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The syndemics of childhood diarrhoea: A biosocial perspective on efforts to combat global inequities in diarrhoea-related morbidity and mortality

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Abstract

Diarrhoea remains the second leading cause of death in children under 5 years. Moreover, morbidity as a result of diarrhoea is high particularly in marginalized communities. Frequent bouts of diarrhoea have deleterious and irreversible effects on physical and cognitive development. Children are especially vulnerable given their inability to mount an active immune response to pathogen exposure. Biological limitations are exacerbated by the long term effects of poverty, including reduced nutrition, poor hygiene, and deprived home environments. Drawing from available literature, this paper uses syndemic theory to explore the role of adverse biosocial interactions in increasing the total disease burden of enteric infections in low-resources populations and assess the limitations of recent global calls to action. The syndemic perspective describes situations in which adverse social conditions, including inequality, poverty, and other forms of political and economic oppression, play a critical role in facilitating disease-disease interactions. Given the complex micro and macro nature of childhood diarrhoea including interactions between pathogens, disease conditions and social environments, the syndemic perspective offers a way forward. While rarely the focus of health interventions, technologically advanced biomedical strategies are likely to be more effective if coupled with interventions that address the social conditions of disparity.

Keywords

biosocial model; childhood diarrhoea; syndemic

Introduction

Diarrhoea is the second leading cause of death in children under the age of 5 years, killing approximately 710,000 children in 2011 (Fischer Walker et al., 2013; WHO, 2013a). Death from diarrhoea usually results from severe dehydration and fluid loss. Most diarrhoea related deaths in children occur in marginalized communities within developing countries (Fischer Walker et al., 2013; Keusch et al., 2006). In these settings, children under 3 years experience on average three episodes of moderate-to-severe diarrhoea every year (WHO, 2013b).

Diarrhoea is usually a symptom of enteric bacteria, viruses, and/or parasitic infection of the intestinal tract. Infection is spread through contaminated food or drinking-water, or from person-to-person. Worldwide, one in six people (1.1 billion individuals) have no source of

safe drinking water, and four in ten (2.6 billion) lack improved sanitation – numbers that are projected to double by 2025 (Mara, 2003; WHO, 2013a). Furthermore, 1.1 billion people practice open defecation (WHO/UNICEF, 2012b). Collectively, these figures suggest inequalities in exposure to the underlying biosocial causes of diarrhoea.

The use of simple oral glucose-electrolyte rehydration solution (ORS) has dramatically reduced acute diarrhoea-related mortality from 4.6 million annual deaths globally during the mid-1980s to the current estimate of 1.6–2.1 million (Keusch et al., 2006; Kosek, Bern, & Guerrant, 2003; WHO, 2013a). However, diarrhoea-related morbidity continues to have significant long-term implications, particularly for young children (Fischer Walker, Perin, Aryee, Boschi-Pinto, & Black, 2012; Keusch et al., 2006). Infants and children are more adversely affected because they are more vulnerable to both social and biological factors that facilitate infection, such as the long term and entwined effects of poverty, malnutrition, and underdeveloped immune systems.

Children malnourished as a result of diarrhoeal disease tend to ‘catch up’ if given a chance (Schorling & Guerrant, 1990). However, for children who are not afforded such assistance, frequent bouts of diarrhoea have deleterious and irreversible negative effects on development (Petri et al., 2008). Diarrhoea inhibits normal physical advancement by directly impairing intestinal absorption of nutrients necessary for optimal growth and development of the body, brain, and neuronal synapses that determine future potentials (Walker et al., 2011). A pooled analysis of nine studies showed that the odds of stunting by age 2 years increased by 1.13 (95% CI 1.07–1.19) for every five episodes of diarrhoea (Checkley et al., 2008). Additional research suggests that by age 7 years, children suffering from multiple bouts of diarrhoea can lose up to 8.2 cm in height (Moore et al., 2001). Multiple studies identify the link between repeated episodes of diarrhoea and cognitive deficits, with a measured loss of up to 10 IQ points and 12 months of schooling by age 9 (Guerrant et al., 1999; Lorntz et al., 2006; Niehaus et al., 2002). Predominant brain myelination and synapse development occurs in the first 2 years after birth (Corel, 1975; Rice & Barone, 2000; Thompson & Nelson, 2001). By the age of 3 years, the human brain has nearly 200% of its adult number of synapses, after which nearly half of the brain synapses are gradually eliminated (Corel, 1975; Huttenlocher, 2002). This early brain development requires 87% of the newborn body’s metabolic budget, 44% at age 5 years, 34% at age 10 years, and approximately 25% in adults (Holliday, 1986).

