Working Paper No. 24

Addressing Social Drivers of HIV/AIDS

Some Conceptual, Methodological, and Evidentiary Considerations

COMMISSIONED BY:

aids2031 Social Drivers Working Group

AUTHORED BY:
JUDITH D. AUERBACH
JUSTIN O. PARKHURST
CARLOS F. CÁCERES
KIMBERLY E. KELLER

Introduction

Interest in enhancing and applying social research in HIV prevention has intensified in recent years as it has become clear that the development, uptake, and effectiveness of the biomedical and behavioral strategies for HIV prevention that have dominated the intervention arena are very much affected by social and cultural contexts.[1] Additionally, there is now much wider recognition that social forces—hereafter referred to as "social drivers"—both influence HIV transmission (by influencing practices that can lead to infection) and offer possible points of intervention at the societal level (by altering those practices or the context in which they occur).

This recognition has resulted in a sense of urgency in the public health community to identify effective social-level, or "structural," interventions that can be rapidly deployed. But the arsenal of structural interventions—or, more generally, evidence-based and evidence-informed strategies for social change—is quite small, and developments in this arena have been hampered by significant methodological and evidentiary obstacles that have yet to be fully explored and redressed.[2,3]

We believe that further progress in this area involves two things: first, a better development and mining of the basic social science that should underlie structural intervention design, and second, a better explication and adoption of research methodologies that can effectively attribute causality and assess the nature of social-level change. Thus, this paper examines key issues in the conceptualization, operationalization, and measurement of social drivers of HIV/AIDS and evaluation of attempts to modify them. Our hope is that a deeper engagement with social science research and methods will increase the public health community's understanding of social drivers and permit the development of appropriate social-level approaches, including structural interventions that, in turn, will have a greater impact on HIV epidemics everywhere.

Constructs and Definitions

In order to mount an effective response to HIV/AIDS, it is essential to first "know your epidemic."[4] This means knowing whom and where HIV infections have hit in any setting. This can be ascertained by a range of methods, including epidemiological and surveillance data, national surveys, rapid assessments, and mapping techniques, that help identify which populations have been most affected and where new infections are likely to occur. Once the "whom" and "where" are known, the next question is, "Why and how?" This is where the social drivers discussion comes in.

HIV is transmitted by specific practices (fn1) among individuals and groups that occur in a social context. There is disagreement in the social sciences about the extent to which an individual's desires and practices are shaped by outside forces (social determinants) and how much they are a reflection of individual decisions to act (social action or agency),[5] but there is a common understanding that much of what humans do, think, and desire is influenced, if not determined by, elements of our society. In sociology, those core elements are norms, values, networks, structures, and institutions.

Norms are rules about behavior that reflect and embody prevailing cultural values and are usually backed by social sanctions (formal and informal). Values are ideas held by individuals and groups about what is desirable, proper, good, or bad. Networks are the webs of human relationships (including dyadic, familial, social, sexual, and drug-using) through which social (including sexual) exchange occurs and social norms are played out. Structures and institutions are the material and operational manifestations of social norms and networks, such as family units, organized religion, legislative and policy apparatus, educational systems, military and industrial organizations, etc., in which social interaction is patterned and, often, controlled. Beliefs, attitudes, behaviors, and practices of individuals and groups are framed by, and in turn influence, the dynamic nature of these elements of society.

These basic constructs about human interaction in social groups underlie the concept of social drivers in the HIV/AIDS context. While there is no standard or agreed-upon definition of social drivers, UNAIDS refers to them as "the social and structural factors, such as poverty, gender inequality, and human rights violations, that are not easily measured that increase people's vulnerability to HIV infection" (emphasis added).[4] As this definition conveys, not only are social-level phenomena difficult to measure, they also are difficult to define and therefore difficult to fully understand.

Indeed, the literature on social drivers and structural interventions is rife with contested and confusing terminology. (To a great extent, this reflects the fact that in the social sciences, all terms are contested, because it is recognized that language is created, interpreted, and used by humans who imbue words with subjective meaning.) For example, in some cases, structural factors are seen to be those aspects of a person's environment that either enable or constrain a given behavior,[6] while in others the term is used more broadly to encompass the determinants of that behavior in the first place.[2] Our use of "social drivers" is meant to refer to the core social processes and arrangements—reflective of social and cultural norms, values, networks, structures, and institutions—that operate around and in concert with individuals' behaviors and practices to influence HIV epidemics in particular settings.

Hypotheses and Causal Pathways

It is important to unpack how these aspects of social influence operate and facilitate HIV transmission in order to mount a social-level response. In both sociological and social-epidemiological conceptualizations, social drivers are understood not as unilateral variables that can be studied adequately in terms of causal, one-to-one relationships between any of them and HIV infection outcomes. Rather, they are interactive phenomena reflective of social and cultural processes, institutional practices, and sets of arrangements that facilitate HIV transmission or its prevention. Social drivers are complex, fluid, non-linear, and contextual, and they interact dynamically with biological, psychological, behavioral, and other social factors. So, for the purposes of conceptualization and operationalization, they must be characterized situationally and contextually.

For example, "gender inequality"—although mentioned in the UNAIDS definition as a major social driver in HIV epidemics—does not operate universally in one single way with respect to HIV vulnerability. Rather, it is generally understood as a reflection of social arrangements and expectations (institutions and norms) about males and females (including transgender, transsexual, and intersex persons) that varies by culture and society and that has differential impact on HIV epidemics. In a number of sub-Saharan African countries, HIV infection rates are three to four times greater among young women than young men, which many analysts attribute to gender inequalities between females and males—in particular, women's lack of economic independence relative to men.[7] But in numerous other countries around the world, where similar gender inequalities prevail, we do not see the same disparities in HIV infection rates between young women and young men (and may, in fact, find extremely high rates among transgender persons and among sub-sets of men who have sex with men [MSM]).

The interaction between gender arrangements and other factors—for example, biological factors, such as higher prevalence of other sexually transmitted infections (STIs), and other social factors, such as family separation due to economic crisis and migration that cause some women to resort to transactional sex as a survival strategy—might be associated with increased HIV risk in sub-Saharan African contexts.[2] In other places, however, extreme gender inequalities might interact with different family dynamics and economic arrangements such that being a woman implies less exposure to public life and also lower HIV vulnerability.[8] Thus, we cannot talk generally about "gender inequality" as a social driver of HIV infections, but must identify the specific ways in which gender (and sexuality) dynamics operate in conjunction with other social and cultural dynamics in particular social contexts to produce vulnerability, or not.

Poverty is another social driver often implicated in HIV epidemics and mentioned in the UNAIDS definition. A common mantra in the HIV community is that poverty is fueling the spread of HIV.[9] Indeed, an abundance of evidence appears to support this, including studies correlating socioeconomic status with indicators of risky sexual behavior,[10,11] and studies showing how manifestations of poverty (such as food insecurity) can be correlated with HIV prevalence.[12] Yet the "poverty drives HIV" hypothesis has been challenged recently by authors who have illustrated how, in several sub-Saharan African countries, it is wealthier groups that often see higher HIV prevalence rates,[13–15] and how across Africa, it is wealthier countries that tend to report higher national HIV prevalence rates.[14]

Neither poverty nor wealth is a social driver per se; rather, it is the context in which some people are wealthy and some people are poor that can lead to relational patterns resulting in forms of sexual networking that can spread HIV. Poor people in some settings may be more likely to engage in particular practices—perhaps earlier onset of sexual activity, or occasional transactional sex—which may increase risk of infection. Wealthy people in some settings may find that their wealth permits greater social and sexual networking, or allows them to have a higher number of regular sex partners—a pattern that may place them at risk, as well. It is therefore incorrect to ask whether it is either poverty or wealth that drives the spread of HIV (which assumes a simple correlation must exist and also ignores the fact that poverty and wealth usually coexist). Instead, it is necessary to understand that context determines the nature of social/sexual arrangements, which interact with both poverty and wealth to contribute to greater or lesser vulnerability.

