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The measurement of the reproduction number

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Abstract:

The German Robert Koch Institute aims to "protect the population from disease and improve their state of health" (RKI 2017). To this end, it develops concrete, research-based recommendations for policymakers and makes data available to the expert public. Since March 4, 2020, it has been publishing the numbers of coronavirus infections reported by health authorities daily; since March 9, these data have included the numbers of people who have died of or with COVID-19; and since March 25, the RKI has reported the estimated numbers of those who have recovered. The important reproduction number, reported daily since April 7, largely replaced all other criteria used for decision-making, but this was the case only for a few months. Since the second wave of the pandemic, the mere number of new infections and later the incidence number proved to be more plausible and practicable in Germany. This paper aims to show that RKI's reproduction number is neither theory-based nor particularly reliable. Nevertheless, there is a simple way to determine the reproduction number precisely and in accordance with epidemiologic theory. The correct calculated *R*-number could serve as reliable compass leading health policymakers through the months of an epidemic.

Keywords:

Classic epidemic model, reproduction number, contact rate, COVID-19, mathematics of highly infectious diseases, public health policy

JEL-Classification:

C32, C61, I12, I18, J11

1. Introduction

Controlling emerging infectious diseases and planning for an adequate response of the public health policy needs scientific advice based on the knowledge and the methods developed by epidemiology in the last hundred years. The need to predict the temporal and spatial extent of an epidemic and the expected number of victims led to the development of mathematical models since the beginning of the 20th century, which fit the data of a variety of diseases very well (Anderson, May 1991: 1-10, 27-57). Even one of the simplest among these models – the Classic Epidemic Model (CEM) with its classification of three different groups of affected people – enables short- and long-term simulations and forecasts. The model defines an important variable that could play a key role in policy advice under the precondition that its values were calculated correctly. This is the "reproduction number" which should be called more correctly "replacement number" (Hethcote 2000: 603-604). Different approaches for computing the reproduction alias replacement number are well known in the literature. Wallinga and Lipsitch (2007: 599-600) show that they lead to rather different numbers with alternative policy implications. "…such large discrepancies do matter in planning for public health interventions." (600) It will be shown here that insufficient embedding of the calculation method into the CEM makes the parameter almost unsuitable for political advice with the result that it plays a marginalized roll in practical health policy after a few months of experimental interpretation of its meaning.

In this study, the replacement number as it is implicitly defined in the framework of the CEM is derived and then compared with the phenomenological approach that was applied by the Robert Koch Institute (RKI). Section 2 presents a brief history of the official *R*-number, the methodological basis of which has been revised several times, but without changing the principles of calculating the reproduction number. Section 3 introduces some basic concepts and mathematical approaches of quantitative epidemiology. Section 4 recapitulates the core equations of the CEM and explains the logical foundations. Section 5 deals with the definition of the replacement number and discusses its operationalization when applied to real data. In addition, it derives some equations that can be used to statistically determine, for example, the average duration of infectivity. Section 6 confronts the correct replacement number theoretically and empirically with the *R*-number published by the RKI. In section 7 some conclusions are drawn.

2. A short history of Germany's official *R*-number

The RKI started to report the values of its *R*-number[1] and made its reporting public in this situation report:

> The reproduction number is the number of persons in average infected by a case. This number can only be estimated and not directly extracted from the notification system. The current estimation is R = 1.3 (1.0-1.6). This is based on the number of cases with disease onset between 31/03/2020-03/04/2020 and 27/03/2020-30/03/2020 and an average generation

[1] This abbreviation was not used by the media only, but also by the RKI. Of course, it stands for "effective reproduction number" that is different from "basic reproduction number." Both parameters describe essentials of a pandemic and are explained below.

> time of 4 days. Cases with more recent disease onset are not included because their low number would lead to an unstable estimation. (Situation report April 07, 2020)

As a rule, the estimates of RKI's reproduction number reflect states of affairs that dates back at least 3 days. Due to delayed notifications by the health authorities, the infection numbers can be considered being correct after at least three days. Even published numbers are subject to revisions for a longer time span. The revised figures are reported daily on the "dashboard" recommended by the RKI.

