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Assessing the Role of Typhoid Fever Carriers in Disease Transmission

Taliikwa Nicholas Ceaser

Department of Pharmacognosy Kampala International University Uganda
Email:ceaser.taliikwa@studwc.kiu.ac.ug

ABSTRACT

Typhoid fever (enteric fever) remains a major public health problem in many low- and middle-income countries, sustained by fecal–oral transmission of *Salmonella enterica* serovar Typhi (*S.* Typhi). While acute symptomatic cases are readily recognized and treated, a smaller but epidemiologically important fraction of infected persons become carriers who intermittently or continuously shed *S.* Typhi for months to years, often without symptoms. Chronic carriage (commonly defined as shedding ≥12 months) can maintain *S.* Typhi in human populations, seed outbreaks, and frustrate elimination efforts, particularly where water, sanitation, and hygiene (WASH) systems are weak, and food handling is informal. This review synthesizes current understanding of (i) carrier states and their biological basis (especially gallbladder persistence and biofilm formation), (ii) the relative contribution of carriers to transmission in endemic vs non-endemic settings, (iii) practical approaches for detection and confirmation of carriers, and (iv) interventions to reduce transmission risk ranging from vaccination and WASH improvements to targeted screening and treatment of high-risk groups. We highlight key evidence gaps, including the lack of scalable diagnostics for carriers and limited field data quantifying carrier-attributable transmission under contemporary antimicrobial resistance (AMR) pressures.

Keywords: typhoid fever; *Salmonella Typhi*; chronic carrier; convalescent carrier; WASH; typhoid conjugate vaccine.

INTRODUCTION

Typhoid fever remains a major public health challenge in many low- and middle-income countries, especially where access to safe water, improved sanitation, and consistent food hygiene practices is limited. The disease is caused by *Salmonella enterica* serovar Typhi (*S. Typhi*), a bacterium that is uniquely adapted to humans. This human restriction is epidemiologically important: unlike many other enteric pathogens that can persist in animal reservoirs, typhoid transmission is sustained through human infection and human shedding of the organism into the environment [1]. Transmission occurs primarily through ingestion of food or water contaminated with feces (and occasionally urine) from infected persons. In endemic settings, widespread environmental contamination such as unsafe water sources, open defecation, poorly maintained latrines, inadequate wastewater disposal, and contaminated street-vended foods can make typhoid appear "community-wide" and diffuse. However, even in such settings, the ultimate source remains human: infected individuals who shed *S. Typhi* during acute illness and, critically, individuals who continue to shed the pathogen after symptoms resolve [2].

This brings into focus the role of the typhoid carrier state. After apparent clinical recovery, some individuals may continue excreting *S. Typhi* for weeks to months (convalescent shedding), and a smaller but highly consequential fraction may become chronic carriers shedding bacteria for a year or longer. Chronic carriage is often associated with bacterial persistence in the gallbladder, sometimes linked to gallstones, biofilm formation, and ongoing intermittent shedding. Because carriers frequently lack symptoms, they may not seek care, may not be tested, and

may unknowingly contaminate food and water. The carrier state therefore represents a "hidden reservoir" capable of sustaining transmission during inter-epidemic periods and seeding new outbreaks even when overt cases appear to decline [3]. From a public health perspective, carriers are particularly important in contexts where food handling practices create opportunities for amplification. A single asymptomatic carrier involved in food preparation at home, in markets, in restaurants, or in institutional settings (schools, prisons, health facilities) can expose large numbers of people. Historically, the case of "Typhoid Mary" illustrated how silent carriage can fuel repeated outbreaks. Yet modern typhoid control must move beyond anecdote toward rigorous quantification of carrier prevalence, characterization of risk factors for carriage, evaluation of practical detection approaches, and assessment of how carriers contribute to transmission compared with other pathways [4].

