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the realization that large numbers of ‘silent spreaders’

could exist undermines the predictions of epidemiological

models in several ways. First, high numbers of undetected

cases would shrink infection fatality rates. Asymptomatic car-

riers could also transform the trajectory of an outbreak by ac-

celerating transmission. But if they're present in massive

numbers—which many scientists consider highly unlikely—

and they become immune after infection, local epidemics could

be over much sooner than expected if the virus runs out of sus-

ceptible people to infect. Finally, silent spreaders could change

the basic reproductive number R0 as long as their proportion stays constant in the popu-

lation—but tends to be lower than what some researchers now believe to be the case. ‘In fact, it seems

like SARS-CoV-2 is more infectious than MERS and SARS, so [R] is likely higher for SARS-CoV-2 than originally estimated.’

It’s even possible people who never show symptoms could play a role in spreading COVID-19. Asymptomatic trans-

mission would also be in stark contrast to SARS and MERS,

where asymptomatic carriers were relatively uncommon and

were not thought to play a significant role in the outbreaks, notes Panovska-Griffiths. While early reports from China

made little mention of possible asymptomatic individuals,

studies elsewhere through March and April revealed signifi-

cant numbers of individuals who tested positive for the virus but never developed so much as a cough. Around 43 percent of residents surveyed in the northeastern Italian town of Vo in February and March tested positive despite having no symp-

toms, and a recent review concluded that asymptomatic indi-

viduals could account for as many as 45 percent of infections. What’s more, some contact tracing data hint that asymptom-

atic people can transmit the virus to others, although it’s still a mystery how often that occurs.

The realization that large numbers of ‘silent spreaders’ could exist undermines the predictions of epidemiological

models in several ways. First, high numbers of undetected cases would shrink infection fatality rates. Asymptomatic car-

riers could also transform the trajectory of an outbreak by accelerating transmission. But if they are present in massive

numbers—which many scientists consider highly unlikely—and they become immune after infection, local epidemics could

be over much sooner than expected if the virus runs out of sus-

ceptible people to infect. Finally, silent spreaders could change estimates of R0 or Re. However, it’s not the numbers of asymptomatic people capable of transmitting SARS-CoV-2 per se that influences

R0—as long as their proportion stays constant in the popula-

tion, estimates of R0 won’t necessarily change. Rather, what matters is how infectious people are. For instance, if infected people who are asymptomatic have shorter or longer infec-

H1N1 flu pandemic, they turned to data from influenza out-

breaks in the 1960s.

For COVID-19, Del Valle, like many other research-

ers, plugged in parameters documented for other coronavi-

ruses, including MERS-CoV and SARS-CoV, to estimate R0. However, the transmission of SARS-CoV-2 turned out to be markedly different from that of these viruses, notes Jasmina Panovska-Griffiths, a mathematical modeler focusing on infec-

tious diseases at University College London and Oxford Uni-

versity. For instance, while MERS and SARS patients typically

shed coronaviruses while symptomatic, studies suggest that SARS-CoV-2 can be contagious even before patients know they’re sick. Such presymptomatic transmission means that the novel coronavirus’s infectious period is longer than that of SARS-CoV or MERS-CoV, throwing off early R0 estimates in the novel coronavirus’s infectious period is longer than that of SARS-CoV or MERS-CoV, throwing off early R0 estimates in

the infectious-recovered (SIR) modeling techniques.

Researchers across the world have developed countless epidemiological models to project the future course of COVID-19 pandemics, and the effect of different public health policies on the spread of the causative virus, SARS-CoV-2. Most, but not all, models being used today give the two versions of R—R0 and Re—a central role. The basic reproductive number R0 describes the spread of a disease at the beginning of an outbreak, and Re, an “effective” version of the metric, describes spread later on.