In the situation report dated April 13, 2020, the RKI refers to a publication in which the methodological principles of calculating the reproduction number are explained. In this publication, the preparation of the data – re-dating of the cases, smoothing of data by a moving average, and the so-called nowcasting – and lastly the calculation of the reproduction number *R* are described in detail by the authors (an der Heiden, Hamouda April 09, 2020: 10 pp.) The nowcasting is based on a method presented by Lawless (1994). After re-dating the daily numbers of positively tested persons to the putative begin of their disease,[2] the *nowcasting* is applied with the goal to bridge time lags in the reporting, including some smoothing of the resulting projections. The calculation of the *reproduction number* is based on point estimates of the expected new cases. It should be noted that the reported confidence intervals are a result of the nowcasting procedure and not a result of the statistical estimation of the reproduction number although something else is claimed (RKI May 15, 2020: 3). A statistical estimation of the "true" reproduction number of the pool of infectious persons in the population would be highly problematic because the data reflect a special sample that cannot be regarded as drawn randomly or being representative.

In detailing the calculation method of the reproduction number an der Heiden and Hamouda (April 09, 2020: 13) write:

> Bei einer konstanten Generationszeit von 4 Tagen, ergibt sich *R* als Quotient der Anzahl von Neuerkrankungen in zwei aufeinander folgenden Zeitabschnitten von jeweils 4 Tagen. Hat sich die Anzahl der Neuerkrankungen im zweiten Zeitabschnitt erhöht, so liegt das *R* über 1. Ist die Anzahl der Neuerkrankungen in beiden Zeitabschnitten gleich groß, so liegt die Reproduktionszahl bei 1. Dies entspricht dann einem linearen Anstieg der Fallzahlen. Wenn dagegen nur jeder zweite Fall eine weitere Person ansteckt, also $R = 0{,}5$ ist, dann halbiert sich die Anzahl der neuen Infektionen innerhalb der Generationszeit.

Several question marks are to be made here. What do the authors mean by the term "generation time" that is used in biological sciences in different ways? What is the purpose of this concept in this context? Why is the measurement interval set at two times four days?

Anderson and May define the *generation time* in the epidemiological context as follows:

> The period from the point of infection to the beginning of the state of infectiousness is termed the latent period… the sum of the average latent and average infectious periods is referred to as the average generation time of the infection… (Anderson, May 1991: 14)

Wallinga and Lipsitch define the generation time implicitly in the following passage:

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[2] To calculate the onset of illness, reported cases are backdated between 5 and 10 days. (an der Heiden, Hamouda 2020: 11) The reported *R*-numbers based on these figures have a time-lag of four days (RKI May 15, 2020: 3).

> The observed value of the growth rate r [of the group of infectious people – GQ] can be related to the value of reproductive number R through a linear equation: R = 1 + rTc … Here, Tc is the mean generation interval, defined as the mean duration between time of infection of a secondary infectee and the time of infection of its primary infector (sometimes this is called the serial interval or generation time). (Wallinga und Lipsitch 2006: 599)

an der Heiden‘s and Hamouda’s understanding is slightly different from the last cited one with replacing the “secondary infectee” – a person who has been infected – with the plural “secondary infectees”:

> Die Generationszeit beschreibt die mittlere Zeitspanne von der Infektion einer Person bis zur Infektion der von ihr angesteckten Folgefälle. Diese Zeitspanne schätzen wir auf etwa 4 Tage… (an der Heiden, Hamouda 2020: 13)

Apparently, it is supposed that there are no further infections originated by the primary infector after the 4-days generation time. In other words, what the authors mean is identical with the average duration of an infected person being contagious to others. As a matter of fact, the authors could not and cannot know the length of infectivity because even after 12 months of pandemic the virus has not yet been studied in enough detail to determine this number. But they could know the preliminary estimations made by their “own” institute and adjust their assumption correspondingly. In the “profile” published by the RKI six month later we read that the infectious time lasts up to ten days and more:

> Der genaue Zeitraum, in dem Ansteckungsfähigkeit besteht, ist noch nicht klar definiert… Bei mild-moderater Erkrankung geht die Kontagiosität 10 Tage nach Symptombeginn signifikant zurück … Bei schweren Erkrankungen gibt es Hinweise, dass die Patienten auch noch deutlich später als 10 Tage nach Symptombeginn ansteckend sein können. (Steckbrief/Profile Oct. 16, 2020)

This contradicts the assumption of a 4-days “generation time.”