Describing and drawing the causal pathways through which these complex and situationally defined social drivers operate to confer vulnerability to or protection from HIV transmission is challenging. In traditional epidemiology, the key element in evidence of a causal relationship is statistical association between two variables or factors; actual causal attribution implies that other conditions are met (e.g., time sequence, biological plausibility, dose response, etc.). A causal factor is assumed to increase or decrease the likelihood of a given outcome by a specific magnitude, usually measured through risk, odds, or hazard ratios. Over the past two centuries, and with the advent of social epidemiology, causal models have evolved from simple binary models (cause-effect) to increasingly complex, multi-level models[16] that can possibly include determinants at the molecular as well as the societal levels. Parallel developments have taken place in biostatistical models, wherein health outcomes do not have single causes but contributing causal factors. The contribution of these factors to disease is often non-linear, and certain of them can modify (i.e., increase or decrease) the effect of others (for example, the role of a genetic factor may increase with age). What is critical to keep in mind, however, is that the effect of each contributing causal factor is often not independent of others: the risk parameter (or the amount to which it contributes positively or negatively to disease occurrence) will change depending on other mediating factors and the context in which it is seen.

In sociology, even these kinds of causal connections are considered too mechanical, as they do not allow for the kinds of self-reflection and meanings people bring to their actions. Indeed, the beliefs, attitudes, and subjective reasons for acting in the ways humans do are themselves causal factors in relationships between variables (or determinants) in social life.5 Through our actions and the meaning we bring to them, we are constantly creating and recreating social institutions in time and space. Thus, society is not static—humans are constantly changing it—and we cannot even accurately describe, much less figure out how to intervene in, social life unless and until we grasp the concepts and meanings people apply to their actions.[17,18] This makes studying humans different from—and much more difficult than—studying events and phenomena occurring in the physical world.

Such challenges have, in many cases, led to many public health practitioners abandoning, or simply ignoring, the social perspective. This reaction may reflect frustration with what appears to them no clear guidance for action from social science. This frustration is understandable, but ignoring complexity and context does not make it go away. And a lack of universal solutions does not mean that there are no ways forward to make better-informed decisions and to design more effective interventions. In the biomedical and clinical science literature, the concept of "biological plausibility" is used to make inferences in the absence of mechanistic data but where an association "makes biological sense,"[19,20] meaning that it is consistent with what is known about biochemical, anatomical, or pathophysiological data or animal models.[19–21] We suggest a similar notion of "sociological plausibility" as a way to draw causal links between social drivers and HIV vulnerability where such associations "make sociological sense"—that is, where they are consistent with what is known about psychological, social, cultural, economic, and political data in specific contexts.

The context specificity and dynamic nature of the social factors that can drive HIV risk and vulnerability renders it inappropriate to make recommendations about interventions without adequate information about local situations. Rather, to have a meaningful impact on HIV epidemics, it is critical to take an approach that includes understanding, knowledge, and ongoing monitoring of local situations and enables contextually relevant responses to be tailored to those situations. Addressing social drivers, therefore, means taking a different tack from past public health strategies that assume a set of fixed-value causal factors exist that can be pre-defined and listed in advance for targeting. Rather, it would be more productive to think of social drivers as specific combinations of dynamic social factors occurring together in specific locations, leading to increased HIV vulnerability. Therefore, the starting point for operationalizing a social drivers approach should be the adoption of methodologies that promote knowledge, understanding, and monitoring of local epidemics and local contexts that can be analyzed in relation to key social factors and arrangements known or thought to be relevant—that is, having a "sociologically plausible" link—to HIV transmission. Any hypotheses and intervention or program choices should derive from this form of analysis.

Operationalization

A number of frameworks have been proposed to characterize and operationalize the range of social drivers and structural factors thought to influence HIV epidemics and serve as potential targets of interventions. Some focus on factors that affect people's ability to access or use protective devices, such as condoms or clean needles, taking the underlying risk behaviors and practices (the pattern of sexual behavior or the use of injection drugs) as given.[6,22,23] Others address broader social forces, such as social stability/instability and social equality/inequality, that may lead to particular patterns of behavior in the first place.[24] These frameworks also differ in how they address scope. Some look at the relatively micro-level phenomena shaping individuals' practices, while others look at much larger macro-level phenomena thought to drive broader behavioral patterns across a large population. Table 1 presents a breakdown of different strategies to address structural factors in HIV prevention, based on these general approaches to social structures. It lists some of the assumptions made in each approach, as well as issues and challenges involved.

Table 1. Issues arising from differing conceptualizations of "structural intervention"

	← Broader			Narrower →
Conceptualization (level) of structural influence	Social change: Factors affecting multiple groups (e.g., a region or country as a whole)	Targeted group structural change: Social structures affecting specifically defined population groups (e.g., MSM, mine workers, schoolgirls)	Immediate constraining/enabling structures: influencing the uptake of specific (desired) behaviors (e.g., enabling harm-reduction activities)	Non-structural: classic behavior change strategies
Assumes	Behavior exists in patterns Patterns are determined by non-individual (social) structures and institutions Social structures and institutions are not fixed but change over time for a variety of reasons Change process may be indirect or slow	Behavior exists in patterns Key groups share factors which determine (some of) their patterns of behavior Patterns are determined by both group-specific and larger social structures and institutions; at least some of these can be targeted for change	Risky behaviors exist and are difficult to change Harm reduction enables change to existing desired behaviors Making key technologies available or acceptable will increase their use	Behavior is individually determined Human agency shapes risk behavior Information shapes human agency
Attempts to change	Situations believed to shape behavioral patterns across groups	Factors believed to shape behavioral patterns for a given group	Rules, services, and attitudes toward a set of harm-reducing technologies	Ideas and views of personal risk

Directness of causality (proximal or distal)	Distal; often indirect influence on behavioral patterns, or influence through multiple interacting factors	Mixed; may be proximal or distal depending on the group and what is believed to shape their behavior	Mixed, but mainly proximal	Proximal
Issues	Identification of social structures and institutions is not precise Interventions difficult to construct and evaluate as social change rarely arises from formal organizations doing interventions The more distal, the more chances for unforeseen consequences	Design of interventions requires detailed information on target group May not identify all relevant factors Chances for unforeseen consequences, making evaluation difficult	There may not always be existing desire to use harm-reducing technologies; e.g., in most cases, people may not wish to use a dirty needle, but many people give reasons why they desire not to use a condom Relatively easy to design or evaluate	Easiest to construct interventions and evaluate Human agency often limited in many settings Historically the most common approach to HIV prevention Historically a highly ineffective approach to HIV prevention on its own
References/ examples	Gupta and colleagues 2008[2]; STOP AIDS Project 2009[25]	Blankenship and colleagues 2006[3]; Cohen 2000[23]	Blankenship and colleagues 2000[6]	Coates and colleagues 2008[27]

Despite the differences in approach, it is evident that the operationalization of social drivers and social responses must begin with a clear understanding of the level at which it is hoped an intervention or larger social response will work (i.e., targeting a specific group of individuals, or acting to reshape the broader social, legal, and/or economic environment), and to what extent fundamental behavioral patterns are seen as fixed or changeable (i.e., to enable already desired practices, or to fundamentally change practices to those more desired and/or to allow people to make informed, autonomous decisions about their actions, and/or to create an environment that is inherently less "risky"). Another way to frame this is by distinguishing interventions that are "ameliorative"—that is, that target proximal risk factors that link social position to health (e.g., lack of access to male and female condoms for commercial sex workers)—from those that are "fundamental"—that target the distal, underlying social conditions that produce disparate health outcomes (e.g., criminalization of prostitution).[28]

Figure 1 attempts to show how a structural approach to HIV prevention might progress through a series of steps in order to plan any intervention of a structural nature. As shown, there are distinct steps where the factors shaping behavior are considered (step 2) and the level of possible structural intervention is explicitly addressed (step 3). The figure illustrates the types of information that may be needed at each step, as well as some potential information sources for such data. It should be clear that a structural approach requires a good deal of information—much of which is specific to the target community—from multiple sources. At the same time, the structural approach enables multiple intervention strategies, depending on what level of structural change is targeted (or how social drivers are framed).

