In the paradigmatic susceptible-infectious-recovered (SIR) model of infectious disease in a fully mixed population, herd immunity is reached when the fraction \( \pi_v \) of the population that is immune to the disease is larger than

\[
\pi_v^c = 1 - \frac{1}{R_0},
\]

where \( R_0 \) denotes the basic reproduction number. Beyond this simplified model, real-world populations exhibit inhomogeneous mixing patterns that can lead to nontrivial epidemic outcomes. One of the inhomogeneities that would be particularly relevant to vaccine-induced herd immunity is the correlation between the vaccination status of interacting individuals. When this correlation exists, the vaccinated and unvaccinated individuals have different compositions of vaccinated and unvaccinated neighbors, leading to a mixing pattern characterized by homophily.

We investigate the effect of such assortative mixing with respect to vaccination status on the herd immunity threshold and the expected epidemic size. We formulate a random network theory of epidemic spreading under homophily with respect to the adoption of an immunity-inducing vaccine. Within the population, a fraction \( \pi_v \) of the population adopts the vaccine, while the remaining fraction \( \pi_u = 1 - \pi_v \) is not vaccinated. Let us denote the conditional probability that a random neighbor of an individual is vaccinated given that the individual is vaccinated by \( \pi_{vv} \) and, similarly, the conditional probability that a random neighbor of an unvaccinated individual is not vaccinated by \( \pi_{uu} \). We quantify the strength of vaccination homophily by the Coleman homophily index

\[
h = \frac{\pi_{vv} - \pi_v}{1 - \pi_v} = \frac{\pi_{uu} - \pi_u}{1 - \pi_u}.
\]

To identify the vaccination threshold \( \pi_v^c \) above which the disease cannot spread, we make use of the next-generation matrix (NGM) method, which can intuitively be interpreted as a description of the local structure of the network by a multi-type branching process. When the vaccine is perfect, the critical vaccine coverage needed for herd immunity is given by

\[
\pi_v^c = 1 - \frac{1}{1 - h \left( 1 - \frac{1}{R_0} \right)},
\]

which reduces to the well-known threshold of Eq. (1) for homogeneous mixing with \( h = 0 \).

Equation (3) indicates that if the homophily strength \( h \) increases, so does the vaccine coverage \( \pi_v^c \) required for herd immunity (see Fig. 1). In other words, the presence of homophily makes herd immunity harder to reach. Notably, the threshold occurs at \( \pi_v^c = 1 \) for \( h \geq 1/R_0 \), implying that above this critical strength of homophily, one cannot attain herd immunity at all unless the entire population is vaccinated.

When the vaccine coverage is below the threshold, an outbreak can result in an epidemic that infects a substantial fraction of the population. The size of such an epidemic can be computed by the percolation theory. We find that the epidemic size monotonically increases as a function of homophily strength for a perfect vaccine, while it is maximized at a nontrivial level of homophily when the vaccine efficacy is limited. This is due to the competition between the herd immunity effect by homogeneous mixing and the epidemic containment by segregation.

In conclusion, our findings suggest that herd immunity is more difficult, if not impossible, to achieve in the presence of vaccination homophily. It also implies that the well-known formula of Eq. (1) underestimates the vaccination threshold by not taking homophily into account. We also show that the behavior of epidemic size as a function of homophily varies depending on the vaccine efficacy. Here, we focused on homophily by vaccination status; however, our framework is general enough to account for homophily by adherence to other epidemic interventions that reduce the susceptibility or infectiousness of individuals.