Meanwhile the authors have changed the method two times. The first change happened on April 30, included the elimination of the 4-day generation time, and replaced it by a 4-day average:

> The number of new cases estimated during the nowcasting process was previously presented as a moving 3-day average to compensate for random effects of individual days. Since April 29, 2020, the RKI has been using a 4-day average, which smooths the course of the bar chart to a certain extent… The result of the R-estimate does not change thereby. Due to the smoothed course of the nowcasting, the calculation of the point estimator of R can be performed in fewer steps. For a given day, this value is now calculated as a simple quotient of the number of new cases for this day divided by the number of new cases 4 days before. (Daily Situation Report, 2020, April 30)

By the way, there was never made a moving 3-day average public. – The next correction was jot down on May 14:

> The R-value reported to date reflects the trend in the number of new cases and can indicate possible changes in trend. However, this value is sensitive to short-term changes in the number of cases – such as those caused by individual outbreaks – which can lead to relatively large fluctuations, especially in the case of a small number of new cases. In addition to this

> sensitive R-value, the RKI therefore now provides a second more stable 7-day R-value, which refers to a longer period of time and is therefore subject to less short-term fluctuations. (Daily Situation Report, May 14, 2020)

This is a change of the averaging method applied in the nowcasting, but not a change of the principles underlying the computing of the reproduction number. Originally, the two authors of the RKI were convinced that the generation time of the Corona virus is equal to four days. This proved to be wrong, but the number of four days was upheld. The term "generation time" was abandoned and replaced by an unfounded 4-day average. This method was applied in the following months and is still in use (April 2021). In addition, a 7-day average was added that comes closer to the observed average duration of infectivity.

The method as hitherto described lacks a theoretical foundation and does not utilize all the empirical possibilities to control the development of the disease statistically. This might be the reason that another explanation of the estimation-procedure (RKI Erläuterung, May 15, 2020) was published which included a reference to Cori et al. (2013) as theoretical foundation. These authors write about their own method:

> The estimation method presented above is developed for the ideal situation in which times of infection are known and the infectivity profile w_s may be approximated by the distribution of the generation time (i.e., time from the infection of a primary case to infection of the cases he/she generates). (19).

This ideal situation is not given.

> Therefore, in practice, we apply our method to data consisting of daily counts of onset of symptoms where the infectivity profile w_s is approximated by the distribution of the serial interval.

However, even after one year of pandemic, neither the length of the "serial interval" nor the associated distribution function is known. Therefore, an der Heiden and Hamouda replaced the unknown distribution function by 4- and alternatively 7-day averages. This is a very crude approximation to the "infectivity profile." This approach could and should be discarded if a more appropriate model is available.

3. Concept formation and basic mathematical approaches

In the case of a dangerous pandemic like Corona[3] an important task of the health system is to identify, isolate, and register people that are infectious, and in addition, of course, to take care of them and to treat them medically if necessary. As long as no immunity of at least two thirds of the population has reached – mainly to be achieved by vaccination – the goal of almost all policy measures and rules consists in inhibiting the transmission of the virus from the infectious to those who are susceptible to it. In the pre-immunity situation deadly infections "can only be avoided if all persons prevent the spread of the SARS-CoV-2 virus with the help of infection control measures" (RKI Febr. 24, 2021: 2) – such measures as social distancing,

[3] "Corona" is a short general term for a highly infectious and to about 3 percent of the affected people deadly disease exactly named COVID-19 (Coronavirus Disease 2019) that is caused by SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus type 2) and by its variants.

hand washing, mask wearing, ventilating indoor spaces, testing, contact tracing, quarantine measures and border closures.

Most people are immune a few weeks after they have been infected, especially when they recovered from an outbroken disease. Alas, how long this immunity to further infections lasts is still not known.

Corresponding to a variety of epidemic diseases, a flexible compartmental model has been developed that comprises all groups of a population that can be distinguished epidemiologically. For Corona, and more generally "for … infective agents, it makes sense to divide the host population into relatively few classes of individuals: susceptible, infected, recovered-and-immune." This is the so-called *SIR-model* of infectious diseases. If necessary, "greater detail and realism can be achieved by adding more compartments or categories to the model…" (Anderson, May 1991: 13-14; more developed Hethcote 2000: 601) The simple SIR-model is an appropriate theoretical approach to Corona whilst our knowledge on this disease is still very fragmentary (RKI Steckbrief/Profile).