The tendency in existing research is to look for and seek to resolve individual causes of diarrhoeal disease. Consequently, causal interactions between disease states and social conditions are understudied. We offer a syndemic perspective on childhood diarrhoeal disease, specifically examining the consequential interactions of pathogens, and pathogenic interactions with health and social conditions that are disproportionately common among disparity populations (those suffering disproportionately from poverty, social inequality, and economic and political marginality).

Drawing from available research and current policy agendas, this paper employs syndemic theory to: (1) examine the role of adverse biosocial interactions in increasing the total disease burden of enteric infections in low-resource populations; and, (2) explore novel

comprehensive disease prevention approaches, consistent with syndemic theory, that are likely to prove effective in generating sustainable and equitable disease prevention solutions. We present this perspective to highlight the potential for a syndemics analysis to enrich and extend policy discussions and planning related to diarrhoeal illness and other health challenges of impoverished populations.

The syndemic perspective

The term *syndemic* is a portmanteau derived from the Greek work *synergos*, meaning two or more agents working together to create a greater effect than each working alone; and *demis*, or “people,” commonly used in public health concept of epidemic, a disease classification that literally means “upon the people.” Much like the synergistic effect of multiple drug agents working simultaneously in a system, whereby the total response is more than the predicted sum of the two individual effects, the synergistic interactions of disease conditions that are perpetuated by social disparities generates a syndemic. The syndemics perspective recognizes that adverse social conditions, including social inequality, marginality, poverty, and other forms of political and economic oppression play a critical role in facilitating the clustering of both infectious and non-infectious diseases that lead to heightened potential for deleterious disease interactions (Singer, 2009).

While the development of a syndemic could indicate less overt inequalities in need of investigation, usually with infectious diseases we are arguing that conditions of poverty or other disadvantage (e.g. malnutrition, trauma, stress, environmental toxins) place bodies at heightened risk for diminished immune capacity, diminished body development and repair, and heightened exposure to pathogens and pathogen clustering interaction. Moreover, in addition to the burden of infection from one pathogen, disadvantaged populations commonly face multiple infectious agents, and these, in turn, often have the capacity to interact within human bodies to multiply their adverse effects. Syndemics theory therefore rests on recognition that health issues are never matters of biology alone but rather involve continuous bidirectional feedbacks between biological and social structure.

In the case of diarrhoea causing enteric infections, the syndemic framework offers an understanding of the complex and dynamic relationship between *micro-parasitism*, the biological proximate causes of disease, and *macro-parasitism*, the human social relations that are the ultimate origins of much disease. Globally, diarrhoea is of gravest threat to populations already at comparatively high risk for a range of threats to health and social well-being. In this, diarrhoeal disease both exposes the vast inequities of our prevailing global social structure and the limitations of current national infrastructures to respond effectively and justly to disease.

Syndemic interactions

Social conditions

Latest global epidemiological estimates on childhood diarrhoea project that only 2% of diarrhoea episodes progress to severe disease, with a worldwide case-fatality ratio of 2% (Fischer Walker et al., 2013). However, diarrhoea incidence and case-fatality ratios are

much higher within low-income countries than in middle- and high-income countries. Asian and African world regions retain the greatest proportion of severe diarrhoea episodes at 26%. Fifteen countries (Afghanistan, Angola, Burkina Faso, China, Democratic Republic of the Congo, Ethiopia, India, Indonesia, Kenya, Mali, Niger, Nigeria, Pakistan, Tanzania, and Uganda) account for 53% of the total episodes of diarrhoea globally and 56% of severe episodes. In 2011, 74% of the total burden of diarrhoea mortality in children under-5 years was in these countries. The highest number of childhood deaths due to diarrhoea in 2011 (50%) occurred in sub-Saharan Africa. This suggests that as disease outcomes become more severe, more of the global burden is concentrated in the most marginalized communities within the highest burden countries.

