic, diagnostic testing, stratified periodic testing
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I. Introduction and summary

Governments around the world are looking for a testing strategy for COVID-19; one that will ensure that the escape from lockdown is as fast as possible, given a certain level of safety. This strategy will inevitably be constrained by our testing capacity: every person in the world cannot be tested every day. Given these constraints, we argue that ‘stratified periodic testing’ is an efficient way of using scarce testing resources, and provides a way for calculating the frequency of testing needed to stop the spread of the virus within a particular group. We also argue that our method is more efficient than ‘universal random testing’, which has been widely discussed since it was proposed by Romer (2020b).

Our proposal is ‘stratified’ because it focuses testing on groups who are at particular risk of spreading the infection. This may be because they have a high basic reproduction number, $R_0$, to begin with, like healthcare workers. Or, it may be because they have a high reproduction number because of the way lockdown has been unevenly applied, for example delivery drivers remaining active. The choice of groups may also take into account fatality rates if they were to spread the virus (like those in care homes for the elderly), or the economic impact if the group needed to be isolated (as discussed by Kasy and Teytelboym (2020)). Constraints on testing capacity mean that this is a more workable and efficient solution than testing the entire population.

We also propose that tests should be ‘periodic’, rather than random. This ensures that each member of the group is certain to be tested at regular intervals, which improves the efficiency of testing. For example, it can prevent some individuals being tested on two successive days, and others having to wait a disproportionate length of time to be re-tested. We show that this increases the efficiency of any given number of tests by approximately 37 per cent, at no extra cost.

Using this approach we find that, under plausible assumptions, 17 per cent of a group would need to be tested each day to reduce the virus's reproduction rate from $R_0=2.5$ to $R'=0.75$ (see section IV). This allows for half the cases to be symptomatic, and therefore self-isolating after 5 days. It also allows for people to be infectious on the day of their test, and for 30 per cent of tests to return false negative results. If there are no false negatives, or if people are not infectious on the day they test positive (which we think would be difficult to achieve in practice), then the required testing rate would be 12–13 per cent per day. Naturally, even less testing would be required if it were to be accompanied by low-cost interventions (e.g. face masks) that reduce the group’s initial $R_0$.

To set our results in context, we also consider ‘universal random testing’. This was proposed by Romer (2020a,b), who argued that randomly testing 7 per cent of the entire population every day would be sufficient to bring the effective reproduction number down to $R'=0.75$. In this proposal he made the important point that the economic benefit of a speedier recovery would be measured in $\text{trillions}$ and this would easily justify spending $\text{billions}$ on testing; he also argued that it is not necessary for such testing to be highly accurate. Unfortunately, as we show, Romer’s calculations did not allow for asymptomatic cases, or delays in isolation, and also contained an error. Using a corrected model we find that universal random testing would require 27 per cent of the population to be tested each day—an impossible task.

So long as testing remains a scarce and relatively expensive resource, we argue that testing of the general population should be reserved for two purposes. Informational
tests will help identify the groups that need to be included in stratified periodic testing, though the samples required for this will be very small relative to the size of the whole population. Antibody tests would allow those that have been infected to get back to work, providing that those who have been infected turn out to be immune to future infection.

The paper is organized as follows. Section II lays out our objective of reducing the effective reproduction rate down to $R'=0.75$, and section III describes our strategy for achieving this: stratified periodic testing. Section IV then describes how to calculate the testing rate needed in a particular subgroup to achieve the objective. Section V calculates the testing rates needed to achieve the same objective under universal random testing, under a variety of assumptions. Section VI compares the results of the two methods, while section VII concludes.

In Appendix 1 we derive the testing rate required to achieve $R'=0.75$ under stratified periodic testing, if people are not infectious on the day they test positive. Appendix 2 considers two papers by Romer (Romer, 2020a,b) which argue in favour of universal random testing, and shows why we think that these papers contain an error. Appendix 3 provides a simple formula which can be used to support the conclusions in the body of the paper that universal random testing strategy would not control the spread of the virus, and which also provides some simple intuition as to what would be required for that strategy to work.

II. The basic ideas

The reproduction number $R$ in an epidemic is the expected number of cases directly generated by any one infectious case. The basic reproduction number, $R_0$, is the initial value of $R$. We assume that the value of $R_0$ for the whole population is $R_0 = 2.5$ (following Romer, 2020a,b).

When $R_0 > 1$, the speed at which the disease spreads depends not only on that value of $R_0$ but also on the rapidity of transmission, and—in particular—on the number of days for which an infected individual remains infectious, and on whether the infectiousness of an individual is particularly great during the early days of his or her infection.

It is clear that an epidemic can only be prevented from spreading by a policy which creates an effective reproduction number, $R'$ ($R$ prime), of less than 1. When that happens, each infected person will infect less than one new person, and the epidemic will die out. If the value of $R'$ is brought down to a number well below 1 then the epidemic will die out quickly.

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1 One can think of an original value for $R_0$ which is the reproduction number of the infection where all individuals are susceptible to infection, and no policy interventions have been adopted. But of course we also want to allow for the effects of policy interventions which might reduce that rate, such as wearing face masks, undertaking social distancing, or allowing the infection to spread so as to reduce the susceptible population. Such features can be included in our model. One way of doing this is to model these effects explicitly, which is how we will allow for the effects of self-isolation. Alternatively one can be implicit, by using an adjusted value for $R_0$ as an input to the calculations, in order to reflect the characteristics of a group who have particular attributes: like having a high proportion of susceptible people, or being already subject to existing interventions like face masks.
In our calculations below we explore the effect of a policy designed to produce an effective reproduction number $R'$ of 0.75. Our means of doing this will be to test some subset of the population each day and then to isolate those who are found to be infected. The effective reproduction number $R'$ in these circumstances is the product of the basic reproductive number, $R_0$, and the fraction of the infectious population that is not isolated. We have chosen the number 0.75 because this was the number chosen by Romer (2020).

Let $\phi$ be the proportion of the infectious population which is isolated. Then we can write Equation (1):

$$R = (1-\phi)R_0$$

(1)

To achieve an objective of $R' = 0.75$ when $R_0 = 2.5$, Equation (1) implies that that $\phi = 0.7$, i.e. that 70 per cent of the infectious population is isolated, and that sufficient tests and isolation are carried out to make this possible.

III. A workable strategy of stratified periodic testing

(i) Stratified testing

We argue that testing should be carried out at different frequencies for different stratified groups, based on their likelihood of infecting others. This likelihood can be deduced from their occupation, geography, likely behaviour when not isolated, and other factors. Testing at rates above 20 per cent per day could be done for carefully selected groups which have a high basic reproduction number ($R_0$) relative to others. Doing that would enable greater rates of isolation in these groups, lowering their effective reproduction number ($R'$) and helping to prevent the epidemic spreading where it matters most. Identifying high-risk groups and testing them frequently appears to be a much lower-risk strategy for containing the spread of infection than randomly testing the entire population (which we consider in sections V and VI). The strategy being advocated here would be easier to pursue if cheap tests were used, even ones that were somewhat inaccurate.

Broadly, there are two types of people who are likely to have a particularly high basic reproduction number relative to others. The first are those who have a high basic

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2 Some of what follows comes from suggestions made to us by Eric Beinhocker, for which we are very grateful.