Figure 1. Steps and information needed in structural interventions

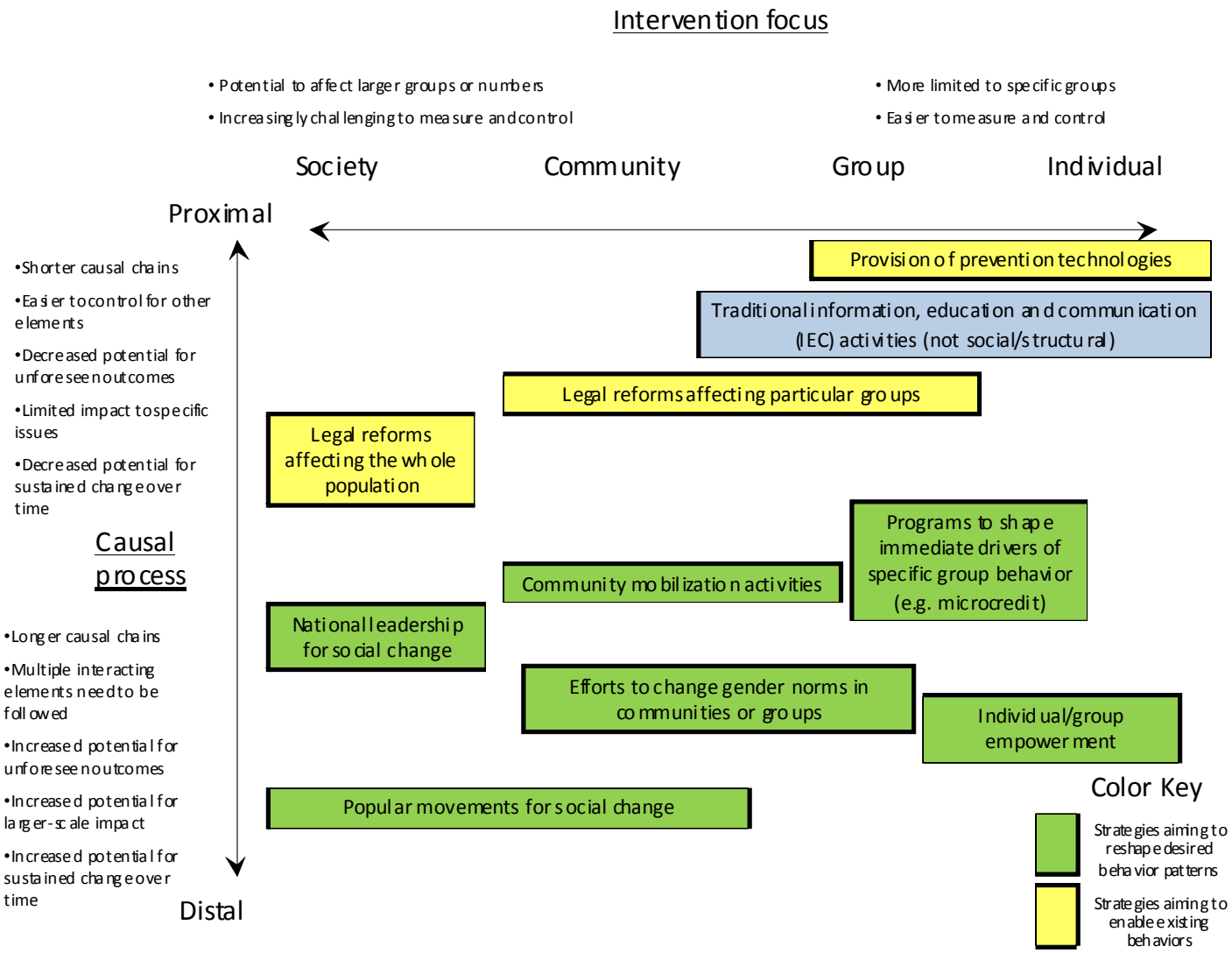
	Step	Information needed	Evidence sources or tools
1	Identify the target populations and/or locations for intervention	Epidemiological data of key affected populations (i.e. "knowing your epidemic")	Epidemiological surveys Surveillance data
2	Identify the key behavioral patterns and drivers of behavioral patterns for the target population	Epidemiological and behavioral data for specific groups In-depth understanding of behavior patterns and determining factors Knowledge of mediating context elements Identification of causal chains leading from deeper structures to risk (i.e. "know your target population")	Survey data Surveillance data Focus group discussions In-depth interviews Observational methods (e.g. expert or 'peer' ethnography) Additional correlating data
3	Choose level of structural intervention	Knowledge of what factors (from step 2) are amenable to change Theory of how changes can be brought about	Historical data/analysis of structural changes in similar contexts Evaluations of past structural intervention efforts.
4	Describe planned and potential changes and outcomes	Potential outcomes, positive and negative, arising from changes to broader structures	Modeling estimations and predictions Comparison with other areas of similar context
5	Design the intervention	Specific program resources, timing, and scope	Project planning tools
6	Implement, monitor, evaluate, and give feedback	Description and measurement of: - intervention mechanisms - contextual features affecting outcomes - mechanisms of social and structural change and - ultimate outcomes	Multiple methods and tools depending on nature of intervention. Process, operational, and outcome evaluation all critical.

One example of the challenges that can be involved in deciding where and how to frame social drivers and guide social responses when multiple factors are at play at different social levels, and where causality may be elusive, can be provided by the example of HIV prevention in migrant mine workers in southern Africa. It may be that mine workers, and the sex workers they often patronize, have limited access to condoms and information, or face specific cultural barriers making use of condoms unacceptable. A structural intervention approach in line with Blankenship and colleague's6 framework might look to intervene to make condoms more available or more acceptable in these communities. Yet studies have shown that the living and working conditions of mine workers may drive men to high-risk sexual activity out of a lack of social support and inadequate workplace safety.[29] Changes to living conditions or improved safety in mines might be seen as structural changes that could help prevent HIV.

Alternatively, one could take a much more macro-level focus and look at the country's dependence on migrant labor overall. The focus then becomes understanding and addressing how a country becomes extremely reliant on migrant labor, the characteristics of migrant pools, and whether alternative economic activities or labor rules and laws might help change the patterns of migration that have been linked to HIV prevalence in various settings. In each case, different bodies of information are needed to help plan the intervention and predict outcomes, and each relies on an active effort to target the intervention to this specific community, in a given context.

The project of social science and social epidemiology is to unpack the social drivers and social arrangements, such as those articulated in the migrant worker example above, to better understand how they operate in concert with HIV transmission dynamics at the individual, couple, and social network levels in particular social contexts, and then to determine how best they can be modified for HIV prevention purposes. An "ecosocial" and multi-level perspective16 is one approach that links biological and ecological analysis with an assessment of the social production of disease, while attending to scale and level of organization (individual, group, society). When considering how to design a social/structural intervention based on this analysis, it is imperative to define clearly the units of observation and the level(s) of change anticipated, and to make explicit any assumptions or hypotheses about the relationships between these units and patterns of risk and vulnerability in specific contexts (see Figure 2).

Figure 2. Conceptual mapping of structural interventions based on the scale of intervention (horizontal axis) and level of intervention (vertical axis).



