Value of anarchy — correlation between compliance to health-care guidelines and actual risk benefits society.

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During pandemics such as the current one, fake news that dismiss the severity of the pandemic can instigate disregard to health-care guidelines in some of its consumers. Measures to reduce the level of infectious contacts in society such as wearing of protective masks, quarantines, limiting social gatherings, thus encounter different levels of compliance by the general public. The basic premise of many decision makers and advisors is that public disobedience leads to an increase in the number of infectious contacts, leading to more load on the health care system, and eventually to increased mortality. From this stand point, spreading of dismissive fake news is bad. This conclusion however, fails to account for the inhomogeneous distribution of risk in the population and the possible correlation between individually perceived risk and individual compliance to health-care guidelines and regulations. People that know they are at risk tend to be more compliant to health-care guidelines and people with low-risk can afford to be less compliant. Here we find that if the converse is also true and individuals at low risk have more contacts with their peers, this has a positive effect for society, and depending on the level of correlation between risk and risk aversion behavior, this can lead to a reduction of up to 50% in hospital load and overall mortality. This effect which we dub 'the value of anarchy' is because such correlations cause the pandemic to spread in-homogeneously in the population, moving faster through the not-at-risk portion of the population and only later penetrating to the population at risk. Thus, if risk aversion and risk are positively correlated, e.g. by correlating compliance and risk, the resulting age-dependent disobedience is beneficial to society.

Epidemiology | Game theory |

In economics and game theory, the price of anarchy is a measure of degradation in system's performance due to the behavior of its selfish constituent agents each seeking its goal [2]. In the context of the SARS-Cov-2 pandemic spread, a common opinion which we held prior to this research, is that individuals that disregard health-care guidelines such as wearing protective masks and avoid social gathering are causing harm to society, by increasing the rate of infections which eventually leads to more load on hospitals and a higher death toll. This simple argument can be formulated mathematically. In a simple compartmental model, there are susceptible individuals not yet infected, there are infected individuals, some of which develop severe symptoms that require hospital admission, and a fraction of those admission will subsequently die. Other infected individuals eventually recover and develop immunity. In this highly simplified yet prevalent model, increasing the rate of infection causes more load on hospitals and increase the death toll, thus leading to the conclusion that it is best to reduce the infection rate in the population.

A major shortcoming of the above model is the neglect of two important factors that can strongly affect the spread of the disease, the resulting load on hospitals, and the overall death toll. The first factor is the existence of an inhomogeneous basal risk level of individuals in the population, which in case of Covid-19 is strongly correlated with age, BMI and background diseases. The second factor is the risk aversion strategy or lack thereof, chosen by individuals in the population in response to two prevalent types of informations available to them — news and the so called fake-news. Here we refer to news as any means to deliver reliable data on the spread of the disease, the load on the hospitals and the actual death toll. By fake news we mean inaccurate or incorrect data. Of course, in reality it is sometimes hard to differentiate between the two types, e.g. because a single sources can distribute both, increasing the confusion.

Here we show that the optimal compliance strategy depends on the society's structure. When the number of individuals at risk is low- more is to be gained by protecting the vast majoritty of lower-risk groups, and neglecting the risk groups.

We measured the spread of an epidemic among a population we generated. we measure the number of people hospitalized for the different parameters, the number of people infected and the general infection rate. we compare between the results of three models: a) the classic ODE model following SIR equations. b) the agent-based random graph model, in which the epidemic spreads on nodes of a d-regular graph. c) the structured graph.

Results when the majority of the population driving the infection isn't at risk, the question arises: is protecting the wider population is more favorable than protecting those at higher risk?

Significance

In economics, the price of anarchy is a measure of degradation in system's performance due to the behavior of its selfish constituent agents each seeking its own goal. Here we show that correlation between the risk aversion strategy of an individual and the actual risk it faces during a pandemic can lead to an opposite effect, whereas some level of anarchy may benefit society. This effect occurs when low risk individuals do not comply to social distancing regulations with their low-risk peers, causing the pandemic to spread non-homogeneously along less risky portions of society first. Both hospital load and overall death toll can be reduced by up to 50 percent, compared to the uncorrelated case.