The greatest burden of disease occurs primarily in younger age groups, with 72% of deaths from diarrhoea occurring in children younger than age 2 years (Fischer Walker et al., 2013). The highest rates of severe diarrhoeal disease occur at age 6–11 months, as infants no longer receive passive protection from trans-placental and breast milk antibodies and begin to experience greater pathogen exposure from food, water and their wider environment (Fischer Walker et al., 2013). Risk of diarrhoea and diarrhoeal mortality then decreases with age. However, morbidity (i.e., stunting and cognitive impairment) related to moderate-to-severe childhood diarrhoea can have profound implications throughout one's lifetime.

Infrastructural limitations and poverty conditions increase exposure to diarrhoea-causing pathogens. Yet, even within marginalized communities there are levels of unhealthy environments that predispose children to increased burdens of disease. In India, for example, where 55% of households defecate in the open, children are two standard deviations shorter than the reference mean (Spears, 2013). Even children in the richest households in India are shorter than the international reference norms (Tarozzi, 2008). While this may be interpreted as an inappropriate use of international normalizing standards (Panagariya, 2012), Spears (2013) argues that the practice of open defecation creates a disease environment that exposes all residents to disease pathogens, though to varying degrees based on household economic capital and place of residence. In India, recurrent diarrhoea and death from diarrhoea are much more common among the lowest wealth index (Avachat, Phalke, Phalke, Aarif, & Kalakoti, 2011; Lahariya & Paul, 2010) despite somewhat universal exposure to diarrhoea causing pathogens.

Disease-disease interactions

Children living in diarrhoea endemic areas are often at higher risk for other infectious diseases which likely also have syndemic interactions with enteric pathogens, such as HIV/AIDS, pneumonia, malaria, TB, and measles. For example, a study of children under-2 years in Kenya revealed significantly greater episodes of diarrhoea in children infected with HIV as compare to those without HIV (van Eijk et al., 2010). Conditions of marginalization increase likelihood of exposure to diarrhoea pathogens and contribute to HIV infection in infants, as barriers to the prevention of mother-to-child-transmission of HIV in southern Africa include lower maternal educational level, limited health service accessibility and medical staff shortages (Gourlay, Birdthistle, Mburu, Iorpenda, & Wringe, 2013).

Interactions of diarrhoea pathogens with other disease states have been well documented (Singer, 2010). Concurrent infections with intestinal helminth parasites have been shown to impair host immunity to enteric pathogens (Chen, Louie, McCormick, Walker, & Shi, 2005; Su et al., 2012; Weng et al., 2007). Among 30 hospitalized patients with strongyloidiasis (round worm) infections in central Kentucky, 16 (53%) were found to have extra-intestinal infections, with enteric organisms identified in 13 (81%) of these patients (Al-Hasan, McCormick, & Ribes, 2007). Extensive research exists on the dynamic relationships between malnutrition-and-diarrhoea and increasingly environmental enteropathy (EE)-and-diarrhoea. In isolation, malnutrition is well recognized as a widespread health problem, accounting for 11% of the total global DALYs (disability adjusted life years) (Black et al., 2008). Malnutrition is associated with 53% of diarrhoeal deaths in children under-5 years (Kosek et al., 2003). In an elegant study in mice, Coutinho and colleagues (2008) revealed the synergistic effect of malnutrition and diarrhoeal pathogens on damaging intestinal architecture, thereby limiting nutrient absorption. EE, the condition resulting from chronic exposure to fecal pathogens, is also marked by alterations in intestinal architecture and increased intestinal inflammation (Korpe & Petri, 2012; Lin et al., 2013), and causes alterations in gastrointestinal tract (GIT) microbiota, which contributes to variation in mucosal immunity. Recent study findings indicate that rotavirus and oral polio vaccines are less effective in children who are malnourished and/or experience two or more episodes of diarrhoea in their first month of life (Haque et al., 2014). Reasons for this are not well understood, though the findings indicate a complex interrelationship between diseases, immune response, and gut microbiomes, further suggesting the potential importance of syndemic interaction.