Apportioning the total population into at least three groups according to the infection status of the people as either being susceptible, infectious, or recovered leads directly to the basic variables of epidemiological models. We symbolize N as the population size; S is the number of people susceptible to infection (at the beginning of an epidemic and without information about the number of immune people in the population, it is set as equal to N); I is the number of infectious people (with an initial value close to zero; be aware that people who belong to this group are not only infected, but infectious, i.e. contagious);[4] and R is the number of people who recover and are assumed to be immune and not infectious anymore (without information, this number is set at zero at the beginning of a disease). The exact conceptual characteristics of these three population groups are not entirely unimportant because the "susceptible" need not be "healthy" and the "infected" need not be "sick" (Donsimoni et al. 2020). Whether this model can be applied to COVID-19 depends mainly on the question if those who recovered are immune and not contagious anymore, at least for a while.

Corresponding to the availability of data these variables can be construed as changing with *time* (dynamic models) or changing with *age* of the considered groups (static models) or changing with both. Because the goal of this study is a discussion of the special R-number published by the RKI we focus on dynamic modelling and assume the variables being integrated over all ages but changing with time.

There is another important difference that divides epidemiological models into two comprehensive classes, the classes of *deterministic* and of *stochastic models*. The stochastic version of the SIR-model comprises the same variables as the deterministic one but is focused on probability functions which are difficult to ascertain even when postulating constant parameters: "An exact calculation of the probabilities would be extremely laborious in all but the simplest cases." (Bailey 1975: 90) In principle, the CEM presented below is a deterministic model; however, parts of the model can be treated econometrically and are then

[4] The distinction between persons who are infected and at the same time contagious and those persons who are infected but no longer contagious is central to understanding epidemiological models. In the referred epidemiological literature, the corresponding concepts that are based on this difference are "infectious" and "infected". It is purely a problem of classification and has nothing to do with dark counts or problems of measurement-adequacy.

basically stochastic[5] – but not in the sense of that class division whose core consists in the determination of probability functions.

4. The mathematical model

The Classic Epidemic Model is given by the following system of differential equations (Hethcote 2000: 604):[6]

(1) $dS/dt = -\beta IS/N$, $\quad S(0) = S_0 \geq 0$
(2) $dI/dt = \beta IS/N - \gamma I$, $\quad I(0) = I_0 \geq 0$
(3) $dR/dt = \gamma I$, $\quad R(0) = R_0 \geq 0$
(4) $N = S(t) + I(t) + R(t)$

Even under the assumption of a constant population number the system can be formulated in slightly different ways when it comes to the task to find an easy method to solve the system (Anderson, May 1991: 122; Bailey 1975: 33-34). Here, β and γ are parameters explained below. Solutions of the system under arbitrarily given parameter values are often published as projections and simulations of the further course of an epidemic.[7] Our concern is quite different; we are interested in the hidden replacement (alias reproduction) number that is implied by the equation system and determined by real data.

Because the available data set is discrete in time ($\Delta t = 1$ day) the differential equations are modified as difference equations:

(1a) $\Delta S = -\beta IS/N$
(2a) $\Delta I = \beta IS/N - \Delta R$
(3a) $\Delta R = \gamma I$

The rationale behind this model becomes clear when defining fractions of the two population groups with and without active pathogens in the total population: $i = I/N$ and $s = S/N$. These fractions can be interpreted as empirically given probabilities of accidentally encountering an infectious or a susceptible person in the population.[8] The probability that these persons meet each other has the value $i \cdot s$ (Hamer 1906). The frequency of infections is assumed to increase with the size of the population – the so-called "mass action principle" (Anderson, May 1991: 7) – resulting (with exception of the parameter β) to the term in the middle of equation (2a) which we abbreviate as *H*:

(5) $\frac{IS}{N} = \frac{ISN}{N^2} = isN = H$

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[5] This is true even if only a simple average is taken from the observed data.

[6] The CEM was taught by an der Heiden in 2007.

[7] A sophisticated model endowed with some compartments and other extensions used for simulations is presented by Kühn et al. (2020). – This is only one example among a variety of similar models.

[8] This interpretation recures on the assumption of a homogeneous mixing of susceptibles and infectives. Otherwise see Bailey 1975: 75.

From a practical perspective, *H* can be interpreted as the number of possibilities to become infected in a population of the extent *N*, so to speak, the "abstract risk situation."