Current typhoid control strategies in endemic regions typically emphasize treatment of acute cases, improvements in water, sanitation, and hygiene (WASH), and, increasingly, use of typhoid conjugate vaccines (TCVs). While these measures are essential, persistent transmission in many settings suggests that certain drivers remain under-addressed. If carriers contribute meaningfully to ongoing transmission, then interventions focused only on symptomatic cases may have limited long-term impact. In addition, antimicrobial resistance (AMR) in *S. Typhi*, including multidrug-resistant and extensively drug-resistant strains in some regions, complicates treatment and could prolong shedding or increase the risk of incomplete clearance, making the carrier issue even more urgent [5]. Therefore, understanding the carrier state is not an academic exercise; it is central to realistic typhoid elimination planning. Identifying who is most at risk of becoming a carrier, how long they shed, the microbial and host factors that enable persistence, and the feasible tools for detection and management could close a critical gap in typhoid control programs, especially where resources are constrained and routine surveillance is weak [6].

Despite ongoing efforts to control typhoid fever through improved WASH interventions, case management, and vaccine introduction, typhoid transmission persists in many endemic communities. A key reason is that control efforts often focus on clinically apparent disease while overlooking asymptomatic and chronic carriers who continue to shed *S. Typhi* into the environment. Because carriers typically do not present with symptoms, they remain unidentified, untreated, and unrestricted in activities that may promote transmission, particularly food preparation and handling [7]. In many endemic settings, routine laboratory capacity to confirm *S. Typhi* infection is limited, and diagnostic testing is commonly restricted to symptomatic patients. Even when carriers are suspected, detection is challenging: shedding may be intermittent, stool culture sensitivity is imperfect, repeated sampling is logistically difficult, and advanced methods (e.g., molecular diagnostics) may be unavailable or unaffordable. As a result, there is insufficient local evidence on (i) the prevalence of typhoid carriage, (ii) the demographic and clinical factors associated with carriage, (iii) the magnitude of carriage-related transmission compared with other routes, and (iv) practical strategies to identify and mitigate carriage in real-world program settings.

This evidence gap creates a programmatic blind spot. Without clear data on carrier contribution, public health programs may underestimate the role of the hidden reservoir, misallocate resources, and fail to interrupt transmission during inter-epidemic periods. Consequently, communities may experience recurrent outbreaks, continued endemicity, and preventable morbidity, including severe complications and deaths [8]. The persistence of typhoid despite interventions therefore raises a critical question: to what extent do carriers sustain typhoid transmission, and what feasible approaches can be used to detect and manage carriers to strengthen typhoid control?

This study specifically aims to assess the role of *Salmonella enterica* serovar Typhi (S. Typhi) carriers as a hidden reservoir that sustains typhoid fever transmission in endemic settings, and to generate evidence that informs feasible strategies for detecting and mitigating typhoid carriage where resources are constrained. The study will (i) estimate the prevalence of S. Typhi carriage within the selected study population and setting, establishing the magnitude of asymptomatic and chronic shedding that may be missed by routine surveillance focused on symptomatic cases. It will (ii) identify key demographic, clinical, and environmental risk factors associated with carriage—such as age, sex, prior history of typhoid, gallbladder disease/gallstones, occupation (especially food handling), and exposures linked to water, sanitation, and hygiene (WASH). Further, the study will (iii) evaluate the likely contribution of carriers to ongoing community transmission by examining pathways through which shedding may translate into new infections, including household contact patterns, food preparation behaviors, and potential clustering of cases around high-risk individuals or workplaces. Finally, it will (iv) appraise detection and management options that are operationally feasible in low-resource endemic contexts, considering the practicality and yield of repeated stool culture and the potential role of rapid serologic or molecular adjuncts where available, alongside realistic clinical–laboratory screening algorithms.

Overall, the study is significant because it addresses a neglected driver of typhoid persistence: silent human shedding. Evidence from this work can strengthen typhoid control policies by supporting targeted screening in high-risk

groups, improving outbreak prevention and response, informing diagnostic prioritization, and enabling smarter allocation of limited resources across WASH, vaccination, surveillance, and carrier-focused interventions for more durable reductions in incidence.