3 At risk occupations might be healthcare and transport workers, geographies would be cities, and behaviours might be religious and social groups that are likely to gather in large numbers.

4 It might even enable the general lockdown to be eased, so that other lower-risk groups could keep working and not need to be isolated.

5 When deciding whom to test, it makes sense to consider the opportunity cost to economic activity if a group is unnecessarily isolated, something which is beyond the scope of this paper. Conversely, it makes sense to automatically isolate some people even without testing them, if they have a very high prior probability of being infected, and the cost of isolation is low. We do not discuss these two issues in this paper, but they are considered by Kasy and Teytelboym (2020, in this issue).
reproduction number to begin with. These are individuals who would have been more likely to infect others, before the infection had begun to spread and any policy interventions had been adopted. Members of large religious congregations are one example, because of their tendency to meet in large groups indoors. Doctors are another, as they have very frequent and unavoidable close contact with others. Their basic reproduction number will, as a result, be very high and very frequent testing will be necessary to ensure that their effective reproduction number is low enough. As a result, there could be very frequent testing for doctors in hospitals to ensure that the effective reproduction rate for them is brought well below 1. It appears likely that the relevant calculations will show that doctors actually need to be tested every day.

But there are also other people who will have a high basic reproduction number because of the uneven application of the lockdown and other factors that might affect variation in infectiousness across groups. For many people, for instance, lockdown means that they are confined to their homes (including many workers who are able to work from home), reducing their basic reproduction number to well below 1. But key workers, who are encouraged to keep working in spite of the lockdown, will have a higher basic reproduction number as a result (e.g. those involved in food production and distribution). The same will be true for all those who are unable to work from home and are given permission to avoid the lockdown (e.g. those involved in construction and manufacturing). Another group that is likely to have a higher basic reproduction number is those who are more exposed to people who are particularly susceptible to the infection (e.g. prison warders and care workers). As testing is rolled out it will become increasingly appropriate to test all such people frequently.

One challenge in all this is that the basic reproduction number may be high in particular groups for idiosyncratic reasons that are hard to anticipate. The kinds of calculation described in the next section could easily be carried out for structured samples in different locations, in order to identify these pockets of infectiousness.

The general principle, then, is that testing should be concentrated in groups that have high reproduction numbers relative to others, in the current state of the world. But this principle should not be interpreted too strictly. In certain cases, other criteria may also be important: for instance, we may want to regularly test groups that interact with those who are more likely to die from the infection (this is another reason to test care workers more frequently) or groups whose absence would have a greater economic impact than others (see Kasy and Teytelboym, 2020).

(ii) Clarifying how this strategy differs from universal random testing

Universal random testing is an alternative regime that has been widely discussed recently (see Romer, 2020b; Long, 2020; Chotiner, 2020). This approach involves, after prioritizing some of the most important groups, mass testing for the whole population, in order to get the effective reproduction number down for the whole population. This

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6 See footnote 4.

7 Romer has provided useful suggestions about who might have priority as tests are rolled out: https://threadreaderapp.com/thread/1248712889705410560.html.
testing is also random, rather than at pre-specified periods. The approach is summarized in a Twitter thread which concludes as follows:

When you strip away all the noise and nonsense, note that once we cover essential workers, it’s easy to test everyone in the US once every two weeks. Just do it. Isolate anyone who tests positive. Check your math. Surprise, $R_0 < 1$. Pandemic is on glide path to 0. No new outbreaks. No need for any more shutdowns.8

Instead, we argue that testing must be focused on particular groups. This is because our findings, discussed in section V below, show that a mass testing plan would still leave the effective reproduction number significantly above 1 unless it was carried out infeasibly frequently.

Nevertheless, there will still need to be random testing of groups in the population, and some random testing of the whole population. But this testing would be for informational purposes only and would only involve testing very small samples of those involved.

Such informational testing will be needed for two reasons. First, random testing of small samples from particular groups will be necessary to track groups in which the basic reproduction number is already known to be high, and where there is greater potential for a high rate of spread. Once identified, these groups will then need very frequent testing of everyone in the group, for the reasons which we have been discussing in this paper. But testing of small samples of wider groups in the whole population will also be needed to identify new groups where the basic reproduction number is high. As before, this may be for idiosyncratic reasons that are hard to anticipate. Once identified, such groups will then also need very frequent testing of everyone in the group.

But the accuracy of this testing for informational reasons will be determined by the sample size, rather than population size. The samples required for these informational purposes will be very small relative to the size of the whole population.

(iii) Periodic tests rather than random tests

Once the frequency of testing has been decided and testing kits are available, testing can begin for everyone in the identified groups. But it is important that this testing be done periodically for each person, rather than there being a random choice of those who are to be tested in each time period.

The rationale underlying periodic testing can be explained by the ‘waiting-time paradox’.9 Random testing, say, of 20 per cent of a group each day, wastes many resources. This is because every day some of those tested will have actually been tested the day before, while others of those who are infectious will, nevertheless, not be tested and so will possibly continue infecting people. By contrast, periodic testing of 20 per cent of a group means that, on days 1 to 5, a different fifth of the group will be tested each day, and that on day 6 the first fifth of the group will be tested again, and so on. It is clear that

8 Romer uses $R_0$ here to stand for what we call $R'$. See again: https://threadreaderapp.com/thread/1248712889705410560.html

9 The waiting time for a Poisson bus service is twice the waiting time for a periodic bus service with the same rate for a randomly arriving traveller.
this means each person tested will have been tested exactly 5 days previously, removing
the problem that some tests are being wasted and that other tests are being postponed
for too long. Because of this, you need to test far fewer individuals in a group to get the
same reduction in $R$.

In section VI, we provide a simple account of this issue, and show how important it is
likely to be. We show that with high testing rates, periodic testing beats random testing
by a very significant factor.

(iv) The testing system: running two kinds of tests in parallel

In this paper, we have been discussing diagnostic testing (i.e. testing for active infec-
tions) as opposed to antibody testing (i.e. testing for those who have had the disease and
are both immune and non-infectious). A combination of the two types of testing might
be effective and realistic if the diagnostic testing capacity remains constrained, but anti-
body tests become widely available. One might then proceed as follows.

- Immediately and frequently perform infection tests on groups and areas with a
  high $R'$ and immediately isolate those found to be positive. Trace\(^{10}\) and test the
  contacts of those who test positive, as these now have a higher probability of
  also testing positive.
- Self-isolate anyone developing symptoms for a minimum of 14 days. These
  people would not be tested unless medically necessary. Trace and isolate their
  contacts.
- If there were enough testing resources available then one could test people at
  the *end* of their isolation period to show that they were clear of virus before
  they were allowed to come out of isolation.
- Make antibody testing kits widely available for home use to enable anyone to
  see if they have had the virus. These would be one-off tests in the sense that
  tests which gave a positive result would not need to be repeated.
- A system to track people with immunity who could then circulate freely if they
  had either (a) had a positive antibody test indicating an acceptable level of
  immunity, (b) had a positive diagnostic test more than some specified number
  of days ago, or (c) had a negative diagnostic test after their symptoms resolved.