The contact rate, β, reflects the actual infection process with regard to how many people are infected by an infectious agent per unit of time (here: per day) on average. This depends on a variety of factors that are not explicitly included in the model, such as population density, the number of daily interactions, and common behaviors (hygiene, shaking hands, etc.). Some of these factors can be influenced pragmatically so that policymakers can intervene into the ongoing process.

A further element of the equation system is the implicit model of exponential growth (and decay). "For example, the transfer rate γI" – the "transfer" from the group of infectious people with the amount I to the group of non-infectious ("recovered") people – "corresponds to $P(t) = e^{-\gamma t}$ as the fraction that is still in the infective class t units after entering this class and to $1/\gamma$ as the mean waiting time." (Hethcote 2000: 603)

There is a further variable that does not play an explicit role in the above-formulated model, and this is the number of deaths, *D*. The model has been interpreted (an der Heiden, Buchholz 2020: 1) in such a way that the number of deaths is included in the number of recoveries *R*. But if the number of deaths is empirically available, the model should be supplemented with it. Both the number of deaths and the number of recovered people reduce the number of infectious cases; therefore, equation (2a) can be made more precise:

(2b) $\Delta I = \beta H - \Delta R - \Delta D$

The number of recovered people is determined by equation (3a). For the number of disease related deaths, the analogous assumption of exponential growth applies to D as to R:

(6) $\Delta D = \delta I$

The parameter δ measures the current number of daily reported deaths related to the number of infectious people. With the parameter δ, the fraction that is still infective after the time *t* (ignoring the new cases) is equal to $e^{-(\gamma+\delta)t}$ with the "waiting time" (the average duration of infectiousness) *T*:

(7) $T = 1/(\gamma + \delta)$

Deaths reduce the population. Therefore, in the case of a short-term event with potentially high numbers of victims, it makes sense to replace the constant population figure by a variable one: [9]

(4a) $S(t) + I(t) + R(t) = N - D(t) = N'(t)$

It should be noted that the accumulated number of infected persons reported by the RKI does not correspond to the number of infectious persons, $I(t)$, which plays a central role in the

[9] A model specified for the long run should additionally include an average birth rate and a death rate that is not related to the disease. – Another model with a separate treatment of immune and gone people coupled with a constant population number can be found in Anderson and May (1991: 58).

classic model.[10] The following equation makes the number of *new infected people* in equations (1a) and (2a) explicit:

(8) $\Delta A = \beta H = \beta IS/N'$

From this follows the total number of infected persons, more exactly, the number of positively tested persons, a number that is well known to the reader because it is reported daily by the media:

(9) $A = \sum_t \Delta A(t)$

5. Derivation of the so-called "reproduction number"

According to Hethcote (2000: 603-604), the reproduction number should be better called "replacement number" because of the danger to confuse it with the basic reproduction number. Conceptual clarity would afford the name "replacement number," as can be seen in this section. It is regrettable, but no serious harm to the attentive reader, that the replacement number *R*, defined by the CEM, uses the same symbol as the number of recovered people. To be as unambiguous as possible, the replacement number defined in the framework of the CEM is named R_{CEM} (citations excepted). Here is its definition:

> The replacement number R is defined to be the average number of secondary infections produced by a typical infective during the entire period of infectiousness… (Hethcote 2000: 603-604)

Let us assume that the pool of infectious people does not change quantitatively, i. e. $I = const.$ In this case, every person that leaves the group *I* has infected exactly one other person and is replaced (!) by this infected person. Of course, this is valid on average only. This special situation defines the replacement number 1 (one). In other words, the equilibrium between inflow to and outflow from the group of infectious people defines the replacement number $R_{CEM} = 1$. Setting $\Delta I = 0$ in equation (2b), this equilibrium is quantitatively characterized by the following relation:

(10) $\beta IS/N' = \gamma I + \delta I$

Now let us suppose that one typical infectious person infects on average $x \geq 0$ other persons before leaving the group of infectious; it follows that the number of new infected people is x times higher than the number of persons leaving the group *I*:

(11) $\beta IS/N' = x(\gamma I + \delta I)$

[10] The RKI introduced daily reports of the number of infectious people under the title "active infected persons" with a delay of eight months, exactly at Nov. 11, 2020.