Methods, Measures, and Evaluation

The continued pace of the HIV/AIDS pandemic requires urgent yet innovative responses. Policy-makers, program developers, and funders must make difficult choices about which HIV prevention strategies to explore and implement, often in the face of incomplete or weak evidence. Although evidence is only one factor that decision-makers use when determining what to do, its value is multi-faceted. Evidence can help us avoid doing harm (e.g., exacerbating infections, causing serious side effects), avoid wasting precious resources (human, material, and financial), improve the effectiveness (including cost-effectiveness) of programs, contribute to the quality of programs (including people's experience of them), and feed back into scientific inquiry to produce new understandings and responses and, subsequently, better evidence. All this should, ultimately, produce better HIV prevention outcomes, specifically reductions in HIV incidence and prevalence.

There is a robust discussion in public health science about how best to evaluate structural interventions and social change with respect to generating evidence of HIV prevention outcomes.2 Social change involves a wide range of shifts and changes, while HIV prevention has one very specific biological outcome of interest. The challenge comes with attempting to integrate social-level interventions into a positivist health-science paradigm characterized by the identification of cause-and-effect relations operating at the level of the individual, irrespective of context. [30]

HIV prevention has been dominated by behavioral interventions, which aim to change knowledge, skills, and behaviors among individuals and small groups, and biomedical technologies, which aim to provide individuals with products and physical tools that they can use to reduce their risk of acquiring or transmitting HIV. These efforts reflect the ascendance and adoption of the evidence-based medicine approach within public health research and practice, which has been influential in determining what constitutes appropriate evidence of intervention effect. This approach relies on systems of rating and ranking study designs and levels of evidence as a way to guide clinical and public health decision-making and practice. In these ratings, the strongest level of evidence (referred to as "Level 1") is given where there are multiple large-scale, randomized controlled trials (RCT) that can be systematically analyzed. Observational and qualitative studies are rated much lower. These rankings have been promulgated by such entities as the Cochrane Collaboration, the U.S. Preventive Services Task Force, and the U.S. Centers for Disease Control and Prevention and, as such, have served over time to elevate the RCT to the level of "gold standard" and to denigrate other methodologies in public health and related social science fields.[31,32]

But, as numerous authors have argued, the RCT has many limitations—both methodological and practical—for social- or population-level interventions. [25,33–37] These limitations include the availability of sufficient populations and sample sizes, contamination across units of comparison (e.g., experimental and control communities), length of follow-up, low external validity (generalizability), high costs, ethical considerations, and inhibition of innovative research questions that may not lend themselves to experimental methods.[32,37]

In an RCT, the intervention under study is allocated randomly to a subset of participants, and outcomes (e.g., infection, behaviors) are measured in both the intervention and comparison groups; when those differ significantly between groups in favor of the intervention group, then the intervention is considered efficacious. This operation assumes intervention principles work in the same way in any population—that is, that the mechanism of action of an intervention is potentially universal, as in a drug trial where the biochemical reactions in one human are expected to be the same in another. How something works is taken as given. RCTs assume that context is controlled for (by comparing "like" individuals and communities), as context otherwise might confound the presumably universal effect: design and analysis are used to allow for the isolation of the actual effect of the specific intervention. Yet social intervention does not work in this way. The mechanism by which social change takes place is not universal. Not only can context not be controlled for, it shapes how the intervention works in the first place34 and is inseparable from the intervention. More generally, the preventive effect of structural changes, such as those related to higher-level environmental change (e.g., legal changes), cannot be tested in experimental designs for practical and ethical reasons.

It is important to recognize what RCTs—and experimental methods more generally—can and cannot do. RCTs are very good at ensuring internal validity and establishing efficacy, but limited when it comes to external validity and establishing effectiveness.[38] They are also very good at assessing whether something works, but not why it works.[39] As such, experimental evaluations may be necessary to ascertain pre-defined kinds of impact, but they cannot be relied on alone to address social drivers or to evaluate interventions seeking to bring about social change.

Understanding social drivers and designing and evaluating social responses requires a broad range of methodologies and standards of evidence (see Table 2). Classical experimental designs may be appropriate for some structural interventions at the social network, community, neighborhood, or venue level, although the requirement for multiple units of intervention (or sites) in order to achieve adequate statistical power can be daunting and expensive.[40] Quasi-experimental designs may be more feasible and appropriate for assessing social change through "natural experiments"—organic, spontaneous social action—or the staged introduction of interventions.[40] Process evaluation and operations research are necessary elements to any evaluation aimed at explaining how the intervention worked and for whom, and what factors facilitated the impact.[34,36] These analyses are particularly helpful when looking to broader implementation and scale-up of programs.

Table 2. Strategies and methods useful for addressing key issues in structural interventions

Structural and social intervention challenges	Specific questions raised	Possible evaluation strategy	Common methods
Causality (effectiveness) of intervention is not certain	Measurement and attribution of causality: *Is the intervention effective?*	Experimentation and measurement before and after Modeling and comparison to "null hypothesis"	Quantitative methods Epidemiological methods Modeling
Mechanism of intervention is not certain	How to identify why an intervention works for a particular group	Process analysis within an intervention or a controlled experiment	In-depth interviews Focus groups Other qualitative methods
Interventions appear to have different impacts for different locations or groups	Investigation of context-specific elements which shape intervention efficacy	In-depth analysis of group dynamics	Ethnographic methods In-depth interviews Focus groups Participatory methods
		Process evaluation within multiple interventions	Standardized or comparative qualitative investigation across sites
		Comparison to other examples	Comparative case study
		Prediction and estimation ("best guesses")	Mathematical modeling and validation through additional data (quantitative)
Longer causal chains (indirect; causality through multiple processes)	Need to trace out causal chains	'Tracing" of causality (mapping back possible reasons for behaviors or outcomes)	Epidemiological and/or behavioral data combined with in-depth qualitative investigation (e.g., ethnography)

	Possible multiple pathways to HIV infection or prevention	Comparing qualitative and quantitative differences between different sub-groups along the paths from distal factor to risk behavior	Multi-method
Multiple factors shape the outcome, each working through its own causal chain	Need to identify other influencing factors	Natural experiments with in-depth analysis, comparison of multiple cases	Comparative multi-method studies
	Need to determine arrangement of those contributing factors that influence any intervention	Case studies combined with in-depth analysis	Comparative multi-method studies
	Need to identify patterns of contributing factors to enable generalizations across contexts	Case studies combined with in-depth analysis	Comparative multi-method studies

Observational data derived from "natural experiments" and designs such as cohort, case-control, cross-sectional, and "ecologic" studies are key for the generation of hypotheses about causal relationships that can be explored through experimental designs. Quantitative and qualitative social science methods—such as surveys, interviews and focus groups, behavioral and participant observation, life histories or narratives, case studies, policy and content analysis, network mapping, and mathematical modeling—also play a key role in the description of patterns and identification of contexts that lead to causal hypotheses.[40,41] In addition, program monitoring and evaluation are key to obtaining field data about the actual benefit of implementation of programs (i.e., effectiveness). Evidence derived from these methods includes measurements of disease occurrence (e.g., HIV incidence and prevalence outcomes), measurement of practices (e.g., self-reported sexual and substance use behaviors), psychosocial attributes (e.g., attitudes, beliefs, and meanings), measures of program reach, and various social indicators (e.g., of educational and economic status, access to services). Data collection linking monitoring and assessment with modeling should be ongoing to account for the dynamic nature of social contexts and HIV epidemics.

Choice of methods for structural intervention design and evaluation should always be informed by the causal hypothesis and the scope and level at which the intervention is attempting to work. There is a continuum of outcomes of interest ranging from changes in the practices of individuals

and groups to processes of social change affecting societies as a whole. Traditional HIV prevention through "information, education, and communication" strategies—sometimes called "behavior change communication"—focuses the intervention at an individual (or sometimes a group) level, and similarly has the reduction of "risk behaviors" of those targeted individuals as the outcomes of interest. Such approaches are conducive to evaluation quantitatively and through group comparison (e.g., individual-level measures and experimental methods). Once the desire is to address broader structures—either those structures directly enabling group and individual behaviors, or more distal structures shaping patterns of social practices—qualitative and observational methods may be more appropriate for providing relevant explanations, as well as outcome measures.