All of this could be done using cheap diagnostic tests, even if they were somewhat
inaccurate. It is not the case that ‘no test is better than an unreliable test’. Our calcula-
tions show that, while accurate tests are absolutely necessary for clinical reasons when
treating an individual person, much more rough-and-ready testing is satisfactory if the
purpose of this testing is epidemiological control through isolation. (In our baseline
calculations discussed below, for instance, we assume that 30 per cent of infected people
wrongly test negative.)

\(^{10}\) Contact tracing can be as simple as testing those in the household and workplace of those who are in-
fected. Technological solutions exist to perform more detailed contact tracing, e.g. using mobile phone move-
ments. However, these involve privacy concerns which, crucially, may take time to debate and resolve. The
authors believe that simple, immediate testing of infected households and workplaces is preferable to detailed
tracking of mobile phone movements. As contact tracing apps become more widespread the degree of con-
tact (separation distance, length of interaction, etc.) will be available to help the targeting of testing resources.
Doing all of this will help governments to track the spread and to determine where hotspots are flaring up. Such information will help them to work out how to selectively tighten, or loosen, containment measures when needed.

Such a testing procedure would involve doing two things at once: the stratified periodic diagnostic testing which we have been discussing would be designed to dampen the spread of the disease in key groups, by catching those in these groups who were infectious but asymptomatic, or pre-symptomatic, or post-symptomatic, and so not self-isolating. At the same time, antibody testing for the entire population would separate out the immune population. Passing an antibody test would enable such people to return to work.

IV. Calculating the required test rate for stratified periodic testing

(i) Finding the periodic testing rate which would get R down to R’ = 0.75

Having described what stratified periodic testing looks like, we now derive more formal results for the testing rate required to get R down to R’ = 0.75, and the testing ‘threshold’ at which R’ = 1. In section V we go on to show that periodic testing is about 37 per cent more effective than random testing, at no extra cost.

We assume that the number of days an infected person is infectious is $d = 14$, following Romer (2020b). Fourteen days is familiar in the analysis of COVID-19 as the period after which the person is either dead or—much more likely—recovered, but no longer infectious. For simplicity we also assume that there is uniform contagion throughout the 14-day period. So, if $R_0 = 2.5$, and if there were no testing which led to the isolation of infected people, then an infectious person would infect 2.5 people in total, or $2.5/d$ persons per day over a period of $d$ days. The results which follow obviously depend both on our assumed value of $d$ and on our assumption that the infectiousness of an individual is not disproportionately great during the early days of his or her infection.

In almost all countries, an important part of the current policy response to COVID-19 is that those who show symptoms are required to self-isolate. Because of this, only asymptomatic people and those who have chosen not to self-isolate, for example with mild or mistaken symptoms, will be spreading the disease, and will be included in those who are tested. We assume that all of those infected are asymptomatic for 5 days (and are subject to testing during that time). After 5 days a proportion of the population, $\alpha$, would be infectious.

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11 We are grateful to Frank Kelly for his assistance in preparing this section.

12 Seven days has been the standard advice for isolation, or 14 days if more than one in a household, however recent data show that the infectious period may last much longer. A recent detailed study of repeatedly tested individuals in Taiwan found a long tail for infectiousness. For further discussion of this point see section IV below. See also https://focustaiwan.tw/society/202003260015.

13 We note that some medical data show an asymptomatic infectious period followed by a hump of maximum infectivity as symptoms develop and a tail as symptoms resolve.
A workable strategy for COVID-19 testing displays symptoms and self-isolates. This means that only a proportion \((1 - \alpha)\) go on infecting others, and being tested, from day 6 onwards.

The proportion of asymptomatic patients isn’t really known, and estimates vary wildly. The World Health Organization suggests that as many as 80 per cent of cases are asymptomatic or mild. Very inexact data from Iceland suggest that all infectious patients are asymptomatic for the first 5 days and that, after this time, only about half become symptomatic. We use these Icelandic figures when constructing the ‘base case’ in our calculations, so \(\alpha = 0.5\).

Following Romer (2020b) we allow for the tests to return false negatives. We let \(n\) be the proportion of false negatives and assume that \(n=0.3\) (i.e. 30 per cent of those who are infected test negative).

We let \(R'\) be the expected number of people that a randomly chosen infected person infects before that person is positively tested (or stops being infective, if sooner). Let \(r_j\) be the expected number of people infected by an individual on day \(j\) of his/her infection, for \(j = 1, 2, \ldots, d\) where the length of infectivity is \(d\). Thus \(R' = \sum_{j=1}^{d} r_j\). Now suppose an individual is tested every \(N\) days, and that for high-risk groups we test very frequently, so that \(N < d\). We assume that the time of the infection is random and an infected individual remains infective on the day of the test, and is only isolated from the following day onwards, since this is more likely than instant isolation. (We also consider the case of instant isolation in Appendix 1 and Figure 3.) Then,

\[
R' = \frac{1}{N} \sum_{i=1}^{N} \sum_{j=1}^{i} r_j.
\]

From this we can deduce that

\[
R = \frac{1}{N} \sum_{j=1}^{N} (N - j + 1) r_j
\]

Here we introduce our self-isolation factor \(\alpha\) into equation (3) by noting that \(r_j\) during the infectious and asymptomatic period (first 5 days), which we denote as \(d_0 = 5\), is still \(r_0 = \frac{R_0}{d}\) and thereafter \(r_\alpha = (1 - \alpha) \frac{R_0}{d}\) for the infectious period in which a person may self-isolate. Substituting these values into \(r_j\) we have three alternative situations to consider: (i) the case when \(N < d_0\) for which the testing rate is more frequent than the move to becoming symptomatic that occurs after day \(d_0\) and therefore equation (3) represents this outcome entirely; (ii) the case when \(d_0 < N < d\) and the testing period \(N\) is between 6 and 13 days inclusive, and finally (iii) the case when \(N \geq d\).

The following is for the case when \(d_0 < N < d\) which is the testing range that tends to deliver an \(R' < 1\) for this model of testing:

\[
R' = \frac{1}{N} \sum_{j=1}^{N} (N - j + 1) r_j = \frac{1}{N} \sum_{j=1}^{d_0} (N - j + 1) r_0 + \frac{1}{N} \sum_{j=d_0+1}^{N} (N - j + 1) \alpha r_0
\]
We take \( r_\alpha = (1 - \alpha) R_0 / d \) to be the daily likelihood that an infected individual will infect another person during the period in which they may become symptomatic and self-isolate with probability \( \alpha \). This gives\(^{14} \)

\[
R' = \frac{R_0}{2Nd} \left[ (N + 1) N - \alpha (N - d_0 + 1)(N - d_0) \right] \tag{5}
\]

We now use equation (5) to solve for the value of \( N \), the number of days between each periodic test that is required to bring \( R \) down from \( R_0 = 2.5 \) to \( R' = 0.75 \). We find that the required periodic testing rate is 17 per cent of the population (or a test every 6 days), when isolation happens the day after a positive test. This assumes a test has a 30 per cent false negative rate. If these tests were perfect, then the required test rate could be 12 per cent of the population per day (or once every 8.2 days). If we instead use instant tests with 30 per cent false negatives (see Appendix 1 and Figure 3), the required testing rate is 13.4 per cent (or every 7.7 days). It is interesting to note that the effect on testing rates is similar for having an unreliable test (30 per cent false negatives versus no false negatives), and having to wait a day for results.

