exceptions. The average degree of the nodes in a specific shell follows roughly a power law with k_S (Fig. 13). The deviations from this average behavior emphasize the importance of spreaders within the core of the network having high values of k_S and potentially smaller degrees, than those with high k and low k_S values.

The complex organization of the nodes in the k-shells is highlighted when we randomly rewire the links in the networks, yet preserving the nodes degree. This rewiring 'restores' all the hubs to the innermost k-shell of the system and imposes a strict hierarchy of nodes in terms of both k and k_S. The bottom row of plots in Fig. 12 shows the scatter-plots of degree k as a function of k-shell index k_S for every node in the network. In all cases, a monotonic relation of k vs k_S is followed in the 'rewired' networks (red symbols), where now all the hubs appear in the highest k-shell) as opposed to the weak correlation between k and k_S in the original networks (shown in black).

VI.   REWIRING HIGHLIGHTS THE IMPORTANCE OF k-SHELL

In Figs. 1a and 1b of the main text we show that the extent of infection can be remarkably different, although we start from two origins with similar degree. The importance of the structure in the dynamics of spreading can be highlighted if we randomly rewire the network. During this process the original degrees of all nodes are preserved, but random neighbors are chosen for each node, destroying thus any correlations and any patterns in the local connectivity. We denote by $P(M|i)$ the probability that a percentage M of the total population will be infected if a disease originates on node i. In Figs. 1a,b of the main text and in Fig.14a we show that two nodes #1 and #2 with similar degree may yield markedly different distributions $P(M|1)$ and $P(M|2)$. After rewiring, these distributions become practically indistinguishable (see Fig. 14b).

VII.   VIRUS PERSISTENCE IN SIS

Many infectious diseases, including most sexually transmitted infections, do not confer immunity after infection, so that they cannot be described via the SIR model. These cases are better simulated through the SIS epidemic model [18]. The dynamics of SIS epidemics is different, since the number of infected nodes eventually reaches a dynamic equilibrium