Carrier states: definitions and epidemiologic importance

Carrier states are operationally defined in public health practice to guide surveillance and control actions. A convalescent (temporary) carrier is typically someone who continues shedding *Salmonella Typhi* for ≥3 months after acute illness onset, whereas a chronic carrier sheds for >12 months after onset or is identified through repeated positive cultures in a person with remote or uncertain illness history. These thresholds are not semantic: duration and confirmation criteria shape how we judge transmission risk, the feasibility of follow-up, and the proportionality of interventions such as exclusion from high-risk work (e.g., food handling) or intensified contact tracing [9]. Although only an estimated ~2–5% of infected individuals become chronic carriers, their epidemiologic significance can be disproportionate because shedding may be intermittent yet prolonged for years, and carriers may occupy roles in food preparation, household caregiving, or institutional support that create large exposure networks. Biologically, chronic carriage is often explained by gallbladder persistence, where *S. Typhi* forms biofilms on gallstones or biliary epithelium; biofilms blunt host clearance and reduce antibiotic susceptibility, enabling long-term survival with periodic seeding of the intestine via bile flow [10]. This mechanism aligns with consistent risk patterns: biliary pathology (especially gallstones), along with older age and female sex, likely partly reflecting gallstone prevalence. Importantly, chronic carriage is not exclusively gallbladder-based; extra-biliary reservoirs have been described, meaning gallbladder-focused screening can miss some carriers. Finally, growing antimicrobial resistance (AMR) raises the stakes: carriers may sustain transmission of resistant lineages, while historical eradication regimens may be less effective or harder to deploy safely today [11].

How carriers drive transmission: pathways and settings

Carriers drive *S. Typhi* transmission through three main pathways: direct fecal contamination of household environments and shared sanitation, contamination during food handling (informal catering, street vending, and routine household meal preparation), and water contamination where sewage disposal is inadequate [12]. These routes broadly mirror transmission from acute symptomatic cases, but differ in timing and detectability because carriers often shed organisms at low levels, intermittently, and without symptoms that prompt care-seeking, allowing silent spread. In endemic settings, the dominant explanation typically centers on unsafe water and sanitation, yet environmental contamination is ultimately "fed" by human shedders, including both acute cases and carriers [13]. Carriers can therefore act as a maintenance reservoir by sustaining transmission during low-incidence periods (inter-epidemic persistence), seeding household clusters, and introducing *S. Typhi* into food chains via asymptomatic food handlers. Because routine clinical surveillance disproportionately captures symptomatic infections and diagnostic confirmation may be limited, the relative contribution of carriers is often underestimated. In non-endemic settings, carriers can function as outbreak catalysts: with minimal background transmission, a single carrier linked to high-reach foodborne networks may trigger multiple cases. This risk underpins public health guidance that prioritizes follow-up, clearance testing, and risk-based restrictions, especially for food handlers, as illustrated historically by events such as the Mary Mallon case, though modern control depends on systematic detection and management rather than isolation alone [14].

Detecting typhoid carriers: what works, what doesn't

Detecting *Salmonella* Typhi carriers remains a public health priority, but no single test is both definitive and easily scalable. Culture-based confirmation is still treated as the reference standard because repeated positive stool and/or urine cultures over time provide strong evidence of carriage and, crucially, yield isolates for strain typing and antimicrobial resistance (AMR) testing information needed for outbreak investigation and treatment decisions [15]. However, culture performs imperfectly in real-world carrier detection: intermittent bacterial shedding can produce false negatives, repeated sampling is logistically demanding, and many settings lack the laboratory capacity to sustain serial cultures; isolate recovery also declines where culture-independent diagnostics are increasingly used. Molecular approaches such as multiplex PCR can deliver faster detection than culture in some contexts, yet carrier identification raises persistent deployment questions, including cost, infrastructure requirements, reduced sensitivity when bacterial loads are low, and the continued need for culture confirmation to support AMR surveillance [16]. Serology and antigen markers mainly indicate past exposure or vaccination and are therefore not definitive for identifying carriers, though they may support screening in research or targeted programs. Pragmatically, screening often focuses on higher-yield groups food handlers linked to outbreaks, household contacts in low-incidence settings, and individuals with biliary disease (e.g., gallstones) but large-scale implementation in high-burden regions is constrained by diagnostics, follow-up, and system capacity [17].