In section V we show that these testing rates required to get \( R' = 0.75 \) are around 37 per cent lower than those required under universal random testing.

(ii) Identifying the testing ‘threshold’ at which \( R' = 1 \)

Figure 1 plots \( R' \) as a function of \( t \), the proportion of the population tested each day. It does this for various values of \( \alpha \), the proportion of infected people who display symptoms and self-isolate from day 6 onwards. Higher levels of \( \alpha \) mean that the effective reproduction rate, \( R' \), is lower for a given level of testing; this effect is particularly marked at lower levels of the daily population testing rate. As we have already seen, when \( \alpha = 0.5 \) the testing rate \( t \) needs to be about 17 per cent to get \( R' \) down to 0.75. Figure 1 also displays the different testing rates needed to obtain a range of different values for \( R' \), for a variety of values of \( \alpha \). This enables us to identify the threshold testing rates, \( t^* \) that reduce \( R' \) to exactly 1: the value which divides outcomes in which the epidemic dies out from outcomes in which it explodes.

For \( \alpha = 0.5 \), this threshold testing rate implied to bring \( R' \) to 1 is \( t^* = 11 \) per cent, i.e. 11 per cent of the population would need to be tested each day. To achieve this, everyone would need to be tested every 9 days.

Figure 1 also shows us how sensitive this threshold testing rate is to \( \alpha \), the proportion of those who are infected who self-isolate from day 6 onwards. For testing rates that are below 20 per cent per day (i.e. less frequent than once every 5 days, the period for which a person is assumed to be asymptomatic), we can clearly see how sensitive this threshold testing rate is to \( \alpha \).

\(^{14}\) For the case \( N < d_0 \) the solution remains unchanged as the frequency of testing is above that which would allow the self-isolation process to occur: \( R' = R_0 \frac{(N + 1)}{Nd} \).

and for the case \( N \geq d \):

\[
R' = R_0 \left[ 1 - \frac{1}{2N} (d - 1) \right] - \frac{\alpha R_0}{2dN} (d - d_0) (2N - d_0 - d + 1). \]
This is an important figure since $\alpha$ may change from population group to population group. For example, contract workers who are paid by the hour have a direct incentive to ignore symptoms. This would translate to a lower $\alpha$ for this group and consequently a much higher $R'$ for any rate of testing. Workers in this category who also have high contact rates as part of their job would therefore be expected to require the highest rate of testing.

The epidemic would be most easy to control if all infected individuals become symptomatic after 5 days, and then all self-isolate. (This would mean that $\alpha = 1.00$.) In such a case, as we can see, $R' \approx 1$ even without any testing. But in this case it would still be necessary to test all of those in a group every 12 days, in order to bring $R'$ down to 0.75.

(iii) Allocating testing over populations with different $R_0$

If scarce testing is to be allocated over individuals with different prior probabilities of infection, these formulas can be used to optimize the allocation.

If we take the simplest form of our periodic testing model, when $\alpha = 0$, for high frequency testing where an individual has a test after a smaller number of days than the length of an infection ($N<d$), then equation (3) simplifies to:

$$R' = \frac{R_0}{2d} (N + 1).$$

Suppose that individual $k$ has prior probability $p_k$ of infection and that we wish to choose $N_k$ so as to allocate a given amount of testing over a set of individuals so as to maximally reduce $R'$. Then, using frequent testing where an individual has a test after a smaller number of days than the length of an infection ($N<d$), the optimal allocation of a given amount of testing should choose $N_k \propto 1/\sqrt{p_k}$.

This shows very clearly that there are diminishing returns to making $N$ very small—that is conducting more frequent tests results in diminishing reductions in $R'$. The
amount of testing allocated, therefore, to higher-risk individuals is naturally higher, but not proportionately so.

(iv) Summary
Periodic testing periods of around 6 days may be sufficient to control the propagation of infection on their own. This is testing approximately 17 per cent of the population each day. This figure is highly sensitive to changes in self-isolation behaviour and the characteristics of the test being administered.

Our model moves us towards a useful framework for determining the frequency with which a subgroup need be tested. For any particular subgroup, the frequency of testing which is required to reduce transmission sufficiently is determined by the both the subgroup’s properties and the properties of the test. A group’s key factors are the capability of its individuals to self-isolate if symptomatic, and the initial propagation rate \( R_0 \). The important figures for the test are the speed of results and the accuracy with which it reports positive results. Our model shows the way in which all of these factors can be traded off against one another.

V. Calculating the required test rate for universal random testing
We now consider universal random testing, and the testing frequency required to get \( R' = 0.75 \). Romer (2020b) suggests that this can be done by randomly testing 7 per cent of the population each day, i.e. testing everybody randomly, at a frequency of about once every 2 weeks. With a population of 300m in the US testing on this scale would require about 20m tests a day. In the UK, with a population of 60m, this would require about 4m tests a day. Romer proposes the immediate allocation of $100 billion in the US to make such an outcome possible.

In contrast, we find that if universal random testing followed by isolation were adopted, more than 21 per cent of the population would need to be tested each day to get \( R' = 0.75 \). This means that everyone in the population would need to be tested, on average, every 5 days. We attribute the difference to a calculation error in Romer (2020b) and our allowance for asymptomatic cases.\(^{15}\) We explain these points in Appendix 2.

(i) Finding the testing rate which would get \( R \) down to \( R' = 0.75 \)
We assume that the whole population is randomly tested. We let \( t \) be the proportion of the population tested each day and then isolated if they are infected. As in section IV we assume patients are infectious for \( d = 14 \) days, symptomatic cases show symptoms and self-isolate after \( d_0 = 5 \) days, but that only half are themselves symptomatic, i.e. \( \alpha = 0.5 \). Of these tests we assume that 30 per cent return false negative results, i.e. that \( n = 0.3 \).

\(^{15}\) Romer (2020b) discussed asymptomatic cases in the lecture but did not allow for them in his calculations.
We also assume ‘instant isolation’, where an infectious person is isolated immediately on the day that they receive a positive test result, and so are not able to infect others, even on that day. In practice this would mean an early morning test with rapid results. This is different from our assumption in section IV, where we assumed ‘delayed isolation’ so that patients remain able to infect others on the day of their test, and only begin to isolate on the next day. We think delayed isolation is a more plausible assumption. But we assume instant isolation in this section so as to make our results more nearly comparable with those of Romer (2020a, b). Nevertheless we extend our analysis to consider the case of universal random testing with delayed isolation in section V(iv).

Our calculations proceed as follows. We consider the impact of an infectious person on others when he or she has a probability \( t \) of being tested each day, and so of being placed in isolation immediately if the test is positive. For clarity, it is helpful to think about the ‘discovery rate’ \( x \), where \( x = t(1-n) \) and \( n \) is the number of tests which show false negatives, which we take to be \( n = 0.3 \). The variable \( x \) shows the probability that, on any day on which this person is infectious, he or she is isolated. This means that \( 1-x \) is the probability that this person will not be isolated, and so be able to infect people on that day and subsequent days.