According to the above cited definition, x is identical to the replacement number if x is the number of persons infected by a typical infective during his or her infectious period *on average*. From this we derive the *replacement number*:

$$(12)\quad R_{CEM} = \frac{\beta IS}{N'\left(\gamma I + \delta I\right)} = \frac{\beta s}{\gamma + \delta} = \frac{\Delta A}{\Delta R + \Delta D}$$

Under the condition of changing health protection rules the parameter β is no longer a constant one and must be considered as a variable. The same can be said on the replacement number R_{CEM} that is also called "effective reproduction number". Its changes depend mainly on the slow changing share of the susceptibles at the population, *s*, and the quicker changing contact rate β. Taking equation (7) into account, (12) can be written as:

$$(13)\quad R_{CEM} = \beta s T$$

With the other parameters measured, this equation can easily be used to determine the average infectious period *T*. With the data delivered by the RKI, I found $T \approx 11$.

As already mentioned, if it is not known at the beginning of the pandemic how many persons are immune by their own nature the number of susceptible persons *S* is set to *N*. In this case, it is $s_0 = 1$, and we get what is definitively and unambiguously called in the epidemiological literature "basic reproduction number" symbolized by R_0:[11]

$$(14)\quad R_0 = \beta T = \frac{\beta}{\gamma + \delta}$$

With an alternative definition of the *contact rate*, β, as a per capita parameter, and with using other Greek symbols, the same mathematical relation can be found in Anderson and May (1991: 75/4.40; 123/6.8) and in Hethcote (2000) if we ignore the absence of δ.[12]

Equation (12) can be used for a simple estimation of the replacement number that is implied by the system of difference equations and determined by the observed cases of infections because all the needed numbers are given. To balance the weekly fluctuations a 7-days average is appropriate (see Fig. 2 below). The result must not be interpreted as estimation of the "true" replacement number of the population because the underlying data are yielded under very restricted conditions. For instance, most people need to show symptoms before being tested. It is believed that the true numbers of infection are 2 to 34 times higher than the observed ones (RKI Steckbrief/Profile, Oct. 16, 2020). If the real, but unobserved number of the infectious is *y* times higher than *I*, the real number of partly unobserved recovered and partly unobserved Corona-connected deaths are also *y* times higher. As long as the preconditions of testing are not changing very much during the measurement period (the days of averaging) the dark figure, *y*, does not have an effect on the replacement number because the factor *y* affects both, the numerator and denominator of equation (12), in the same way. Nevertheless, the problem of an unknown bias in the reported data remains.

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[11] There are different methods and arguments to usher the basic reproduction number. I found the simplest ones in Andersons and May (1991: 17).

[12] Hethcote (2000) defines the replacement rate at time zero by σs_0 "which is the product of the contact number σ and the initial susceptible fraction s_0." (607) "The contact number $\sigma = \beta/\gamma$ is the contact rate β per unit time multiplied by the average infectious period $1/\gamma$." (606)

The number of infectious plays a crucial role in the spread of a deadly disease. It is a determining factor in all the basic equations of the CEM. The replacement number serves to control this group. Another possibility for controlling the group of infectious people is its growth rate *k*:

$$k = \frac{\Delta I}{I} = \frac{\Delta A - \Delta R - \Delta D}{I} = \frac{R_{CEM}\left(\Delta R + \Delta D\right) - \Delta R - \Delta D}{I}$$
$$= \frac{\left(R_{CEM} - 1\right)\left(\Delta R + \Delta D\right)}{I} = \left(R_{CEM} - 1\right)\left(\gamma + \delta\right) = \frac{R_{CEM} - 1}{T}$$

From this it follows:

(15) $R_{CEM} = \frac{k}{\gamma + \delta} + 1$

Equation (15) corresponds to the equation (3.1) that are recommended by Wallinga and Lipsitch (2007: 601) while using the concept "generation time" as a definition of *T*.