Choice of methods, then, must respond to the issues addressed. Is the intervention attempting to enable existing behaviors and practices? Is it meant to alter patterns of behavior for a specific group (yet not change the larger society in which they act)? Or is it attempting to alter a pattern of practices and the structures in which they are produced and enacted across a country as a whole? The answers to such questions will fundamentally affect the methods chosen and the balance of those methods for evaluation.

Even if the ultimate outcome of interest is quantifiable, such as HIV incidence, structural interventions themselves may be less direct in their causality than biomedical or behavioral interventions. Clearly, in those cases, an experimental design would have limited value and could even artificially separate the intervention under study from the context that is essential to its pertinence. This should not mean, however, that those structural interventions should be considered in principle unproved or not amenable to testing. Rather, it should mean that for those cases alternative designs should be acceptable as legitimate, and criteria should be set to establish when reasonable evidence of effectiveness (not efficacy) exists. Ultimately, structural interventions can only be expected to produce context-dependent evidence of effectiveness in reducing HIV risk at the individual level. These approaches are more suited to creating change that will reduce HIV vulnerability, thereby creating the enabling environment for risk reduction. Rigorously conducted social science methods and the evidence they generate are valid for making claims about effectiveness of social-level approaches, including structural interventions.

There are a number of things to consider when evaluating the quality and strength of evidence from structural (or other) interventions for HIV prevention for the purposes of deciding what to implement and scale up. With respect to quality, some criteria include transparency in research design, including theoretical framework and data analysis techniques; representativeness of the data (at least within a specific population or community); appropriate analysis of all relevant data; internal and external validity; and plausibility of findings.[42] In considering the strength of evidence, it is important to assess the intervention's feasibility, potential for adverse outcomes and unintended consequences, acceptability in the target population (or community), potential effect size, and whether it produces other health or social benefits.[42] Table 3 describes what features might be included in high-quality or low-quality structural interventions.

Table 3. Comparison of elements of high and low quality structural interventions

Element of intervention	High-quality structural interventions	Poor-quality structural interventions
Reasoning behind research or intervention design	Transparent: hypothesized mechanism of effect described for the target group; assumptions stated	Unexplained or vague
Intervention level (proximal or distal)	Research and evidence driven: identification of that level as important to the behavior of the target group	Arbitrary: based on "hunches" or "common sense," or done because that is what the implementing agency normally works on
Consideration of other mediating factors and limitations on effect	Considered	Not considered
Potential for adverse outcomes or unexpected consequences	Explicit: based on review of similar cases or valid theories and evidence	Not considered
Potential size of effects and spill-over	Considered	Not considered
Evaluation	Multi-method evaluation Triangulation of sources for complex phenomena Attempts to assess causal chains and how change in distal structures leads to behavior shift (e.g., through process evaluation and operational research) Outcomes linked to contexts Ideally some biological outcome or marker included if length of intervention makes effects at that level plausible	Limited Only single outcomes evaluated, without investigation of processes at work or operational elements Only final outcomes evaluated, not upstream causal chains Non-biological, non-behavioral outcomes only (such as "awareness" or "numbers reached")

One approach for assessing more distal-level structural interventions and social change involves the triangulation of multiple data sources in a multidisciplinary, multi-method case study (fn 2). This approach may be particularly helpful to decision-makers when there is a dearth of Level-1 data and when a rapid response is needed.[43] Conventional analysis emphasizes data considered of the highest scientific rigor (Level 1, generated through RCTs), relies on statistics as a basis for conclusions, emphasizes internal validity, is based on independent samples, and employs mathematical modeling. In comparison, triangulation analysis emphasizes using the best possible existing data, focuses on plausibility as a basis for drawing conclusions (with or without statistics), emphasizes external validity, is based on interconnected pieces of the same picture, and is interpretive. Triangulation also can be seen as an iterative methodology, in that there is, at least ideally, an interactive loop between data-gathering from multiple sources, data analysis (including observational), and refinement of hypotheses.[43]

Another approach for ex post evaluation of a broader-level structural intervention or policy change (e.g., a national response to the HIV epidemic that started at a certain point of time) is modeling (see, for example, Kirby 200844). The "natural" evolution of the epidemic could be modeled based on actual epidemiological data from the period prior to the broad-scope policy change. This model then would be tested to assess its ability to describe actual epidemiological outcomes from the period after the policy change was implemented. If the null hypothesis that the epidemiological facts can be described by the model cannot be rejected, then no evidence would have been found of the effectiveness of the policy change. On the other hand, if there is no good fit between the model predictions and the actual data, then an alternative hypothesis (i.e., the program had an impact) would be needed. Two key issues in this approach are (1) the model assumptions are fundamental and should be well discussed, and (2) the isolation of specific effects of program components (e.g., effect of a harm reduction program rather than of a condom distribution program) would likely not be possible.

Illustrative Cases

Following are three illustrative examples along the spectrum from rigorously designed experimental interventions to indigenous social change that highlight some of the important conceptual, methodological, and evidentiary issues in social and structural approaches that we have raised above.

SHAZ!: Feasibility Study of A Microcredit Intervention

As noted earlier, poverty and gender inequality are often construed as structural pathways to HIV/AIDS risk for women and girls globally.[4,7] By addressing gender inequality and economic empowerment, microenterprise—also referred to as microfinance and microcredit—is one potential intervention model for HIV/AIDS prevention where rates of infection are alarmingly high among women and girls. Small loans are used for income generation and have the potential to reduce poverty directly while also facilitating better health.[45] Additionally, microfinance programs have been shown to foster empowerment for women, improve child health and welfare, and increase women's agency in intra-household decision-making.[46] A number of

microfinance models exist and include a wide range of activities, such as basic life-skills training, development of commercially viable products and services, access to markets, financial training, and financial support. [47]

SHAZ! (Shaping the Health of Adolescents in Zimbabwe) is a microcredit and life-skills training and mentorship program, based on "gender and power" and "women's empowerment" theories, that attempted to address gender inequality, poverty, and HIV-risk. The program aimed to apply to a population of young orphaned girls in Zimbabwe a microcredit approach that has been successful in economically empowering adult women. The ultimate goal of SHAZ! was to reduce HIV risk, and a pilot study, using a mixed-method approach, was first conducted with a small sub-sample of 50 girls to test its feasibility. The mean age of participants was 17·5 years, and of the [49] participants for whom there were complete data, 86% had lost either their mother or both of their parents. Forty-three percent of the girls had ever had sex, and of these, one-quarter said that they had been tricked or coerced into having sex. Four of the sexually active participants were HIV positive.

Quantitative and qualitative findings from the quasi-experimental pilot study shed light on the feasibility challenges of a microcredit intervention in this population. Participants had consistently high levels of attendance at life-skills training. Eighty percent of the participants developed business plans to receive microcredit loans. While a 70% rate of loan repayment was expected at six months, only 20% of the girls had begun loan repayment and 6% had repaid in full by that point. Semi-structured interviews conducted three to four months after participants received loans found that for those five individuals who had begun repayment on schedule, reported previous business experience or family support were crucial to success. The sole participant who had paid back her loan in full reported having both capital and family support. Those girls who were unable to begin loan repayment on time reported neither family support nor assets and capital.

The SHAZ! pilot study illuminates the difficulties of achieving intended intervention outcomes in a highly volatile political and economic environment such as Zimbabwe. SHAZ! tailored the microfinance model to account for the economic situations and levels of training of the participants. Most of the girls in the study had to start from scratch as they had no business or service to "scale up." Loans were given out in one lump sum rather than in weekly installments and no group-based lending model was used, which meant that there was no social pressure to repay loans.[48]

Most importantly, there were a number of unintended consequences of note in this study. Ten of 14 participants engaged in the "buying and selling" of goods. This type of business—purchasing goods in town and transporting them for sale in areas where these goods were scarce—meant that the girls had high transportation costs, and moving goods back and forth made the girls vulnerable to theft and extraction of bribes for release of goods by police. Furthermore, this model forced participants to sell on credit, which demanded costly and time-consuming follow-up trips for collection. The girls' personal safety was also threatened as a result of traveling to unfamiliar places without safe accommodation. In-home theft occurred where secure places to store money and goods were lacking. The mentorship aspect of the intervention proved

unsuccessful also, as participants did not have the funds for transport to meet with mentors. In addition, the girls were discouraged by the lack of structured support these mentors provided.

