

Dismantling the Herd Immunity Threshold Orthodoxy: Heterogeneous Transmission Networks, Superspreading Dynamics, and the Recalibration of Population-Level Immunological Benchmarks in Post-Pandemic Epidemiology

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Abstract. The classical herd immunity threshold (HIT) framework, rooted in homogeneous mixing assumptions derived from compartmental SIR models, has historically guided vaccination coverage targets across national immunization programs. Emerging epidemiological evidence from the post-COVID-19 era challenges the universal applicability of these deterministic benchmarks. This study synthesizes multi-country seroprevalence datasets, network-based transmission models, and individual-level contact matrices to interrogate whether fixed HIT values remain defensible under conditions of pronounced population heterogeneity. Employing a stratified meta-analytic framework across 27 national cohorts (2021–2024), we demonstrate that superspreading events and assortative social mixing systematically lower effective reproductive numbers below conventionally predicted thresholds. Our findings advocate for context-sensitive, dynamic immunological benchmarks that incorporate spatial clustering, age-structured susceptibility gradients, and waning immunity kinetics, with direct implications for adaptive vaccine policy design.

Keywords: herd immunity threshold, heterogeneous transmission networks, superspreading dynamics, seroprevalence epidemiology, compartmental SIR models, population immunological benchmarks, waning immunity kinetics, vaccine policy optimization, age-structured susceptibility

Introduction

Few constructs in modern public health have achieved the institutional authority of the herd immunity threshold (HIT)—the theoretical proportion of an immunized population sufficient to interrupt endemic transmission of a pathogen. Since its formalization through

the work of Kermack and McKendrick in 1927 and its subsequent operationalization in mass vaccination policy throughout the latter twentieth century, the HIT has served as the foundational numerical target around which national immunization programs are architected. Expressed canonically as $HIT = 1 - (1/R_0)$, where R_0 denotes the basic reproduction number under conditions of homogeneous susceptibility and random mixing, the threshold has been applied globally as though it were a biologically fixed property of the pathogen alone. Yet this operationalization obscures a fundamental epidemiological reality: human populations are neither homogeneous in susceptibility nor random in their patterns of contact. The post-COVID-19 epidemiological landscape, spanning 2021 through the present, has generated an unprecedented volume of granular, population-level immunological data—multi-wave seroprevalence surveys, genomic transmission reconstructions, and real-world vaccine effectiveness studies—that collectively destabilize deterministic HIT assumptions. In contexts marked by pronounced sociodemographic stratification, occupational exposure clustering, and vaccine access inequity, empirically observed transmission interruption has occurred at coverage levels substantially divergent from R_0 -derived predictions. Simultaneously, the phenomenon of superspreading—wherein a minority of infectious individuals account for a disproportionate fraction of onward transmission events, as quantified through the overdispersion parameter k —has been formally incorporated into transmission models, yet its implications for HIT recalibration remain underexplored in programmatic public health guidance. Recent scholarship in network epidemiology has demonstrated that when transmission occurs preferentially along high-degree nodes within heterogeneous contact networks, effective population immunity can be achieved at substantially lower absolute coverage than classical threshold theory predicts. Conversely, in spatially fragmented or vaccine-hesitant subpopulations, local susceptible pockets can sustain resurgent transmission even when aggregate national coverage nominally exceeds established thresholds. These dual dynamics—threshold depression in high-connectivity clusters and threshold elevation in fragmented communities—expose the inadequacy of any single, static immunological benchmark applied uniformly across epidemiologically diverse populations. This paper is structured as follows. Section 2 reviews the theoretical genealogy and mathematical architecture of classical HIT models, tracing assumptions that have persisted unchallenged in policy frameworks. Section 3 details our multi-country stratified meta-analytic methodology, including data harmonization protocols across 27 national seroprevalence cohorts. Section 4 presents empirical findings on the divergence between canonical and observed immunological thresholds, stratified by demographic, geographic, and behavioral covariates. Section 5 develops a recalibrated, context-adaptive HIT framework integrating superspreading coefficients, age-structured waning immunity gradients, and spatial heterogeneity indices. Section 6 discusses operational implications for adaptive vaccination campaign design and surveillance architecture. Section 7 concludes with recommendations for international health governance bodies.

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