Gut microbiota

GIT microbiota are essential for the development of the gut-associated lymphoid tissue and play a vital part in shaping the immunological repertoire of the GIT (Mayer, 2003; Quigley, 2008), regulating the epithelial barrier function and GIT motility, and providing support for digestion and host metabolism. Microbiota prevent colonization by pathogens in large part through competition for nutrients and access to adhesive sites and receptors on the epithelial surface (Sekirov, Russell, Antunes, & Finlay, 2010). Evidence suggests that GIT microbiota are 3–4 times lower following episodes of acute diarrhoea (Albert et al., 1978; Tazume et al., 1993; Tazume et al., 1990). Iatrogenic suppression of GIT microbiota that occurs following antibiotic use, perhaps for repeated episodes of diarrhoea or other bacterial infections including pneumonia, can also disturb the normal mechanisms of GIT microbiota and possibly increase likelihood of subsequent pathogenic infections (Cryan & O'Mahony, 2011). Finally, normal colonization of the gut appears to commence at birth (Palmer, Bik, Digiulio, Relman, & Brown, 2007). The microbiome of un-weaned infants is simple, becoming more diverse and numerous after 1 year of age due to diet and environment (Kurokawa et al., 2007; Palmer et al., 2007). This makes infants and young children especially vulnerable to infection by multiple enteric pathogens, a process further facilitated by weaning from the protective components of breast milk.

Growing evidence suggests the existence of an intimate bi-directional relationship between the GIT microbiota and the brain, termed the “Brain-Gut-Microbiota Axis” (Grenham,

Clarke, Cryan, & Dinan, 2011). Close interactions between systems indicates that any adverse effects caused by repeated enteric pathogenic infections early in life influencing normal GIT microbiota could dramatically influence neuronal functioning (Grenham et al., 2011; Lynn & Vanhanen, 2006). A study in mice without GIT microbiota revealed that the core neuroendocrine pathway, the hypothalamic-pituitary-adrenal axis, is overstimulated by mild restraint stress, but normal function resumes when GIT microbiota recolonize (Sudo et al., 2004). Additionally, brain-derived neurotrophic factor, necessary for neuronal growth and survival and expression of neurotransmitter receptors in the cortex and hippocampus, is decreased in mice without GIT microbiota (Sudo et al., 2004). Conversely, neuronal function can influence the GIT microbiota contributing to pathogenic infection. In mice, stress induces permeability of the gut allowing pathogens and pathogenic antigens across the epithelial barrier. This translocation activates a mucosal immune response, which causes temporary and long-term alterations in the composition of the microbiome (Bailey & Coe, 1999; Kiliaan et al., 1998; O'Mahony et al., 2009; Rhee, Pothoulakis, & Mayer, 2009). While germ-free animal models are not identical to human children who experience microbiome composition disruptions, they suggest that high stress environments experienced by children living in marginalized conditions may alter the GIT microbiome sufficiently enough to increase the likelihood of repeated pathogenic infection by various microorganisms causing moderate-to-severe diarrhoea in children.

Pathogen-pathogen interactions

Diarrhoea-causing pathogens may also be interacting with each other in ways that increase the severity of disease symptoms, as co-infections are frequent. Recent findings of the Global Enteric Multicenter Study (GEMS), a prospective case-control study of hospitalized paediatric patients with moderate-to-severe diarrhoea in seven diverse sites in Africa and Asia (Kotloff et al., 2013), suggest that co-infection with multiple diarrhoea causing pathogens is common, at least among children with symptoms severe enough to interact with the formal health system. One or more pathogens were identified in 83% of diarrhoea cases (compared to 72% in controls) and two or more agents in 45% of cases (compared to 31% in controls). Similarly in Brazil, 14% of previously collected rotavirus positive samples contained enteric adenovirus or norovirus (Ferreira et al., 2010). In India, diarrhoeal stool specimen analysis revealed that 63% of specimens had polymicrobial infections (Sinha et al., 2013).

The interactions between enteric pathogens that cause diarrhoea are not yet fully clear. However, research is emerging that offers an indication of possible synergistic effects of pathogens in promoting disease. Observational studies suggest that molecular interactions may occur as a result of co-infections that exacerbate disease symptoms. For example, a prospective study of paediatric patients hospitalized for acute gastroenteritis in Rome, Italy identified co-infection in 18% of patients (Valentini et al., 2013). Co-infected patients experienced more severe clinical presentations. A case-control study of 22 communities in Ecuador found evidence of rotavirus-Giardia and rotavirus-*E. coli* co-infections (Bhavnani, Goldstick, Cevallos, Trueba, & Eisenberg, 2012). Researchers reported that during co-infection the pathogenic potential of each agent appeared enhanced. Collectively, these findings suggest that moderate-to-severe diarrhoea in children may be the consequence of

microbial interactions. Of course, these findings might also serve as markers for poor social conditions, whereby multiple pathogens are able to infect children, though not acting synergistically to worsen health outcomes. Additional investigations are needed to tease out the molecular and anatomical level interactions that may or may not be contributing to more severe clinical presentations.