Reserved for Publication Footnotes

In our research, we tested the affects of matching those at higher risk of death, with those who choose to protect themselves. We compare the overall death rates and strain on hospitals for different proportions of Risk Groups within the population, and the influence of the decision of matching those at Risk, and those who choose to take caution. We apply estimated parameters for the virus of COVID-19. Our results are as follows: There are two competing effects: 1. Behavioural effect: Those who choose not to protect themselves spread the virus more readily. 2. Medical effect: Those who are at higher risk, often are the main victims of the virus. When the part of the population at High Risk is below a certain threshold, then it is preferable to protect the larger, not-cautious group within the population. However, when the high-risk group is larger than that threshold, it becomes preferable for lowering the overall virus death toll, for the groups at risk to protect themselves. This result holds for both the goal of lowering Mortality and reducing stress on hospitals, although the threshold itself differs. We validated and compared our work for an agent-based simulation, for two graphs: a D-regular random graph, and for a structured graph, with a clustered structure imposed, simulating the population of Israel. We found that when comparing the peak stress on hospital systems, the numerical model, and the structured model give similar results. When comparing for death toll, we find that by applying a structure we shift that threshold slightly, and that there is large dependance on the initial infectors and the results are quite noisy.

A key change, is that in the case of negative matching, or when the risk group isn't cautious, the risk groups fill the hospitals. When the matching is positive, there are many hospital patients who are not at risk, even though upon infection they are 10 times less likely to be hospitalized.

Graph percolation:

We find the following: When those not at higher risk (who are the majority in this case), choose to lower their connections, by protecting themselves, the overall spread of the virus through the population is sometimes negative - the virus actually dies out, however before it does so, the high-risk minority, who aren't cautious, will get sick more, and can in turn actually have a higher death toll.

By imposing a structure on the graph, such as in ours, reflecting a general society, causes the infection to spread more quickly as a general rule. The structure causes clustering to appear - families are densly clustered sub-groups, and infection within them is faster. This causes the epidemic threshold to be reached faster.

The decision threshold for lowering the death toll is higher than the limit for lowering the peak hospital capacitiy. Here, we present the results for the agent-based simulation for when the risk-group proportion is 0.3 and 0.4. There are essentially two negating effects here, when matching between Risk and caution: When the Risk group is cautious, they, who will be the primary victims of the Virus, are safer. On the other hand, when the risk-group aren't cautious - the majority of the population - at lower risk, slow the spread of the virus. when the part of the population that is initially in a high-risk group is 40 percent of higher, the preferable option is to protect the risk groups. however, if the risk group is 30 percent , the better strategy to lower the death toll is to protect the non-risk group, as they are a larger part of society. If trying to lower the stress on the hospitals, as a limited and critical resource, then we find that at 30 and 40 percent it is preferable to protect the wider, non-high risk population. In this case, unless the portion of risk-groups is larger than 45 percent, then the low-risk groups fill the hospital.

For example: if we take the part of society aged over 60 to be the portion at high risk, then in Israel that equates to around the 30 percent option, wheras in Italy, the portion is closer to 40 percent meaning it would be better (so as to lower the death toll) to protect the general population. In both countries the toll on hospital systems will be high. The key to this difference is that when the group at risk is small, by protecting the wider population, the virus doesn't spread as the reproductive coefficient is less than 1. We assumed here that there is no prejudice of contacts of risk-groups towards each other- that they are randomly scattered throughout the population. The "match coefficient", we mark as C, reflects that lowering in the strengths of links between these groups in the population.

Method The number of cautious individuals of the population isn't stable, the lower the "correlation", the more healthy individuals choose to be protected. we use the equation:

$$R = \beta/\gamma$$

to estimate and then measure the spread rate ergo: the exponential rate of healing is slower than that of infection.

risk-caution division: We split the population into four groups, using the following two criteria: a. cautious - how much one chooses to protect him or herself, and comply with the regulations advised to avoid infection. b. risk - whether or not one is at higher risk rate of death upon infection. in our model we consider each individual to be binarized to either be cautious or not cautious, and at high risk or not, and no middle ground. each person is initialized randomly into these states, 2 sets of binary criteria- 4 groups overall. the distribution of this decision follows the following development:

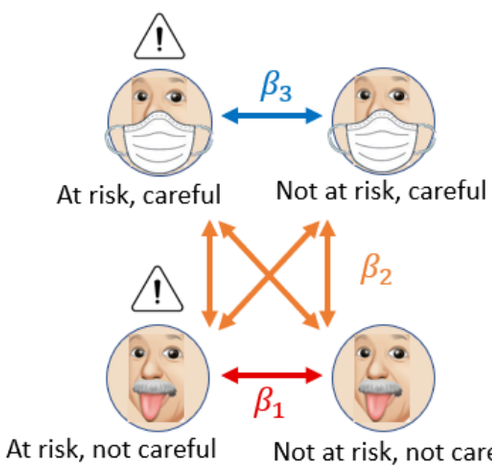

Fig. 1. Schematic diagram of the bacterial metabolic network showcasing different auto-catalytic cycles. They are all coupled through ribosomes, at the point of translation. Color codes are for the different classes of loops.

we marked the match between cautiousness and sensitivity as "correlation": given that an individual is at high risk - what is the probability that he is cautious, while if he isn't at high risk - what is the probability that he's cautious?

$$P(B|S) = P(nB|nS) = P(B \cap S)/P(S) \qquad [1]$$

we scale by a factor of 2 and subtract 1, to get values between -1 and +1.

for correlation -1: all risk are not cautious, e.g. not risk ⇒ is cautious

correlation +1: risk ⇒ cautious, not risk ⇒ not cautious

for correlation 0: the sensitivity and caution is independant.

$$P(B|S) = P(B) \quad [2]$$

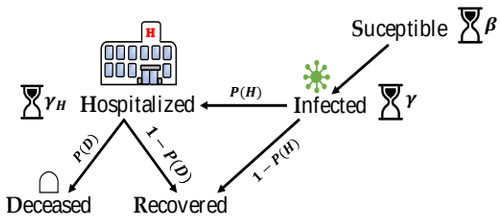

Fig. 2. Schematic diagram of the path of an individual in the network. P denotes probability, and the hourglass denotes exponentially generated time delay.

development of SB correlation matrix: if we go to the matrix of caution-risk division we find: $\begin{bmatrix} SnB & SB \\ nSnB & nSb \end{bmatrix}$ we define to be: $\begin{bmatrix} s*(1-c) & s/2*(1+c) \\ (c+1)/2\cdot(1-s) & 1-(c+1)/2 \end{bmatrix}$

development of SB matrix:

$$c' = \frac{c+1}{2} \quad [3]$$

$$P(S \cap B) = c' \cdot P(S) \quad [4]$$

$$P(S \cap nB) = (1-c') \cdot P(S) \quad [5]$$

$$P(nS \cap nB) = 1 - c' \quad [6]$$

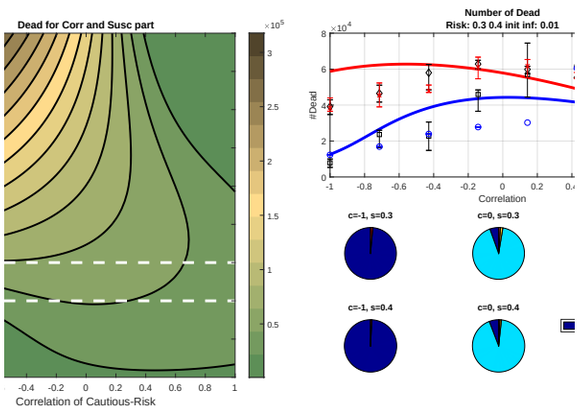

Fig. 3. Left: map of the number of Deceased for different Risk group parts and Cautious-Risk matches. Upper right: No. of Deceased, compared to structured and agent-based simulation. Lower right: Pie graph of which group the Dedeacsed belong to.

SIHRD

in our model, each individual can be in one of five following states:

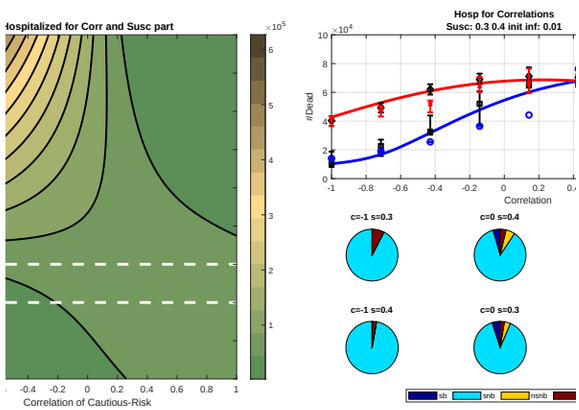

Fig. 4. Left: map of predicted peak hospital capacity for different Risk group parts and Cautious-Risk matches. Upper right: Peak hospitalization capacity, compared to structured and agent-based simulation. Lower right: Pie graph of which group the hospitalized individuals belong to.