If there were no self-isolation of those who became symptomatic, then the probability of an infectious person remaining undetected on day \( j \) is \( (1-x)^j \) and the expected number of infections caused by an infected person on day \( j \) is \( r_j \). Therefore, in expectation, an infected person would infect \( r_1(1-x) \) persons on day 1 of their infection, \( r_2(1-x)^2 \) persons on day 2, and so on, up to \( r_{14}(1-x)^{14} \) persons on the final day \( d \). We assume that the infection rate is constant over the infected period and so \( r_j = R_0/d \).

We allow for self-isolation by letting the proportion who are symptomatic, \( \alpha \), self-isolate after 5 days. So, only a proportion \((1-\alpha)\) go on being tested from day 6 onwards. We can thus write our key equation as follows:

\[
R' = \frac{R_0}{d} \sum_{j=1}^{d} (1-x)^j - \frac{\alpha R_0}{d} \sum_{j=6}^{d} (1-x)^j.
\]

or, in more compact notation:

\[
R' = \frac{R_0}{d} \frac{(1-x) + (1-x)^2 + \ldots + (1-x)^5 + (1-\alpha) \left\{ (1-x)^6 + (1-x)^7 \ldots + (1-x)^{14} \right\}}{14} = 0.75
\]

Our ambition is to get to a position where each infectious person only infects 0.75 other people on average, so that the virus dies out. We seek to find the value of \( x \) which would achieve this. Recalling that \( R_0 = 2.5 \), and letting \( d = 14 \), we then can solve for \( x \) in equation (8):

\[
R_0 \left[ (1-x) + (1-x)^2 + \ldots + (1-x)^5 + (1-\alpha) \left\{ (1-x)^6 + (1-x)^7 \ldots + (1-x)^{14} \right\} \right] / 14 = 0.75
\]

or, in more concise notation,

\[
\frac{R_0}{d} \sum_{j=1}^{d} (1-x)^j - \frac{\alpha R_0}{d} \sum_{j=6}^{d} (1-x)^j = 0.75
\]
The solution to this equation can be obtained numerically\(^\text{16}\) for various values of \(\alpha\). When \(\alpha = 0.5\), as roughly observed in Iceland, \(x \approx 0.146\).

But \(t = x/(1 - n)\). And \(n\), the proportion of false negatives, is equal to 0.3. So the required probability of testing on each day, \(t\), is given by \(t = 0.146/0.7 \approx 0.209\). That is to say, the proportion of people tested on any day must be as high as 21 per cent in order to achieve the required 14.6 per cent discovery rate \(x\).

Recall from equation (1) that \(R' = (1 - \phi)R_0\), where \(\phi\) is the proportion of the infectious population which is isolated. With \(R_0 = 2.5\) and \(R' = 0.75\), this means that \(\phi = 0.7\). Thus, over the 14 days in which a person is infectious, this person will, on average, be in isolation for 70 per cent of the time. We have shown that to achieve such a very striking outcome, the probability of testing an infected person who is not yet isolated on any day must be at least 21 per cent. With random, population-wide testing, this means, in effect, that everybody in the population has to be tested about every 5 days.

(ii) Identifying the testing ‘threshold’ at which \(R' = 1\)

The dotted line in Figure 2 plots \(R'\) as a function of the proportion of the population tested, under Universal Random Testing. As we have already seen, when half of those who are infected self-isolate from day 6 onwards, i.e. \(\alpha = 0.5\), \(t\) needs to be equal to about 21 per cent to get \(R'\) down to 0.75. More than this, the figure also displays the different testing rates which are required to obtain a range of different values for \(R'\). And it does this for different values of \(\alpha\) as well.

Figure 2 enables us to identify the threshold testing rates, \(t^*\) that reduce \(R'\) to exactly 1, and so just stop the epidemic from exploding.\(^\text{17}\) For \(\alpha = 0.5\), this threshold testing rate is \(t^* = 13\) per cent. To achieve this, everyone would need to be tested, on average, every 8 days. This is still way above Romer’s proposed testing rate of 7 per cent. Figure 2 also displays the sensitivity of the results to various values of \(\alpha\), the proportion of infected people who display symptoms and self-isolate. It shows that, at one extreme, when all cases are symptomatic after 5 days and then self-isolate (i.e. when \(\alpha = 1.00\)), the situation seems

\(^{16}\) Equation (8) is more difficult to solve than Equation (5). This is because with periodic testing there is a fixed period of time between tests for any individual, whereas with random testing there is, instead, a given probability of being tested on any day and so, for any individual, the time between tests is probabilistic.

\(^{17}\) It would be good to find a simple way of calculating the threshold testing rates, \(t^*\), for any population, based on the value of \(R_0\) for that population, and given any assumed value for \(\alpha\), without having to solve the complex non-linear model being discussed in this section.

It turns out that we can do this by using a simple approximation that ignores the dynamics of the infection process, thereby producing an equation which is easy to solve. In Appendix 3, we set out this simple approximate method for calculating the threshold testing rates \(t^*\) for a population, depending on the values of \(R_0\) and of \(\alpha\) that are appropriate for that population. We show that the approximation is relatively accurate, even though the calculation rests on two simplifying assumptions.

It might be useful to carry out the kind of calculations described in Appendix 3, even despite the fact that, in this paper, we are recommending using periodic testing rather than the random testing. This is because, as we show in section VI of the paper, periodic testing is more efficient than random testing. As a result, calculating the amount of testing needed to bring \(R'\) down to 1, if testing were to be random, might well provide a lower bound for the threshold testing rate when using periodic testing. It might be useful to have a simple way of calculating this lower bound, even though the method of calculation is only an approximate one.
A workable strategy for COVID-19 testing

Figure 2: The share of the population that must be tested each day to achieve a particular $R'$ using Universal Random Testing. It assumes 30 per cent false negatives ($n = 0.3$); and that test results are instant so infected people self-isolate the day of their test.

Notes: $\alpha$ is the proportion of infected people who display symptoms and self-isolate. Higher levels of $\alpha$ mean that the rate on infection ($R'$) is lower for a given level of testing.

Figure 3: The share of the population that must be tested each day to achieve a particular $R'$ using Stratified Periodic Testing. It assumes 30 per cent false negatives ($n = 0.3$); and that test results are instant so infected people self-isolate the day of their test.

Notes: $\alpha$ is the proportion of infected people who display symptoms and self-isolate. Higher levels of $\alpha$ means that the rate on infection ($R'$) is lower for a given level of testing.

just about manageable: the epidemic would stop exploding even without any testing. Nevertheless, the required value of $t$ which would ensure that $R' = 0.75$, is about 8.2 per cent. This percentage is actually a little above that proposed by Romer because infected asymtomatic people do a lot of damage in the first 5 days! At the other extreme, with $\alpha = 0.00$, the situation is much worse: the probability of testing per day, $t^*$, which is required to stop the epidemic exploding is now nearly 20 per cent (19.1 per cent) and the value of $t$ required to ensure that $R' = 0.75$ is about 26 per cent (26.1 per cent).