6. Comparing the *R*-numbers

6.1 Theoretical comparison

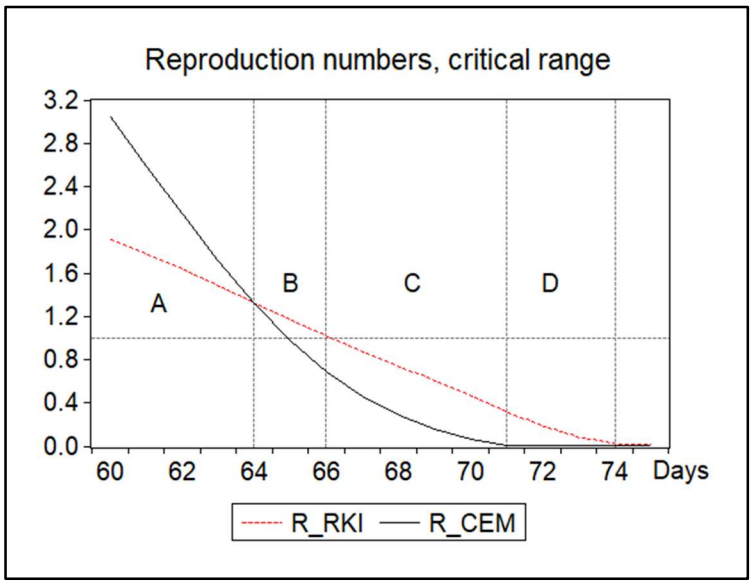
The numbers of new infected people and their development plays a crucial role in Germany. The leading institute for advising health policy uses a bunch of parameters to assess the situation, but mainly the numbers of new infected persons back up its policy advice. As already mentioned, the RKI changed its computing method of the *R*-number several times. For comparing it with the replacement number that was derived from the CEM we endow the two parameters with an index and assume that the case numbers are identical with the (by nowcasting) adjusted number of new infections. Moreover, we focus on the 4-days average. With the help of these simplifying assumptions, a theoretic comparison between R_{RKI} and R_{CEM} can be made explicit very briefly using the following formula (Quaas 2020):

(16) $R_{RKI}\left(t\right) = \frac{\sum_{j=0}^{3} \Delta A_{t-j}}{\sum_{j=4}^{7} \Delta A_{t-j}} = \frac{MA\left(\beta H, t\right)}{MA\left(\beta H, t-4\right)} \approx \frac{\overline{\beta}_t}{\overline{\beta}_{t-4}} \neq \overline{\beta}_t s_t T = R_{CEM}\left(t\right)$

Under the realistic assumption that the frequencies H does not change very much in the regarded time span, the fraction in the middle of the equation can be shortened, and it can be seen that the RKI does not calculate the replacement number defined by the epidemiological model according to equation (12) but the change of the averaged contact rate β.

In the critical situation around the equilibrium of inflow to and outflow from the group of infectives, contradicting conclusions can be drawn from different *R*-numbers that are expressions of the same epidemic curve:

Figure 1: Theoretic comparison of the reproduction numbers



Fictitious data; own computations with the CEM

There are the following ideal-typical situations:

A: The replacement number is higher than the increase in new infections. The distance between the two indicators becomes smaller over time.

B.1: While the increase in the number of new infections is still positive, the reproduction alias replacement number is passing the critical value of one, signalizing that the disease is under control.

It is possible that the intersection point of the two curves is below $R = 1$. Then the following applies:

B.2: While the replacement number is still positive, the increase in the number of new infections has passed the critical value of one, signalizing erroneously that the disease is under control.

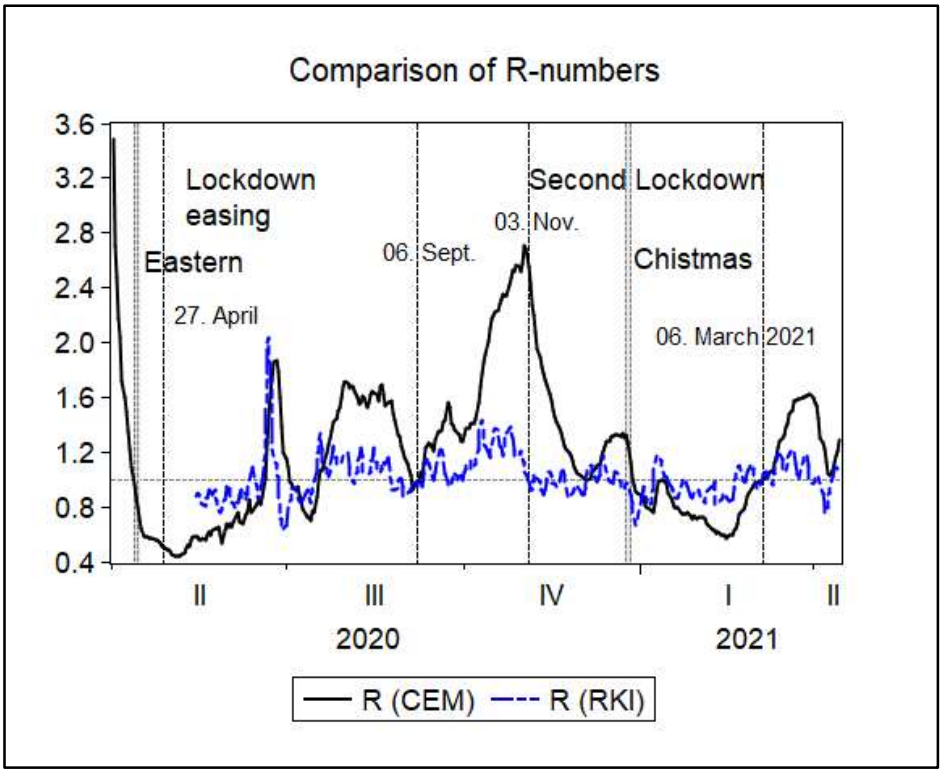
C: The replacement number is significantly lower than the increase in new infections.

