Networks of SARS-CoV-2 transmission

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The basic reproduction number, $R_0$, (the number of infections caused by a case in a homogeneously susceptible population), for a particular infection is dependent on the epidemiological triad of the biological characteristics of the pathogen, the environment, and the characteristics of the population (1). Even for diseases with similar transmission characteristics, $R_0$ varies by population owing to differential opportunities for onward transmission according to the contact patterns and the size of the transmission network of an infected individual (1). Although transmission can happen in many settings, some factors facilitate a greater risk of infection because of compounded risks often driven by network dynamics (frequent contacts, close proximity, and prolonged contact) and structural-level determinants (such as poverty, occupation, and household size) (2–4). Understanding drivers of transmission risks and heterogeneity could be used to improve modeling and guide population- and setting-specific mitigation strategies.

In the context of an epidemic, although each contact carries a risk of acquiring an infection, real-world social networks are complex, often exhibiting extreme heterogeneity in the number of contacts, which have large-scale effects on the spread of infection (5). In infectious diseases, the population attributable fraction (PAF) represents the total contribution of a risk that could be averted if that risk were avoided (6). Even for lower-risk exposures, the PAF could increase with higher exposure frequency mediated through greater numbers of contacts (2, 6).

For example, the risk of infection depends on the likelihood of transmission within a particular environment and the frequency at which people visit that setting. At an individual level, settings that are associated with higher-risk factors and visited frequently are likely to pose a higher risk of infection and contribute substantially to cumulative infections than those that may have a higher risk but are visited infrequently. This could mean that a small relative risk of a high-frequency exposure can drive the PAF, suggesting that public health interventions could prioritize resources to eliminate a small risk among many.

However, in reality, risk factors concentrate among the relatively few who have disproportionately higher exposure and onward transmission risks (2, 7). This individual heterogeneity is evident in data, which consistently indicate higher risks of infection due to higher frequency of exposure and multiple contacts (see the figure). In many countries, those working in low-paid and public-facing jobs had the highest risk of being infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) (4). Long-term–care facilities such as nursing homes, homeless shelters, and prisons, as well as workplaces such as meat-packing plants, have been associated with large-scale outbreaks of COVID-19, which were then linked to sustained widespread community transmission (2, 8). These settings often represent environments where risks for infection are compounded and multiple transmission networks intersect (7). There is also a clear intersection of COVID-19 risk and socioeconomic inequities, given the network effects of occupation, crowded housing, job insecurity, and poverty (2, 4).

The disproportionate risks associated with network dynamics have also led to differential disease burden (4, 9). According to an analysis from Scotland, patients living in areas with the greatest socioeconomic deprivation had a higher frequency of intensive-care admission and higher COVID-19–related mortality (10). Health care units in the most deprived areas also operated over capacity for a more prolonged period (10). In a US study, those working in food and agriculture, transportation or logistics, manufacturing, health services, and retail had significantly increased excess mortality related to COVID-19 (9). Moreover, differential living and working conditions often manifest as racial disparities because of structural racism. An analysis by the Office for National Statistics highlights the finding that occupations in the UK with higher COVID-19–related death rates include health and social care workers, security guards, drivers, construction workers, cleaners, and sales and retail assistants, which are occupations that also feature higher proportions of minority ethnic groups (4). For most occupational categories, the risk ratios comparing mortality during the pandemic with that during nonpandemic time were higher in nonwhite ethnic groups (4, 9).

In addition to heterogeneity in risk of exposure and disease burden, there are also heterogeneities in risk of onward transmission. Per-contact, direct onward transmission risks are driven by multiple factors, including closeness of social interactions, symptom status, the severity of illness, environment, and time of exposure (2, 6). For example, the average per-contact risk is lowest for community exposures, intermediate for social and extended-family contacts, and highest in the household (11). Transmission risk is lower when the index case is asymptomatic, increasing with symptom severity (12). Indirect onward transmission risks or the total number of downstream infections that stem from an individual over multiple chains of transmission represent important contributions to the overall PAF driven by the size of the transmission networks associated with living and working conditions (4, 7, 13).

Although some high-frequency contacts are driven by social gatherings, which are modifiable with education and enforcement, most high-risk exposures represent nonmodifiable risks due to living and working conditions (2, 3, 7). Therefore, risk factors that are nonmodifiable in the short term are likely to represent a much larger PAF than those modifiable by individual choices about social contact. Specifically, the onward transmission risks from someone who can work from home and has enough space for self-isolation, even if they are infected, may be minimal; but the PAF will be higher for someone with a large network associated with working and living conditions (see the figure).

There is now international consensus that those living in the most economically deprived neighborhoods and largest households have an increased risk of infection and disease burden (3, 4). In addition, inequities further concentrate risk through connections between networks. In Toronto, long-term–care staff diagnosed with COVID-19 were disproportionately more likely to reside in neighborhoods with the highest infection rates, which are also the most economically deprived and ethnically concentrated (14). In a COVID-19 outbreak investigation among large industries in Ontario, one-third of cases linked to workplace outbreaks spilled over to households, further increasing the burden of...
Downstream infection risks vary according to network patterns

Case A depicts a person with a small network, who can work from home and self-isolate if needed. Case B represents a person who works in a public-facing job or in an unsafe workplace and lives in a multigenerational or large household. Overall risk of exposure and onward transmission risk differ substantially between these two individuals, representing a disproportionately high transmission chain in case B. Intervention strategies should focus on breaking chains of downstream transmission.

the same characteristics, are mostly used to model COVID-19 cases and the impact of interventions. However, they infrequently integrate the effects of differential population mixing, socioeconomic factors, and networks across compartmental effects. It is now clear that individual heterogeneity has large-scale effects on disparities seen in the risk of infection and disease burden, which is confirmed in network-based disease modeling (1, 6, 7, 11). Public health policies implemented based on the assumption of equal risk of acquisition and transmission across all socioeconomic groups, ages, and occupations left certain communities exposed to a higher risk of infection, resulting in differential burdens of disease (1–3, 7). Leveraging network heterogeneity in infectious disease models may better demonstrate these differential risks.
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Science 373 (6551), 162-163.
DOI: 10.1126/science.abg0842