The larger political and economic context also likely contributed to the poor feasibility outcomes, as inflation increased from 56% in 2002 to 238% in 2004, when the intervention occurred and, thus, the values of the loans declined rapidly during this time. (The situation is no better today, as inflation sky-rocketed to 11·2 million percent in 2008, according to the CIA World Factbook.49)

The SHAZ! pilot program's experience illustrates the difficulties in accounting for the range of social, political, and economic factors that can influence the efficacy of any particular intervention. But this does not mean that microfinance programs should not be attempted, as they have been effective in economically empowering women as a way to reduce gender inequality and intimate partner violence in a number of other settings, including South Africa.[45] However, their effect on HIV outcomes has yet to be demonstrated, which could take many years. This leaves open the question of whether it can be proved that addressing gender inequality will actually yield reductions in new HIV infections.

Uganda: Multiple Methods for Evaluating an Indigenous Community Response

It is well established that HIV prevalence in Uganda declined dramatically and systematically in the early- to mid-1990s from its peak in the late 1980s. Realizing that sexual behaviors were driving the country's HIV epidemic, President Yoweri Museveni engaged many sectors of society, through a broad-based public health intervention, to change social norms surrounding sexual relationships between men and women. It appears that changes in informal institutions led to changes in individual sexual behavior, which ultimately led to a dramatic and significant decline in HIV prevalence.[50]

While this decline has been attributed to changes in sexual behavior, questions about the type of sexual behavior change still remain with respect to the relative importance of abstinence (delay of onset of sexual intercourse), partner reduction outside of marriage (or outside of primary relationships), and condom use in influencing the dynamics of the Ugandan HIV epidemic.[51] In a recent analysis, Kirby[44] undertook a triangulated methods approach in an attempt to settle this debate. The study analyzed eight types of evidence: (1) models of HIV prevalence and incidence in Kampala and other cities in Uganda, (2) reports of behavior change in the primary newspaper in Uganda, (3) surveys with questions about perceptions of personal behavior change, (4) large demographic and health surveys (DHS) collected in 1988/89 and 1995 and large Global Program on AIDS (GPA) surveys in 1989 and 1995 with questions about reported sexual behavior, (5) smaller, less representative surveys of reported sexual behavior collected in other years, (6) interviews with key informants and focus groups, (7) reports of numbers of condoms shipped to Uganda, and (8) historical documents describing the implementation of HIV prevention programs in Uganda.

While each source of data has limitations for explaining changes in sexual behavior, Kirby argued that the limitations of one study may be offset by the strengths of another study.

Collectively, these sources showed the timing of the shifting social norms relative to changes in HIV prevalence. Modeling of HIV incidence and prevalence and reports of behavior changes in newspaper articles are consistent in suggesting that sexual behavior began to change around 1987, when incidence peaked and began to decline. News articles show that this decline was due largely to the promotion of "zero grazing" (monogamy rather than concurrent sexual partnerships). Reports of shipments of condoms to Uganda also indicate the timing of increased condom distribution, although such reports cannot point to actual condom use.

Additionally, DHS and GPA questions about behavior change and the resulting survey data gathered in 1988/89 and 1995 offer comprehensive and strong evidence that the Ugandan population began to experience later sexual debut for men and women and decreases in extramarital sexual relationships. Additionally, these data suggest large increases in condom use by 1995 in extramarital and non-cohabiting partnerships. Finally, documents describing programmatic efforts to address HIV/AIDS point to an emphasis on population-wide behavior changes and identify when these changes occurred. Kirby's analysis showed that by 1986, HIV prevention efforts in Uganda were primarily focused on monogamy and partner reduction, and that by the early 1990s, condom provision and use was also strongly encouraged.

Uganda has become a case study in how a government-led public health intervention may affect informal institutions surrounding sexual relationships and lead to dramatic, long-term population-wide shifts in norms and practices with epidemiological impacts. Kirby's triangulated methods approach provides a consistent picture of when and which sexual behaviors changed that ultimately led to a dramatic decline in HIV prevalence. As such, it makes the case for the validity of multiple data sources, both scientific and lay, for generating confirmatory evidence of indigenously generated behavioral, social, and epidemiological change.

CPOL: Adapting and Replicating Experimental Interventions

Various U.S.-based trials conducted in the 1990s showed interventions using popular opinion leaders (POL) to promote the development of safer-sex cultural norms to be efficacious in reducing sexual risk among MSM in small cities,[52] black MSM,[53] and women in lower-income housing.[54] These interventions were applications of the "theory of diffusion of innovations."[55] Given the need to develop new, low-cost options for HIV/STI preventive interventions in international, resource-limited settings, an intervention model was conceived that adapted the POL program to settings in lower- and middle-income countries (LMIC). The U.S. National Institute of Mental Health invited applications from sites composed of one U.S. research institution and one LMIC partner to participate in a cooperative agreement to test the Community Public Opinion Leader (CPOL) intervention. Five sites participated in the study, each with a distinct population: (1) market workers in Fu Jhou, China; (2) winery patrons in slums in Chennai, India; (3) young unemployed men on the street and young women and transgender persons who interact with them in shanty-towns in Lima, Chiclayo, and Trujillo, Peru[56]; (4) students using dormitories in Saint Petersburg, Russia; and (5) patrons of commercial places in rural growth points across Zimbabwe (fn 3). For the final definition of study populations, extensive ethnographic work was conducted, as were epidemiologic studies that demonstrated high prevalence of HIV/STIs and/or risk behaviors. The ethnography also guided the identification of individual CPOLs (about 15% of the total target populations) who, if willing to

participate, would be trained to share with their peers their positive experiences with safer sex and the importance they gave to HIV prevention.

The intervention, tailored to each site, was set to last two years. In each site, between 20 and 36 randomization units were allocated to either the intervention or the comparison arm. Measurements, conducted at baseline and at 12 and 24 months, included an extensive social/behavioral questionnaire using CAPI (computer-assisted personal interviewing), as well as testing for HIV, HSV-2, syphilis, Chlamydia, gonorrhea, and Trichomonas. Non-viral diseases diagnosed were treated, and pre- and post-test counseling were provided.

The primary hypothesis guiding the trial was that, after two years, the prevalence of unprotected sexual intercourse with non-steady partners and/or incidence of any STI would be lower in the intervention communities than in the controls. After two years of intervention roll-out in each country, no differences were observed between experimental and control groups in either risk behaviors or STI incidence, neither in the composite five-country sample nor in any of the countries taken separately. Moreover, while unprotected sex with non-steady partners decreased in both intervention (by 32%) and control (by 34%) communities, STI incidence remained high in both.[56]

A number of questions arise when analyzing these study outcomes. First are those related to assumptions about the operation here of social diffusion theory. For example, can safer sex messages be treated as an innovation? What would be the effectiveness over time of preventive messages of this sort? Can social innovation be implemented based on a top-down approach of this kind? Second are questions related to the comparability of sites. For example, is it valid to include five very diverse populations across the world, with different levels of HIV prevalence and different epidemiological dynamics, in this kind of trial? Third are questions about timing: Could an intervention based on changes in cultural norms through CPOLs produce changes leading to reductions in STI incidence that are identifiable after two years? Finally, there are questions related to measurement: Could the measurement approach (i.e., intensive yearly behavioral and biological assessments preceded by counseling) applied to both intervention and control participants (followed by STI treatment, if needed) itself constitute a significant individual-level intervention that offset any effort made at the level of social norms in the intervention communities?