Data for the extent to which infectious people become symptomatic is currently extremely unreliable and furthermore the range of possibilities seems very wide. Even if over half of infected people self-isolate, around 20 per cent of the population would need to be tested every day to get $R'$ to 0.75. The outcomes depicted in Figure 2 suggest that, on the balance of probabilities, universal random testing would be unworkable.
(iii) Random daily testing of 7 per cent of the population is really a risky ‘throttle’ strategy

Returning to the share of asymptomatic cases observed in Iceland (i.e. the case when $\alpha = 0.5$), Figure 2 shows that by setting a testing rate of 7 per cent of the population, as advocated by Romer (2020b), the epidemic actually remains explosive with $R'$ equal to about 1.3. In this situation, infection would spread rapidly in the earlier stages, since the testing rate is not high enough to control and eliminate the disease; all testing would do is slow down the inevitable spread of the disease. It is clear that the speed at which the disease spreads depends not only on $R'$, but also on the number of days, $d$, for which infected individuals remain infectious, and on whether infectiousness is spread evenly over the time of infection—as assumed here—or is instead concentrated in the early days of infection.

Obviously as the disease spreads, the number of people susceptible to infection begins to fall, and then to fall rapidly. Ultimately, the fact that more and more people have had the disease and so are immune will bring the spread to an end and the proportion of those who have been infected will tend towards a constant level which has become known as the ‘herd immunity’ proportion. It can be shown, for any $R$, that this proportion is given by $(1 – 1/R)$. Without testing, and a value of $R_0$ of 2.5, the herd immunity proportion is as high as 60 per cent.

Universal random testing (as advocated by Romer (2020a, b)) of 7 per cent of the population per day would reduce $R'$ to 1.3, and so reduce the herd immunity proportion, to which the population is tending in the long run, to about 23 per cent. But in the earlier stages of infection, when there is almost no immunity, the disease would still spread rapidly as the testing rate would not be high enough to slow the spread in a controlled manner, particularly if each person infected other people quickly. Thus, in sum, we can say that universal random testing at this rate would only slow the spread (or ‘flatten the curve’), but would not control the initial explosive phase of the epidemic. It would not stop very large numbers of people being infected.

This is therefore a risky strategy. Peak levels of infection might rise beyond the capacity of the health system to deal with the epidemic. Even if testing rates were then increased, lags in responses would mean that the spread of infection would only be gradually reduced. Meanwhile, the virus would go on spreading towards the herd immunity level.

(iv) The robustness of our conclusion that universal random testing is unworkable

Of course, there are many changes to our assumptions which could modify our calculations.

In particular, there would be significant reductions in required testing rates if whole households were to be self-isolated in the event that anyone in the household tested positive. If, for example, only one person in a household were tested at any time, and

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18 Note that the herd-immunity proportion only depends on the value of $R$, and does not depend on the number of days, $d$, for which infected individuals remain infectious, or on whether infectiousness is spread evenly over the duration of illness. These two things only influence the speed at which the herd-immunity proportion is approached.
households consisted on average of two people, then each positive test would remove
two people into isolation. That is, testing would become more effective.

On the other hand, our calculations have deliberately assumed that anyone testing
positive immediately isolates themselves, and so is not infectious on the day of the test.
Using the more cautious assumption that those testing positive are only isolated on the
following day, and so only stop infecting other people then, equation (8a) now becomes:

\[ R' = R_0 \left[ 1 + (1-x) + (1-x)^2 + \ldots + (1-x)^4 + (1-\alpha) \left\{ (1-x)^5 + (1-x)^6 + \ldots + (1-x)^{13} \right\} \right] / 14 \]

(9a)

or, in more concise notation:

\[ R' = \frac{R_0}{d} \sum_{j=1}^{d} (1-x)^{j-1} - \frac{\alpha R_0}{d} \sum_{j=6}^{d} (1-x)^{j-1}. \]

(9b)

The results found using this equation are much worse than those described in section
V(i). With \( \alpha = 0.5 \), the critical testing rate, \( t^* \), is now 16 per cent, and the testing rate
required to get \( R' \) down to 0.75 is now as high as 27 per cent. A bit over a quarter of the
population would need to be tested every day!

VI. Comparing stratified periodic testing with universal
random testing

We can now compare the benefits of stratified periodic testing with those of uni-
versal random testing. In section IV and Figure 1 we showed that under stratified
periodic testing with half of cases being asymptomatic, a 30 per cent false negative
rate, and isolation the day after a positive test, the testing rate needed to bring \( R \)
down from \( R_0 = 2.5 \) to \( R' = 0.75 \) would be 17 per cent of the population each day (or
testing each person every 6 days). In section V(iv) we showed that under universal
random testing, using the same assumptions, the required testing rate would be 27
per cent, or testing each person about every 4 days. This is a 37 per cent reduction
in the number of people needing to be tested each day—the same impact on \( D' \) for a
significantly reduced cost.

The advantages of stratified periodic testing are robust to other assumptions. If we
assume that people immediately self-isolate after testing positive, and so are not infec-
tious on the day of their test, then we would only need to test 13.4 per cent of the popu-
lation per day to achieve \( R' = 0.75 \) (as shown in Appendix 1 and Figure 3), versus 21 per
cent of the population per day under universal random testing (as shown in section V(i)
and Figure 2).

If, instead, we assume that the tests return zero false negatives (\( n=0 \)), but people are
infectious on the day of their test, then we must test 12 per cent of the population each
day under stratified periodic testing to achieve \( R' = 0.75 \), versus 19 per cent under uni-
versal random testing.

We conclude that for the universal random testing proposed to be workable (which
would require testing, say, less than 10 per cent of the population per day) policy-mak-
ers would require confidence that:
(i) nearly all infected patients are symptomatic and self-isolate, reducing the burden on testing after the incubation period,
(ii) the tests are sufficiently effective, and complied with, that they capture more than 70 per cent of infected cases (and ideally close to 100 per cent),
(iii) testing is conducted quickly, early in the morning, and people are isolated on the day of the test, and
(iv) whole households are isolated when any member is infected.

Unfortunately, we do not feel that all these conditions can be met given our current state of knowledge about the virus. So, we do not believe that whole-population random testing would be a good use of resources. Instead, stratified periodic testing is both workable, and an efficient use of finite testing resources.

VII. Conclusions

This paper proposes ‘stratified periodic testing’ as a strategy for ensuring that \( R' < 1 \) and so preventing an explosive re-emergence of COVID-19. Such a strategy is necessary for lockdown to be ended and for economies to return to work.

The testing would be ‘stratified’, in the sense that it would focus on specific subsets of the population who currently have the highest reproduction number \( R \). The criteria for choosing who would be tested could be amended to also take into account the vulnerability of subgroups, or the loss of economic activity if they are forced to self-isolate at home. This ensures that infected people can be identified and isolated quickly. The testing would be ‘periodic’, in the sense that each member of the subset would be tested at regular, defined intervals, rather than testing within the group being done at random.

Those who test positive would be quickly isolated at home, as would anyone with symptoms. The tests need not be perfect: if they are cheap but deployed widely within particular groups, then false negatives can be offset by increasing the frequency of testing. The effectiveness of the programme can be improved by simple tracing of the contacts of those infected: for example testing those in their workplaces and households, rather than fully tracking mobile-phone movements with the associated privacy concerns. It would also be improved by ensuring that all members of a household are isolated if any member tests positive.

