

## Multimedia Appendix 2: Supplementary Methods

### Epidemic wave dynamics attributable to urban community structure

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**Detecting multiwave epidemics.** To automatically identify epidemic curves exhibiting multiple waves, we define a new two-peak metric ( $TP$ ), given by

$$TP(S) = \max_{1 \leq i < j \leq n} \left( \sqrt{(s_i - v_{i,j}) \times (s_j - v_{i,j})} \right) \quad (S1)$$

where  $S$  is the epidemic time series  $(s_1, s_2, \dots, s_n)$  and  $v_{i,j} = \min_{i \leq k \leq j} (s_k)$  is the minimum value occurring in the time series between  $s_i$  and  $s_j$ .  $TP$  indicates the depth of the deepest valley in the epidemic time series (specifically, the geometric mean of the heights of on each side of the deepest valley).

**Percolation-based approximations of  $R_0$ , epidemic size and community bridging.** Given a semi-random network with degree distribution  $\{p_k\}$  and a pathogen with per edge transmission probability  $T$ , the reproduction number is given by

$$R_0 = T \left( \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle} \right) \quad (S2)$$

where  $\langle k \rangle$  is the average degree and  $\langle k^2 \rangle$  is the average squared degree [29]. To calculate global  $R_0$ , we consider the total degree of each node in the network; for community-specific  $R_0$ , we restrict to within-community degree. The expected size of an epidemic (proportion infected) is given by

$$S = 1 - \sum_{k=1}^{\infty} p_k (1 + (u-1)T)^k \quad (S3)$$

where  $u = \frac{\sum_{k=1}^{\infty} k p_k (1 + (u-1)T)^{k-1}}{\sum_{k=1}^{\infty} k p_k}$  is a self-referencing equation solvable by root finding methods [30]. For

specific communities, we simply restrict to within-community degrees (e.g., denoted  $S_A$  and  $u_A$  for community  $A$ ).

The probability that a node  $a$  in community  $A$  sparks an epidemic, once infected, is equal to one minus the probability that none of  $a$ 's contacts in  $B$  spark an epidemic (which occurs if transmission does not occur from  $a$  or if it does occur but fails to seed an epidemic in  $B$ ), as given by

$$\varphi_a = 1 - \prod_{b \in \beta_a} \left( (1-T) + T(1 - \varepsilon_b) \right) = 1 - \prod_{b \in \beta_a} (1 - T \varepsilon_b) \quad (S4)$$

where  $\beta_a$  denotes the set of  $a$ 's contacts in  $B$ ,  $\varepsilon_b = 1 - (1 - T + T u_B)^{\kappa_b}$  is the probability that  $b$  sparks an epidemic in  $B$  [29], and  $\kappa_b$  denotes the within-community degree of  $b$ . Early in an epidemic, the probability that a random transmission event will reach a given node is proportional to its degree [30]. Thus, the probability that an early transmission event within  $A$  will immediately seed an epidemic in  $B$  is given by

$$\phi_{A \rightarrow B} = \sum_{a \in A} \left( \frac{\kappa_a}{\sum_{a \in A} \kappa_a} \right) \phi_a. \quad (\text{S5})$$

For an epidemic starting in  $A$  to cause a distinct secondary wave in  $B$ , it must eventually spark an epidemic in  $B$ , but not too early that it appears synchronous. This is approximately given by

$$\Gamma_{A \rightarrow B}(\gamma) = (1 - \phi_{A \rightarrow B})^\gamma - \prod_{a \in A} (1 - v_a \phi_a) \quad (\text{S6})$$

where  $v_a = 1 - (1 - T + Tu_A)^{\kappa_a}$  is the probability that  $a$  becomes infected during the epidemic within  $A$  [29]. The first term is the probability that the epidemic does not spread to community  $B$  before  $\gamma$  within-community  $A$  transmission events, and the second term is the probability that it never spreads to  $B$ . To calculate this probability for epidemics spreading from community III to I and from community I to II, we assumed  $\gamma = 50$  and  $\gamma = 200$ , respectively, based on the range of lags observed between subsequent waves in our stochastic simulations (see Multimedia Appendix 1, Figure S1). Epidemics typically appeared synchronous if the source community seeded an epidemic in the recipient community within fewer transmission events.