susceptible - can be infected, infected - is infecting others, hospitalized - cannot infect others but is straining critical health resources, recovered - either from infected or from hospital and finally deceased - can no longer affect anyone.

we assume that for the relevant simulation period, upon recovery one is rendered immune.

we measure the output for two criteria: the number of deceased overall, and the maximum number of people hospitalized. the number of deceased- for obvious reasons one wants to know the outcome, also the stress on the health system is critical, as if the hospitals becomes over-full the death toll could be raised significantly.

Description of our model model: ode model: as mentioned before, we have divided the population into four states of reponse and position relative to the epidemic: cautious-not cautious, risk - not risk. we used the codenames: sc for the risk and cautious group, snc for the sensitive yet not cautious group, and such forth: [sc,snc,nsnc,nsc]. each of those in turn is distributed over five states: susceptible, infected, hospitalized, recovered and deceased. thus our state vector is of 20 states, and we use an SIR-based ODE model: for us we have so we have an "SIRHD" model.

parameters of our model:

at each stage, the time one will spend in a state is generated exponentially. here we define the rates of transition.

beta: the infection rate, from a susceptible person meeting an infectious person, with 3 possible options: cautious-cautious, cautios-not cautious (or vice versa) and not cautious - not cautious.

we find offer the following development ot beta: beta being the rate at which new infections occur, needing to be measured relative to gamma, refering to eqn. 1. we get the following equation:

$$B' = \beta \cdot (I_B B \cdot P(BB) + I_B nB \cdot P(BnB) + I_n BnB \cdot P(nBnB))$$

also, assuming independance of connections between cautious and other cautious:

$$P(B \cap B) = P(B)^2$$

$$P(nB \cap nB) = P(nB)^2$$

$$P(B \cap nB) = 1 - P(B \cap B) - P(nB \cap nB)$$

we thus receive, using:

$$I = [0.05, 0.15, 1]$$

we get that:

$$equation for beta tag$$

gamma: the rate at which a person transitions from being infectious to later states of either hospital or recovery. population size: the number of people in the simulation, pSens: part of the population risk to the epidemic, or in a higher risk group. correlation: the match of the probability of one being cautious upon being risk, and vice versa, or whether risk people are cautious or not. pH: probability of hospitalization upon the end of one's infectious time, value for risk group and not. pD: probability of death upon the end of one's infectious time, value for risk group and not.

$$\frac{dS_{SB,nSB}}{dt} = S_{SB,nSB} \overbrace{((I_{SB} + I_{nSB}) \cdot \beta_{BB}}^{infections\ from\ cautious} + \overbrace{(I_{SnB} + I_{nSnB}) \cdot \beta_{BnB})}^{infections\ from\ non-cautious} \quad [7]$$

$$\frac{dS_{SnB,nSnB}}{dt} = S_{SnB,nSnB}((I_{SB} + I_{nSB}) \cdot \beta_{BnB} - (I_{SnB} + I_{nSnB}) \cdot \beta_{nBnB}) \quad [8]$$

continuing in a similar fashion, we will display values all four states:

$$S, nS, B, nB$$

as a vector:

$$\frac{d\vec{I}}{dt} = + \overbrace{\beta \cdot \frac{d\vec{S}}{dt} \cdot \vec{I}}^{new\ infections} - \overbrace{\gamma \cdot \vec{I}}^{recovery} \quad [9]$$

$$\frac{d\vec{H}}{dt} = + \overbrace{\gamma \cdot P(H_{S,nS}) \cdot \vec{I}}^{new\ hospitalized} - \overbrace{\gamma_H \cdot \vec{H}}^{leaving\ hospital} \quad [10]$$

$$\frac{d\vec{R}}{dt} = + \overbrace{\gamma \cdot (1 - P(H_{S,nS})) \cdot \vec{I}}^{new\ recovered} + \overbrace{\gamma_H \cdot (1 - P(D_{S,nS}) \cdot \vec{H}}^{recovered\ from\ hospital} \quad [11]$$

$$\frac{d\vec{D}}{dt} = + \overbrace{\gamma_H \cdot (P(D_{S,nS}) \cdot \vec{H}}^{deceased\ from\ hospital} \quad [12]$$

stochastic random graph: furthermore, we verified our results with a stochastic agent-based simulation on a random graph.