We argue that this is better than ‘universal random testing’ which is currently being discussed in many places. Romer (2020a,b) suggests that by testing 7 per cent of the population every day we can get the effective reproduction number of COVID-19 to around 0.75 and thereby curb the epidemic. Unfortunately, Romer’s calculations contain errors. By correcting his method, and using reasonable assumptions about asymptomatic carriers, we believe that, if this strategy were followed, at least 21 per cent of the population would need to be tested each day to get the effective reproduction number down to well below 1 (e.g. to a value of 0.75). For obvious reasons, we do not see this as a feasible population-wide strategy.

Any testing strategy should be thought of as a complement to other measures that can reduce the spread of COVID-19 at little economic cost. For example, those who can work from home with little loss of productivity should continue to do so, retirees
should continue to self-isolate, and people in public places should wear masks and regularly wash their hands.

A process of stratified periodic testing might well become part of a strategy of ensuring that workers can get back to work quickly and safely in sectors of the economy in which work cannot be performed at home. This process should continue until widespread vaccines or treatment for the virus are available.

Appendix 1: Stratified periodic testing with instant isolation

In section V, in which we considered the strategy of universal random testing, we started by looking at a theoretically perfect test for which a positive identification of an infected individual would result in their not being able to infect anyone even on the day of the test. Even though this is an unrealistic assumption, it is useful to also apply it to our proposed strategy of stratified periodic testing, so as to enable a direct comparison of the results obtained under the two different strategies.

To model instant isolation we remove the anticipated extra day of infection that would have otherwise occurred, changing the sum used in the earlier section from \( j = 1 \ldots i \), to \( j = 1 \ldots I - 1 \).

For completeness we now explicitly set out the equations for these calculations.

(a) For low-risk individuals we can test much less frequently, so that \( N \geq d \). Then we obtain:

\[
R' = \frac{1}{N} \left( \sum_{i=1}^{M} \sum_{j=1}^{i-1} r_j + (N - d)R_0 \right)
\]

and hence

\[
R' = R_0 - \frac{1}{N} \sum_{j=1}^{d} jr_j.
\]

So, if \( r_j = R_0/d \) we can solve for the required rate of testing. It is

\[
R' = R_0 \left[ 1 - \frac{1}{2N} (d + 1) \right] - \frac{\alpha R_0}{2dN} (d - d_0) (2N - d_0 - d - 1).
\]

(b) Likewise for testing when \( d_0 < N < d \) we now sum to \( i-1 \) as opposed to \( i \):

\[
R' = \frac{1}{N} \sum_{i=1}^{N} \sum_{j=1}^{i-1} r_j
\]

Then

\[
R' = \frac{1}{N} \sum_{j=1}^{N} (N - j)r_j
\]
giving:

\[ R' = \frac{R_0}{2Nd} [(N - 1)N - \alpha (N - d_0 - 1)(N - d_0)] \]

(c) For high-frequency testing for when \( d_0 > N \) and therefore \( \alpha \) is not relevant to the dynamics:

\[ R' = \frac{1}{N} \sum_{j=1}^{N} (N - j)r_j \]

\[ R = \frac{R_0}{2d} (N - 1) \]

The results are given in Figure 3. As in Figure 1, higher levels of \( \alpha \) mean that the effective reproduction rate, \( R' \), is lower for a given level of testing; this effect is particularly marked at lower levels of the daily population testing rate. As the tests are now instant, the amount of testing in order to reduce \( R \) has fallen. For \( \alpha = 0.5 \) the required testing rate to achieve a value of \( R' = 0.75 \) is now every 7.7 days (\( t = 13.4 \) per cent) as opposed to every 6 days for a test that gave the results a day later.

**Appendix 2: Universal random testing**

We now explain why we think there is a mistake in the way \( \varphi \), the proportion of the infectious population which is isolated, is calculated in Romer (2020b).

Romer assumes random testing of the whole population. The probability that each person is tested each day is set to \( t = 0.07 \), the proportion of false negatives is \( n = 0.3 \), and \( l = 14 \) is the number of days that each person who tests positive is placed in isolation.

The proportion of the infectious population which is isolated, \( \varphi \), is calculated using an equation (1a)\(^{19} \) that is similar to, but not the same as, what we have called equation (1) in our paper:

\[ \varphi = t(1 - n)l. \]  

(1a)

Since \( t = 0.07 \), \( 1 - n = 0.7 \) and \( l = 14 \), Romer claims that \( \varphi = 0.69 \). This value of \( \varphi = 0.69 \) would, he says, produce his desired value for \( R' \), since:

\[ R' = (1 - \varphi)R_0, \text{ or } R' = (1 - 0.69) \times 2.5 \approx (1 - 0.7) \times 2.5 = 0.75. \]

Drawing on these calculations, Romer (2020b) suggests that there should be testing of 7 per cent of the population each day. Notice that, although the Romer (2020b) lecture mentions self-isolation of those who have symptoms, there is no allowance for such an action in the calculations in the slides.

We think that equation (1a) is incorrect. We say this because in our paper above equation (1) reads \( \varphi = t(l - n)d \). This has the variable \( d \) on the right-hand side, showing the number of days for which a person remains infectious. By contrast equation (1a) has \( l \)

\[^{19}\text{See minutes 16 to 20 of the recording of Romer (2020b), and the accompanying slides.}\]
on the right-hand side—the variable \( l \) which is the number of days that each person who tests positive is placed in isolation.

To see most clearly, and simply, why equation (1a) cannot be right, imagine what would happen if the rate of testing were doubled to \( t = 0.14 \). Then, equation (1a) would give \( \phi = 1.38 \). The person would be in isolation for more than the entire period of 14 days. So, the equation must be wrong.

How can we understand the inclusion of ‘\( l \)’, the number of days that an infected person is placed in isolation, on the right-hand side of this equation? One possibility is that the inclusion of ‘\( l \)’ is simply a mistake. Romer states that \( \phi \) is the fraction of the infected population that are isolated, whereas \( t(1 – n)l \) is the expected length of isolation any infected person is likely to face, after one round of testing. These are clearly not the same thing.

Another possibility is to make a set of assumptions about Romer's set-up that bring the meaning of \( \phi \) and \( t(1 – n)l \) closer together. For instance, consider the following approach. First, interpret ‘\( l \)’ as the ‘number of days that an infected person is infectious’, rather than ‘is placed in isolation’. Second, define ‘\( Z \)’ as the number of infected people not in isolation. Third, imagine there are ‘\( l \)’ periods where, in each period, a fraction \( t(1 – n) \) of those infected people not in isolation, \( Z \), are removed and put into isolation. And finally, assume that in each period the number of infected people not in isolation, \( Z \), remains the same (i.e. the infected who are put into isolation are replaced with newly infected people). Then it follows that, after ‘\( l \)’ periods, \( t(1 – n)I \times Z \) people will be in isolation. Now, \( t(1 – n) \) is indeed equal to \( \phi \), the proportion of the infected population not in isolation who are put into isolation—but with two very significant caveats. First, it assumes that everyone who will be isolated over the ‘\( l \)’ days is isolated on the first day. And second, because \( Z \) is constant over time, it follows that \( \phi \) may also be greater than one if \( t \) or \( l \) is large enough, or \( n \) is small enough.  