Public health and clinical interventions to reduce carrier-driven transmission

Public health and clinical strategies to curb carrier-driven typhoid transmission must address both symptomatic "acute" shedders and asymptomatic carriers, since each contributes fecal contamination of water, food, and household environments. The most scalable, carrier-agnostic approach in endemic settings is strengthening WASH and food safety: reliable safe water, improved sanitation, and consistent hand hygiene reduce exposure regardless of whether the source is identified. Typhoid conjugate vaccines (TCVs) add a powerful indirect effect by preventing infection and disease, thereby shrinking the pool of individuals who could later become chronic carriers; even if TCVs do not clear established carriage, lowering incidence reduces new carrier formation over time (given an estimated ~2–5% conversion). In outbreak or non-endemic contexts, control often relies on robust case management plus follow-up cultures to document clearance, with temporary restrictions for high-risk occupations (especially food handling) until microbiological proof of non-shedding [18]. For confirmed chronic carriage, prolonged antibiotics have been used, but efficacy is inconsistent due to biofilm-associated gallbladder colonization and antimicrobial resistance; selected patients with gallbladder disease may benefit from biliary intervention (including cholecystectomy), though feasibility and equity constraints limit population-level use in low-resource settings. Finally, carrier-focused control must avoid stigma and coercion: programs should emphasize informed consent, counseling, and supportive risk-reduction plans rather than punitive measures.

What we still don't know (and why it matters)

Despite decades of typhoid control efforts, several critical unknowns still limit how effectively we can detect and interrupt chronic carriage, and these gaps directly affect transmission reduction strategies in endemic settings. First, the attributable fraction remains unclear: in today's communities, what proportion of sustained transmission is actually driven by chronic carriers versus repeated acute infections? Without robust field data to parameterize transmission models, programs risk misallocating effort. Second, scalable diagnostics are still missing. Many carriers shed intermittently at low levels, so inexpensive, field-ready tests that maintain high sensitivity and specificity under real-world conditions are a major unmet need [19]. Third, in the AMR era, the most effective antimicrobial regimens for eradication of chronic carriage are uncertain, likely varying by local resistance profiles and patient factors, making "one-size-fits-all" guidance unreliable. Fourth, growing evidence suggests non-gallbladder reservoirs may contribute to persistence, implying that detection and treatment approaches designed around a gallbladder-only paradigm could miss important carrier states. Finally, program design evidence is thin: we still lack strong cost-effectiveness data for targeted screening of high-risk groups (e.g., food handlers or patients with biliary disease) when integrated with TCV rollout and WASH improvements in high-burden contexts.

CONCLUSION

Closing the remaining evidence gaps around chronic typhoid carriage is essential for making transmission-reduction strategies in endemic settings more precise, cost-effective, and resilient in the AMR era. Current control approaches risk "shooting in the dark" because the true contribution of chronic carriers to sustained community transmission relative to recurrent acute infections remains poorly quantified. Addressing this requires contemporary, field-based studies that link carriage status, shedding dynamics, and onward transmission to properly parameterize models and guide resource allocation. In parallel, progress hinges on scalable diagnostics able to detect intermittent, low-level shedding with high real-world sensitivity and specificity, enabling feasible screening outside specialist laboratories. Therapeutically, eradication regimens must be re-evaluated against local resistance patterns and patient heterogeneity to avoid ineffective, generalized guidance. Recognition of potential extra-gallbladder reservoirs further underscores the need to broaden biological assumptions underlying surveillance and treatment. Finally, robust implementation and cost-effectiveness evidence is needed to define when and how targeted screening of high-risk groups should be integrated with TCV scale-up and WASH interventions, maximizing impact in high-burden communities.

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