As outlined, these complex relationships between disease states, pathogens, microbiota, and the brain suggest an adverse feedback loop involving high-stress environments resulting from social conditions of poverty and marginalization with diarrhoeal disease. These relationships are accelerated by and accelerate the development of multi-disease syndemics that increase the burden of disease and negatively impact the social condition of sufferers (e.g., costs of care, long term health impacts).

Approaches to addressing childhood diarrhoea

Multiple direct and indirect strategies involving diverse stakeholders have been adopted to address diarrhoeal disease in children. In what follows, examples of direct, indirect and syndemic approaches are described and the effects on reducing disease burdens are discussed.

Direct approaches

The international bodies of UNICEF and WHO have guided the global public health agenda on childhood mortality, with a primary focus on the use of biomedical technologies. The *Integrated Management of Childhood Illness* (IMCI), developed in the mid-1990s to reduce under-5 mortality, focuses on health facilities. Although the program has been adopted by more than 100 countries, clinical coverage and quality of clinical care for sick children has not reached high enough levels to achieve the expected reductions in mortality or morbidity (Chopra, Binkin, Mason, & Wolfheim, 2012). Bottlenecks have been a primary impediment to access to and use of biomedical therapies (Gill et al., 2013). These include: inequitable supplies of key commodities and unequal provision within communities through public or private avenues; delayed development of national policies supporting use of certain newer strategies or integration of strategies into national plans (i.e., rotavirus vaccine); limited funding and resources; poor management and coordination of efforts; and inadequate advocacy. For example, despite evidence of effectiveness, the median coverage of ORS has decreased since the late 1980s, with only a third of children receiving this life-saving intervention (WHO/UNICEF, 2012a). Furthermore, even a decade after WHO/UNICEF released recommendations for treatment of diarrhoea with zinc, global uptake of zinc for the management of diarrhoea remains almost negligible (Bhutta et al., 2010).

WHO/UNICEF *Integrated Community Case Management* (iCCM) strategy for diarrhoea, pneumonia, and malaria launched in 2011 offered a community-level focus for disease prevention and treatment. Despite resurgence in the use of community health workers (CHW) in the delivery of case management of childhood illnesses, evaluations of these community-based strategies suggest that the quality of services is poor (Gill et al., 2013; Strachan et al., 2012). Contributing factors include: poor incentives to attract or retain workers, inadequate training, no systematic approach to supportive supervision, and limited

provision of appropriate treatments such as antibiotics, pre-packaged ORS, and zinc to CHWs despite their skills and access to the communities.

The recently announced WHO/UNICEF *Integrated Global Action Plan for the Prevention and Control of Pneumonia and Diarrhoea* (GAPPD) offers a more comprehensive approach to addressing childhood diarrhoea (and pneumonia) by offering a cohesive policy framework upon which local political and civil society leaders take the lead in the development of coordinated strategies. In so doing, the GAPPD aims to: close service delivery gaps by bringing together critical services and interventions; promote best practices and proven strategies; and establish healthier environments. The effectiveness of this integrated, multi-dimensional approach that attempts to speak across sectors is yet to be determined.

One concern with the GAPPD approach is the lack of direct attention to addressing social drivers of disease, including the promotion of poverty alleviation, improvement of water infrastructure, or assessing whether local communities are interested in the approaches suggested and willing to comply. For example, an analysis of 15 highly cost-effective interventions for the reduction in childhood diarrhoea (see Bhutta et al., 2013) published to coincide with the announcement of the GAPPD, did not include estimations for broader-based investments in water infrastructure, focusing instead on biomedical solutions. A qualitative analysis of the discussions of the multi-country workshop consultations, which ultimately led to the development of the GAPPD, identified limited or no country-level awareness and support for water, sanitation, and hygiene initiatives (Gill et al., 2013). This lack of national engagement in discussions of water and sanitation improvements possibly reflects the perceived magnitude of resources that are needed to develop or upgrade effective water supply and sanitation systems. This lack of conversation also highlights inequities in current geopolitical arrangements as country ministries feel beholden to the priorities of multilateral funders (Gill et al., 2013), resulting in external interests being favoured over national priorities, possibly contributing to limited national political will and civic engagement.