The null findings of the CPOL trial, while not necessarily proving that the intervention was ineffective, leave us with several interpretive questions that, for the most part, suggest that this two-year randomized controlled trial was not the best option to test the CPOL approach to prevention. They also leave us with a better understanding of the kinds of knowledge required to better design our theories of change and conceive the studies we need. Unfortunately, the CPOL trial ended without the opportunity to conduct a much-needed qualitative assessment to understand the way this intervention (and the trial used to evaluate it) were received by the communities over time—with regard to credibility, quality, feasibility, and acceptability—and which factors may have played a role in its effects (or lack thereof) on HIV/STI levels in the community. Perhaps the CPOL model is one for which the social, epidemiological, and demographic context is critical and not amenable to the kind of control required of an RCT, and where a multi-method context-specific assessment would be more useful.

Conclusions and Recommendations

Progress in HIV prevention requires new approaches that engage with underlying social-structural drivers of patterns of practices that influence vulnerability and facilitate the spread of HIV. Patterns of behavior and practices will arise from combinations of drivers, operating in specific social, economic, and political contexts. As such, no single causal pathway can be drawn from a social driver to a set of practices or behaviors; rather, a range of potential outcomes may arise. Associations made between social drivers and HIV vulnerability should have "sociological plausibility" and should draw from extant social science and epidemiological data. Engaging with social drivers requires methods and approaches beyond traditional conceptualizations that seek to identify and intervene on single, causal determinants or universal mechanisms of influence.

Many terms, methods, and notions of evidence are contested, but action in the face of a continuing pandemic is urgent, so some compromise is required. It is important to keep interrogating what constitutes "evidence" (and who gets to decide what constitutes it), but we must avoid becoming mired in epistemological and paradigmatic combat that stymies action. Thus, both evidence-based medicine/science-based research "true believers" and pro-qualitative, "counter-science" adherents[32] must give a little. HIV prevention science—currently dominated by biomedical and clinical science paradigms—must better integrate understandings and approaches (theoretical and methodological) from the social sciences and from social epidemiology, as social science methods are valid for social-level analysis and assessment. At the same time, social scientists must not reject experimental methods where they may be appropriate and valid for testing the efficacy of new HIV prevention approaches.

HIV prevention researchers and advocates must reject and resist oversimplified language for social drivers. Statements that particular social-structural factors "do" or "do not" lead to HIV transmission are almost always too simplistic; language should shift to discussing how, in what circumstances, and for whom particular combinations of factors contribute to HIV vulnerability (or, conversely, resilience). In order to be rigorous and scientific, design of HIV prevention programs and interventions aiming to address social-structural factors must:

- Begin with an assessment of the social and structural factors that may be shaping patterns of risk behavior in targeted populations and settings;
- Identify (hypothesize) sociologically plausible causal chains between distal structural factors and specific individual or group practices;
- Identify levels of possible influence, in line with the HIV prevention program's or intervention's scope and aim;
- Make explicit any assumptions about such influences and aims, including potential expected and unexpected consequences of the program or intervention; and
- Build in evaluation mechanisms appropriate to the aim, level, scope, and method of the program or intervention as a way to enable validation of assumptions, investigation of the mechanisms by which structures affect risk and vulnerability, and appropriate assessment of outcomes and impact.

This is an important moment—and opportunity—in the history of the HIV/AIDS pandemic, as there is (finally) a much clearer and broader recognition of the need to incorporate social science approaches more fully into the global response. We hope that this paper contributes to the possibility of seizing that opportunity, with the ultimate goal of preventing new HIV infections and improving the lives and well-being of people worldwide.

Footnotes

1. We use the term "practices" to convey the social dimension of the actions that usually are implicated as "risk behaviors." Practices are socially produced behaviors that are organized and patterned by culture (Kippax 2008,[57] 2003[58]). Sexual or drug injection behaviors that transmit HIV infection are practices that are socially produced; that is, they are patterned by social norms and the meanings people give to their actions. As Kippax notes, "These meanings are formed in the relations between people with reference to the interpersonal contexts and networks in which they are enacted and in response to prevailing socio-cultural, economic, and political structures" (2008: 490). Sexual practices, for example, are influenced by prevailing norms and structures related to gender, love, intimacy, sexuality, pleasure, fertility, etc., and, thus, vary by setting and situation.

2. For reviews of the literature on mixed and triangulated methods, see Saukko (2003[59]) and Teddlie and Tashakkori (2003[60]).

3. A special issue of the journal AIDS was dedicated to a description of the trial design (AIDS 2007; 21[Supplement 2]).

References

1 Solomon S, Venkatesh K. Structural interventions in social contexts. Pp. 550–70 in Mayer K, Pizer H (eds.), HIV Prevention: A Comprehensive Approach. London: Academic Press/Elsevier. 2008.

2 Gupta G, Parkhurst J, Ogden J, Aggleton P, Mahal A. Structural approaches to HIV prevention. Lancet 2008; August: 52–63.

3 Blankenship K, Friedman S, Dworkin S, Mandel J. Structural interventions: concepts, challenges and opportunities for research. Journal of Urban Health 2006; 83: 59–72.

4 UNAIDS. Practical guidelines for intensifying HIV prevention: towards universal access. Geneva: UNAIDS. 2007.

5 Giddens A. Sociology, 4th edition. Oxford: Blackwell Publishing. 2001.

6 Blankenship K, Bray S, Merson M. Structural interventions in public health. AIDS 2000; 14 (Supplement 1): S11–S21.

7 Krishnan S, Dunbar M, Minnis A, Medlin C, Gerdts C, Padian N. Poverty, gender inequities and women's risk of HIV/AIDS. Ann NY Acad Sci 2008; 1136: 101–10.

8 Obermeyer C. HIV in the Middle East. BMJ 2006; 333: 851–54.

9 Gilles P, Tolley K, Wolstenholme J. Is AIDS a disease of poverty? AIDS Care 1996; 8: 351–63.

10 Hallman K. Gendered socioeconomic conditions and HIV risk behaviours among young people in South Africa. African Journal of AIDS Research 2005; 4: 37–50.

11 Rwenge M. Poverty and sexual risk behaviour among young people in Bamenda, Cameroon. African Population Studies 2003; 18: 91–104.

12 Weiser S, Leiter K, Bangsberg D, et al. Food insufficiency is associated with high-risk sexual behavior among women in Botswana and Swaziland. PLoS Medicine 2007; 4: 1589–98.

13 Shelton J, Cassell M, Adetunji J. Is poverty or wealth at the root of HIV? Lancet 2005; 366: 1057–58.

14 Chin J. The AIDS Pandemic: The Collision of Epidemiology with Political Correctness. Oxford: Radcliffe Publishing. 2007.

15 Mishra V, Bignami-Van Assche S, Greener R, et al. HIV infection does not disproportionately affect the poorer in sub-Saharan Africa. AIDS 2007; 21 (Supplement 7): S17–S28.

16 Krieger N. Theories for social epidemiology in the 21st century: an ecosocial perspective. Int J Epidemiol 2001; 30: 668–77.

17 Popay J, Williams G, Thomas C, Gatrell A. Theorising inequalities in health: the place of lay knowledge. In Bartley M, Blane D, Davey Smith G (eds.), The Sociology of Health Inequalities. Oxford: Blackwell Publishers. 1998.

18 Somers M. The narrative constitution of identity: a relational and network approach. Theory and Society 1994; 23: 605–649.

19 Dicker R. Analyzing and interpreting data. Pp. 132–72 in Gregg M (ed.), Field Epidemiology, 2nd Edition. Oxford: Oxford University Press. 2002.

20 Gehlbach S. Interpreting the Medical Literature, 5th Edition. New York: McGraw-Hill. 2006.

21 Hoffer L. Complementary or alternative medicine: the need for plausibility. CMAJ 2003; 168: 180–82.

22 Sumartojo E. Structural factors in HIV prevention: concepts, examples, and implications for research. AIDS 2000; 14 (Supplement 1): S3–S10.