Appendix 3: A simple method for calculating the testing threshold when testing is random

In this Appendix we set out a simple method for calculating the threshold testing rate, for random testing, which would reduce the effective reproduction number, \( R' \), to the value at which the disease does not die out. The calculation employs a simple approximation which ignores the dynamics of the infection process. If we ignore the dynamics, we do not have to solve a complex equation like equations (7) or (8) which sum a number of effects in a non-linear way, over many time periods.

Our method of calculation builds on the following insight: for \( R' \) to be less than 1 when there is universal random testing then, on any given day, a person with COVID-19 is more likely to go into isolation than to spread it to someone else. Relying on this

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20 Intuitively, the problem here is that you are taking a fraction \( t(1 – n) \) of the infected population not in isolation, \( Z \), and putting them into isolation in each period—but because \( Z \) replenishes over time, if you isolate a large enough proportion of \( Z \), \( t(1 – n) \), enough times, \( l \), then you will end up with more infected people in isolation, \( t(1 – n)*Z \), than there are infected people not in isolation, \( Z \).
insight, we can ignore the dynamics of the process and simply solve for the value of $t$ for which this condition will hold. We proceed in two steps.

(a) For simplicity, we first examine the extreme case in which none of those who are infected becomes symptomatic and self-isolates; this corresponds to the case considered in section V in which $\alpha = 0$.

Consider any group of $z$, as yet unidentified, infectious people. Assuming that this group is a small fraction of the overall population, the number of people who will be infected by this group on any given day is $(R_0/d)z$, where $d$ is the number of days that an infectious person remains infectious.

The number of these $z$ people who, on this same day, will go into isolation because they have tested positive will be $t(1-n)z$. But there will be additional infectious people who cease to infect others because, although they did not test positive on that day, the period during which they had the disease and were infectious will have come to an end. This happens with probability $1/d$; so there will be $[(1 - t(1-n))/d]z$ such people.21

Thus, for $R'$ to be less than 1, we require that:

$$
\{t (1 – n) + [1 – t (1 – n)]/d \} > R_0/d. \tag{10}
$$

This means that, for this extreme case, the threshold testing rate is given by

$$
t^* = (R_0 – 1) / [(d – 1) (1 – n)]. \tag{11}
$$

If $R_0 = 2.5$, $d = 14$, and $n = 0.3$, we get $t^* = 16.5$ per cent. That is, this method says that, to get $R$ less than 1 by randomly testing the whole population, one needs to test at least 17 per cent of the population. That is, the threshold testing rate, $t^*$, is 17 per cent.

This is a lower value than what we found for $t^*$ using the full dynamic model in section V when $\alpha = 0$. The result there was that $t^* = 19.1$ per cent. The discrepancy between these two results arises precisely because of the dynamic process of the epidemic: the simple calculation carried out here ignores the fact that, as time passes, testing will remove some of the infected people, so that they are no longer available to be tested on later days. That is what made equation (7) so complex.22 For this reason the result produced using this method will always underestimate the required testing rate. This simple method thus provides a (quick and dirty) lower bound for the true value of $t^*$. Nevertheless, the fact that this calculation is so simple, and the intuition provided by thinking about the problem in this way, may make it useful to carry out this calculation.

(b) This calculation can be readily extended to include the more general cases considered in section V in which a proportion of those who are infected become

\[21\] This $1/d$ probability is the chance that an infected person becomes non-infectious independently of testing. An intuitive way to think about this is that, in choosing someone at random, there is a $1/d$ chance that that person is on their last day of infection and so will become non-infectious the following day. This is only an approximation since it requires that the value of $R'$ resulting from testing is equal to 1. That is because if $R' > 1$ then the virus would be spreading and hence an individual would be less likely to be on their last day of infection; conversely if $R' < 1$ then a higher proportion of the infected population would be more likely to be about to end their infectious period.

\[22\] It is possible that this is what Romer (2020b) was effectively assuming. See the final paragraph of Appendix 2.
symptomatic after a certain number of days and so self-isolate. Consider here a proportion \( \alpha \) who self-isolate after a number of days \( d_0 \) out of the total number of days of infection \( d \). We can approximate an adjusted value of \( \alpha \) which we call \( \alpha' \). This is the probability that someone who is infected self-isolates on any particular day (independently of any test or of reaching the end of their infectious period), such that at the end of the infectious period the chance the individual has self-isolated is \( \alpha \), namely;

\[
\alpha' = \frac{\alpha}{d}.
\]  

We then introduce \( \alpha' \) in equation (10) to create a new condition that the testing rate, \( t \), must satisfy for a population following this self-isolation rule:

\[
\{ t(1-n) + \left[ 1-t(1-n) \right]/d + (1-t(1-n)) \left( 1-\alpha' \right)/d \} > R_0/d. \tag{13}
\]

This means that the threshold testing rate is now given by

\[
t^* = \frac{R_0 - d\alpha' - 1 - \alpha'}{[(d - d\alpha + \alpha' - 1) (1-n)]}. \tag{14}
\]

Suppose, as in the previous case, that \( R_0 = 2.5, d = 14, \) and \( n = 0.3. \) Then for a population for whom the first 5 days are asymptomatic, who then become symptomatic and self-isolate with probability \( \alpha = 0.5 \), equation (13) produces a value for \( \alpha' \). This leads, using equation (14), to a correspondingly reduced threshold testing rate of \( t^* = 11.0 \) per cent. In other words, this method says that, to get \( R' \) less than 1 by randomly testing the whole population, one would only need to test 11 per cent of the population because some of the population will self-isolate after 5 days.

This is a smaller value than what we found for \( t^* \) in section V, in this case with \( \alpha = 0.5 \), using the full dynamic model. The result there was that \( t^* = 13 \) per cent. A discrepancy between these two results arises partly for the same reason that it did in the case in which \( \alpha = 0 \): the simple calculation carried out in both cases ignores the fact that, as time passes, testing will remove some of the infected people, so that they are no longer available to be tested on later days. But in addition, in this case here with \( \alpha = 0.5 \) we are making a second simplifying assumption, that there is a ‘random’ self-isolation process \( \alpha' \) each day, such that at \( d = 14 \) days the chance that someone has self-isolated is exactly equal to \( \alpha \). This is as opposed to the detailed model in section V in which, after day 5, there is a step of size \( \alpha \) in the chance of someone self-isolating, so that nobody self-isolates for the first 5 days and then a proportion \( \alpha \) self-isolates for a 9-day period with certainty.

Nevertheless, despite these two simplifying assumptions, it might still be useful to carry out the simple calculation described here, even despite the fact that, in this paper, we are recommending using periodic testing rather than the random testing being discussed here. This is because, as we have shown, periodic testing is more efficient than random testing. As a result, calculating the amount of testing needed to bring \( R' \) down to 1, if testing were to be random, might well provide a lower bound for the threshold testing rate when using periodic testing. It might be useful to have a simple way of calculating this lower bound, even though the method of calculation is only an approximate one. We say this in the light of the current uncertainty about the true value of \( \alpha \) in populations and the strong impact that this will have on the infection rate. In such circumstances, it seems helpful to have a calculation for \( t^* \) which one can carry out quickly for different values of \( \alpha \), without having to solve for \( t^* \) over and over again, using the complex non-linear approach presented in section V.
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