the epidemic dynamics and state vector are identical, however this is an agent based simulation, and each node, or 'agent' runs it's own schedule. in this way we can apply a structure on the graph, reflecting population dynamics, which aren't random, and have high clustering and locality.

the epidemic spreads between 'people' represented by nodes of a graph which can either be a randomly generated d-regular graph, or a 'structured' graph we generate. the structure roughly reflects the structure of a society, whereby the epidemic spreads by contacts, when contacts can be strong, such as at home, or at work, or random contacts which are weak.

for generating our structured graph we build "households" with a number of parents who work (or not, based on statistics taken from the Israel National Statistical Centre) and a number of children, who go to school. the connections these people have are split into 4 types: random, school, work and home. the strength is as follows: house: 2.4 school/work: 1 random: 0.5 where the connection strength changes the infection rate beta. beta is then adjusted globally, so that the average infection rate remains identical for graph size. the process is as follows: each family is generated, then connected within itself, then children are connected to schools, each with a class of 25 members. workplaces are generated with 10 adults each worker contacts, and finally random connections are made across the population.

the initial infection group are generated randomly, then we compare results of disease propogation in the three models.

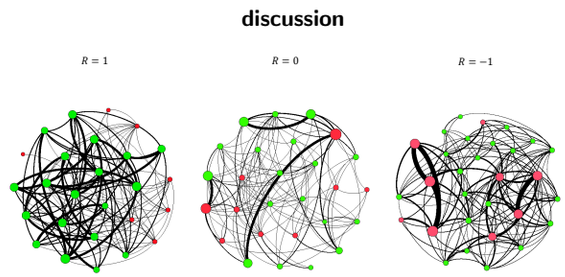

Fig. 5. Schematic map of Graph. Susceptible individuals are scattered around graph

we find the following: when the number of susceptible people in the population is low, it is better to "protect" the non-risk group, as they are the vast majority of the group. by doing this we are lowering the overall spreading ability of the pandemic, to a level where hospitals remain emptier as fewer people of all types are infected. we find that by favouring the susceptible groups, the number of deceased rises, as more are infected before herd immunity is introduced, and thus a larger portion will reach hospitals and higher death tolls ensue. however, this is true until reaching a certain critical point, after which it becomes favorable to protect susceptible groups. in this we find that the driving force behind the epdemic are the non-susceptible groups, and that the people filling the hospitals will often be these same groups. we find the counter-intuitive result that by favouring the non-susceptible groups over the susceptibles, we can often reach a better outcome overall.

if we apply some context, in countries with younger populations, such as Israel, the outcome could be preferable if pratection of the non-susceptible majority is applied. however, in countries with older populations, the number of individuals at high risk is larger, tipping the scales making protection of the susceptibles preferable.

we find that the results from an agent-based solution differs from the non-agent based one in that ********** *not sure what to say.******

An explanation for the finding that negative correlation would be preferable, is that we actually have a case of the following: A significant number of people (the majority, being un-susceptible), are cautious which would be analogous to vaccinated/innoculated, that the eqpidemice threshold is breached - each new infection, fails to infect more people, before healing. this then allows us to explain the surprising effects of negative correlation: the majority is cautious, thus less people manage to get infected, to a point at which the more negative the correlation the faster the epidemic decline. what we also find then is that when positively correlated, the epidemic runs it's full course, spreads through the non-cautious, non-risk individuals and thus fills hospitals with younger people (assuming younger people are healthier and at lower risk). In this case, the full epidemic threshold is reached when a majority has been infected.

$$T_epi = 1 - 1/R$$

this is interesting because two things are happening: 1. for negative correlation: Risk groups spread the epidemic, causing most of those sick to be at higher risk. however the cautious, not at risk majority blocks the epidemic from spreading fully. the key factor here is the initial part infected: if low enough: almost no people will be sick. those sick will be at a high risk of death, but the overall toll can be quite low if caught early on. We assumed that initially 1 percent of the population are infected. 2. for positive correlation: Risk groups naturally choose to protect themselvers, however the not-at risk mojorty spreads the epidemic through society. all risk group-infection will be caused by spread from non-cautios relatives. the overall death toll may be lower, as those sick are healthier. however this may be higher also, as the overall outbreak will spread effectively throughout the breadth of society.