D: The danger has passed, but there are still new infections and thus an increase greater than zero.

6.2 Empirical comparison

A comparison of R_{CEM} und R_{RKI}, both based on the observed development and calculated with 7-day averages over the periods of the first and the second wave of CORONA-19 in Germany, shows which of the theoretically possible situations turned out to be frequently real:

Figure 2: Comparison based on a 7-days moving average



Data source: RKI; own calculations

A presentation of the confidence intervals reported by the RKI can be dispensed with, as these did not play a role in the public interest. Even more important is the fact that RKI's confidence intervals do not originate from estimating the *R*-number but are an upshot of its nowcasting.

As can be seen, the *R*-number published by the RKI is much more volatile compared to the replacement number defined by the CEM and computed by a simple moving average based on equation (12). The assumption that estimates using raw data are somewhat more prone to reporting artifacts does not hold (an der Heiden, Hamouda 2020/version 4: 15). Even though both curves rely on a 7-day average, the RKI-numbers are fluctuating with a 7-day frequency. One is tempted to average these figures once again for making the inherent trend more visible. This trend seems to coincide with the curve that represents R_{CEM}; to what extent tell us the correlation between the two time-series, $r = 0.68$ (298 observations, probability = 0.00, time-lag adjusted).

The RKI-numbers seem to have the advantage of signalizing a change in the development a few days earlier than the correct replacement number. But this is the result of the re-dating of the cases according to the putative onset of a disease. Although the lead is partly compensated by the time-lag of the *R*-number, the re-dating makes it seem as if the information was already available earlier than it really was.

It can also be seen in Fig. 2 that the values of R_{RKI} are often located nearby the critical realm around $R = 1$. To these cases, Schaade (RKI update May 12, 2020: press conference)

explained: Only when *R* is "permanently" and "clearly" above 1 – such as 1.2 to 1.3 – this is a signal that one is again in an exponential course and must take countermeasures. In the rather frequent situation fluctuating a bit below and above one, R_{RKI} does not tell us in which direction the case numbers are moving. The R_{CEM} makes the direction of the development clear by moving in the short time of a few days towards its more extreme amplitudes.

7. Conclusion

Under the authoritative expert advice of the RKI, German policymakers have several times succeeded in decisively reducing the contact rate in a relatively short time and, thereby, containing the spread of the first and the second wave of the coronavirus epidemic. When the numbers are down after a lockdown it was and will be important to prevent them from rising again, if possible, while easing of restrictions simultaneously. In view of the high and, likely, increasing economic costs of each additional day of contact restriction, it is advisable to use a theoretically well-thought-out model to calculate the contact rate and the corresponding replacement number, which reflect the actual pandemic situation unambiguously. The classic epidemiologic model allows stable estimates, signals clearly rising or falling trends in the development of the observed pandemic data and is transparent because it can be calculated in a simple way utilizing available data.

The rationale of RKI's method is inappropriate. It controls the inflow to the group of infectious people and ignores the observed outflow by implicitly postulating that an infected person is not contagious any more after 4 or 7 days. Thus, the calculation method contradicts the RKI's own findings about the duration of infectivity and ignores RKI's estimation of the number of recovered and gone people.

The "reproduction number" published by the RKI is nothing else than the change-rate of the number of new cases projected by nowcasting and averaged over four and/or seven days.

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