Indirect approaches

Taking a more indirect approach to childhood diarrhoea, the Tanzanian government has implemented pilot programs of a form of water 'rights and fees,' with the aim of improving water distribution, particularly during times of scarcity (World Bank, 1996). Consistent year-round water supply is necessary for hygiene and sanitation practices and food productivity thereby indirectly reducing exposure to enteric pathogens and preventing malnutrition. The program requires all commercial water users to register with the government and pay a fee to obtain a 'water right' that entitles them to an annual value of water resources. Unfortunately, evaluations of this program suggest that this form of government taxation has generated greater income and health inequalities, disproportionately burdening small-scale land owners who cannot afford to purchase the 'water rights' (van Koppen et al., 2004). Nevertheless, analysts suggest that small alterations in the program (i.e., eliminating taxation on small-scale farmers) could make the approach effective in addressing social inequalities that influence disease (van Koppen et al., 2004).

Syndemic approaches

In Brazil, via progressive social policies which complement the country's market-oriented reform policies, a syndemic approach to diarrhoea prevention has been implemented. Policies on poverty alleviation and reduction of inequalities are in line with historical evidence that suggests improvements in living conditions, not direct biomedical interventions or supplies of water, have a more significant influence on reducing infectious diseases (McKeown, 1978; Wegman, 2001). Though also attempted in other countries in different ways, Brazil's *Bolsa Familia Program* (BFP) is the world's largest conditional cash transfer program for poverty reduction. Initiated in 2003, BFP distributed funds to over 25% of Brazil's population, or 13.4 million families in 2011, with a total budget of US\$11.2 billion (Ministerio do Desenvolvimento Social e Combate a Fome, 2013). Cash transfers range from US\$18 to US\$175 per month, depending on the income and composition of the family (Lindert, Linder, Hobbs, & Briere, 2007). The conditional cash transfers require that children are sent to school where they receive at least one meal per day, vaccinations are completed per the Brazilian immunization program schedule (including rotavirus vaccine), routine health check-ups and growth monitoring are maintained per Ministry of Health guidelines, and women attend post-natal care services and receive health and nutritional education. An analysis of the effects of BFP on child survival revealed that under-5 mortality decreased as program coverage increased, with the greatest positive impact occurring on poverty-related malnutrition and diarrhoeal disease (Rasella, Aquino, Santos, Paes-Sousa, & Barreto, 2013).

The BFP and other conditional cash transfer programs are not without their limitations and may not be applicable in all situational contexts, particularly given the significant national investment required and potentials for misappropriation of funds. Nevertheless, the syndemic perspective presented here indicates that greater involvement of diverse stakeholders and strategies with equal or greater investments on improving social conditions can effectively address the biosocial dynamics of childhood diarrhoeal disease. The success of the BFP suggests that by operating through multiple mechanisms – improving social conditions by decreasing poverty and barriers to health care; improving food access and education; providing biomedical technology that directly addresses specific diarrhoeal diseases and disease-disease interactions – as per the syndemic perspective – results in significant improvements in childhood diarrhoea.

Implications

Syndemics represent a critical element in the health profiles of disparity populations and constitute an important contributor to health inequality. The study of syndemics has been termed a 'promising new frontier for public health action in response to the critical challenges of our time' (Leischow & Milstein, 2006, p. 405). Given the complex nature of childhood diarrhoea including interactions between existing disease conditions, pathogens, gut microbiota, the brain, and social environments, the syndemic perspective offers a way forward for potential interventions. Interventions that focus only on technologically advanced biomedical strategies are likely to fail, as they do not address the social conditions of disparity populations that promote disease clustering. While improving social conditions

among marginalized communities is infrequently the focus of global public health interventions, history suggests that it is the most advantageous way forward. As noted by Farmer and Becerra (2001, p. 5):

epidemic and endemic disease – are not solely biological; neither are they purely social. Yet, conventional studies typically rely on disciplinary approaches and fail to reveal the full complexity of these epidemics. Only by embracing a transdisciplinary, biosocial approach can we hope to describe fully these ‘tropical’ epidemics, and intervene successfully.

The GAPPD represents an emerging paradigm for public health interventions and development by emphasizing horizontal structuring, plurality across multiple sectors, collaboration between public and private partnerships, and ownership. Programs like Brazil’s BFP prove that such diverse approaches, though requiring significant national investment and coordination, can have profound effects on improving health conditions of marginalized communities.

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