here we used data from covid to show that this can be borderline at around a 30-40 percent risk group rate. below which it is preferable to be negatively correlated, above which it is better to protect the elderly.

analogy to percolation theory eexlantnion of results percolation theory givs us that the critical limit is:

$$p_c = \frac{1}{d-1} \qquad [13]$$

we find that each node must infect more than 1 new nodefor the virus to spread at all. when we compare that to eqn [3] we get that ... we assume the epidemic spreads equally through the population, and randomly initialize a small number of sick. We have the option to control the protection of groups - either isolate risk groups, or isolate groups at low risk, assuming they drive the infections between households and across groups. We are faced with this choice, a forced version of imposing general lockdowns in population, or to isolate groups at risk.

1. Johnson, PW, (2002) The curious history of Faa di Bruno's folrmula. American mathematical monthly 109:217.
2. Roughgarden T, (2005) Selfish routing and the price of anarchy. (MIT Press).
3. Jun S, Si F, Pugatch R and Scott M, (2018) Fundamental Principles in Bacterial Physiology - History, Recent progress, and the Future with Focus on Cell Size Control: A Review. Reports on Progress in Physics 81:056601.
4. Kinshuk B, Kolomeisky AB and Igoshina OA, (2017) Elucidating interplay of speed and accuracy in biological error correction. PNAS 114:5183–5188.
5.
6. Haccou P, Jagers P and Vatutin VA, (2005) Branching Processes: Variation, Growth, and Extinction of Populations (Cambridge University Press).
7. Olofsson P, Sindi S (2014) A Crump-Mode-Jagers branching process model of prion in yeast. J. Appl. Prob 51:453-465.
8. Kimmel M, and Axelrod DA, (2002) Branching Processes in Biology (Springer).
9. Scott M et al., (2010) Interdependence of Cell Growth and Gene Expression: Origins and Consequences. Science 330:1099-1102.
10. Klumpp S, Scott M, Pedersen S and Hwa T (2013) Molecular crowding limits translation and cell growth. PNAS 110:16754–16759.
11. Dai X et al., (2016) Reduction of translating ribosomes enables Escherichia coli to maintain elongation rates during slow growth. Nature microbiology 2:16231.
12. Maitra A and Dill KA (2015) Bacterial growth laws reflect the evolutionary importance of energy efficiency. PNAS 112:406-411.
13. Stokes JM, Davis JH, Mangat CS, Williamson JR and Brown ED (2014) Discovery of a small molecule that inhibits bacterial ribosome biogenesis elife 3:e03574.
14. Reuveni S, Ehrenberg M and Paulsson J (2017) Ribosomes are optimized for autocatalytic production. Nature 547:293-297.
15. Shajani Z and Williamson JR, (2011) Assembly of bacterial ribosomes. Annu. Rev. Biochem. 80:501-526.
16. Mizushima A and Nomura M, (1991) Assembly mapping of 30S ribosomal proteins. Biochimie. 73:739-755.
17. Nierhaus KH, (1991) The assembly of prokaryotic ribosomes. Biochimie. 73:739-755.
18. Li, GW, Burkhardt, D, Gross, C, Weissman, JS, (2014) Quantifying absolute protein synthesis rates reveals principles underlying allocation of cellular resources. Cell 157:624–635.
19. Milo R, Phillips R, (2015) Cell Biology by the Numbers (Garland Science).
20. Presse S et al, (2015) Principles of maximum entropy and maximum caliber in statistical physics. Rev. Mod. Phys. 85:1115.
21. Nomura M, Tissieres A and Lengyel P, (1974) Ribosomes: Monograph 4 (Cold Spring Harbor).
22. Davies JH, et al., (2016) Modular assembly of the bacterial large ribosomal subunit. Cell 167:1610-1622.
23. Bremer H and Dennis PP (2008) Modulation of chemical composition and other parameters of the cell at different exponential growth rates. EcoSal Plus doi: 10.1128/ecosal.5.2.3.