23 Cohen D, Scribner R, Farley T. A structural model of health behavior: a pragmatic approach to explain and influence health behaviors at the population level. Prev Med 2000; 30: 146–54.

24 Auerbach, J. Key issues in HIV prevention research. Invited presentation. Kaiser Family Foundation International Health Journalism Fellowship Program briefing session in advance of the XVII International AIDS Conference, Mexico City, August 1, 2008.

25 STOP AIDS Project. Understanding, formulating, and implementing structural interventions. Unpublished draft. 2009.

26 Auerbach J, Smith W. Confronting the “evidence” in evidence-based HIV prevention: current scientific and political challenges. BETA: Bulletin of Experimental Treatments for AIDS 2008; 20: 44–50.

27 Coates T, Richter L, Cáceres C. Behavioural strategies to reduce HIV transmission: how to make them work better. Lancet 2008; August: 36–51.

28 Geronimus A. Addressing structural influences on the health of urban populations. Am J Public Health 2000; 90: 867–72.

29 Campbell C, Williams B. Beyond the biomedical and behavioural: towards an integrated approach to HIV prevention in the southern African mining community. Social Science & Medicine 1999; 48: 1625–39.

30 Raphael D, Bryant T. The limitations of population health as a model for a new public health. Health Promotion International 2002; 17: 189–99.

31 Mykhalovskiy E, Weir L. The problem of evidence-based medicine: directions for social science. Social Science & Medicine 2004; 59: 1059–69.

32 Denzin N. The elephant in the living room: or extending the conversation about the politics of evidence. Qualitative Research 2009; 9: 139–60.

33 Black N. Why we need observational studies to evaluate the effectiveness of health care. BMJ 1996; 312: 1215–18.

34 Pawson R, Tilley N. Realistic Evaluation. London: Sage Publications. 1997.

35 Victoria C, Habicht J-P, Bryce J. Evidence-based public health: moving beyond randomized trials. Am J Public Health 2004; 94: 400–405.

36 Pawson R, Greenhalgh T, Harvey G, Walshe K. Realist review—a new method of systematic review designed for complex policy interventions. Journal of Health Services Research & Policy 2005; 10 (Supplement 1): 21–34.

37 Sanson-Fisher R, Bonevski B, Green L, D'Este C. Limitations of the randomized controlled trial in evaluating population-based health interventions. American Journal of Preventive Medicine 2007; 33: 155–60.

38 Cartwright N. Are RCTs the gold standard? Biosocieties 2007; 2: 11–20.

39 Deaton A. Instruments of development: randomization in the tropics, and the search for the elusive keys to economic development. Princeton, Center for Health and Wellbeing, Princeton University. http://www.princeton.edu/~deaton/downloads/Instruments_of_Development.pdf (accessed May 15, 2009).

40 Institute of Medicine Committee on the Social and Behavioral Science Base for HIV/AIDS Prevention and Intervention. Assessing the social and behavioral science base for HIV/AIDS prevention and intervention: workshop summary. Washington, DC: National Academy Press. 1995.

41 California HIV/AIDS Research Program. Establishing evidence for social & behavioral HIV prevention. Unpublished meeting summary. 2008.

42 Ross D, Wight D, Dowsett G, Buve A, Obasi A. The weight of evidence: a method for assessing the strength of evidence on the effectiveness of HIV prevention interventions among young people. Pp. 79–102 in Ross D, Dick B, Ferguson J (eds.), Preventing HIV/AIDS in Young People: A Systematic Review of the Evidence from Developing Countries. UNAIDS Interagency Task Team on HIV and Young People. WHO Technical Report Series, no. 938. 2006.

43 Rutherford G. Beyond T2: implementing effective interventions and measuring their impact. Presentation at 10th Annual CFAR Research Symposium: Biomedical Approaches to HIV/AIDS Prevention, University of California, San Francisco, February 24, 2009.

44 Kirby D. Changes in sexual behaviour leading to the decline in the prevalence of HIV in Uganda: confirmation from multiple sources of evidence. Sexually Transmitted Infections 2008; 84 (Supplement 2): ii35–ii41.

45 Pronyk P, Hargreaves J, Kim J, et al. Effect of a structural intervention for the prevention of intimate partner violence and HIV in rural South Africa: a cluster randomised trial. Lancet 2006; 368: 1973–83.

46 Center for Interdisciplinary Research on AIDS. Can microfinance programs reduce HIV risk in developing countries? Policy Update 2007; Spring. http://cira.med.yale.edu/law_policy_ethics/mcrofin_global.pdf (accessed May 15, 2009).

47 Stratford D, Mizuno Y, Williams K, Courtenay-Quirk C, O'Leary A. Addressing poverty as risk for disease: recommendations from CDC's consultation on microenterprise as HIV prevention. Public Health Reports 2008; 123: 9–20.

48 Dunbar M, Maternowska C, Kang M-S, et al. Findings from SHAZ!: a feasibility study of a microcredit and life-skills HIV prevention intervention to reduce risk among adolescent female orphans in Zimbabwe. Journal of Prevention and Intervention in the Community. In press.

49 Central Intelligence Agency. World Factbook 2009. Washington, DC: Central Intelligence Agency. https://www.cia.gov/library/publications/download/ (accessed May 15, 2009).

50 Stoneburner R, Low-Beer D. Population-level HIV declines and behavioral risk avoidance in Uganda. Science 2004; 304: 714–18.

51 Auerbach J, Hayes R, Kandathil S. Overview of effective and promising interventions to prevent HIV infection. Pp. 43–78 in Ross D, Dick B, Ferguson J (eds.), Preventing HIV/AIDS in Young People: A Systematic Review of the Evidence from Developing Countries. UNAIDS Interagency Task Team on HIV and Young People. WHO Technical Report Series, no. 938. 2006.

52 Kelly J, Murphy D, Sikkema K, et al. Randomized, controlled, community-level HIV prevention intervention for sexual risk behaviour among homosexual men in U.S. cities. Lancet 1997; 350: 1500–1505.

53 Jones K, Gray P, Whiteside Y, et al. Evaluation of an HIV prevention intervention adapted for Black men who have sex with men. Am J Public Health 2008; 98: 1043–50.

54 Sikkema K, Kelly J, Winett R, et al. Outcomes of a randomized community-level HIV prevention intervention for women living in 18 low-income housing developments. Am J Public Health 2000; 90: 57–63.

55 Rogers E. Diffusion of Innovations, 4th Edition. New York: Free Press. 1995.

56 Cáceres C, Konda K, Salazar X, et al. NIMH collaborative HIV/STI prevention trial: new populations at high risk for HIV/STI in low-income, urban coastal Peru. AIDS and Behavior 2008; 12: 544–51.

57 Kippax S. Understanding and integrating the structural and biomedical determinants of HIV infection: a way forward for prevention. Current Opinion in HIV and AIDS 2008; 3: 489–94.

58 Kippax S. Sexual health interventions are unsuitable for experimental evaluation. Pp. 17–34 in Stephenson J, Imrie J, Bonell C (eds.), Effective Sexual Health Interventions: Issues in Experimental Evaluation. Oxford: Oxford University Press. 2003.

59 Saukko P. Doing Research in Cultural Studies: An Introduction to Classical and New Methodological Approaches. London: Sage Publications. 2003.

60 Teddlie C, Tashakkori A. Major issues and controversies in the use of mixed methods in the social and behavioural sciences. Pp. 3–50 in Tashakkori A, Teddlie C (eds.), Handbook of Mixed-Methods in Social and Behavioral Research. Thousand Oaks, CA: Sage Publications. 2003.

Acknowledgements

The authors would like to thank Geeta Rao Gupta, David Bell, Rob Goble, and Jessica Ogden for their very thoughtful reviews and suggestions about the paper, and Reilly O'Neal for her excellent copy-editing of the document.