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Social and behavioural research prospects for sexually transmissible infection prevention in the era of advances in biomedical approaches

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Abstract

In the past two decades, major advances in biomedical intervention approaches to prevent HIV and many sexually transmissible infections (STIs) have shown great promise. However, challenges to prevention remain in the area of achieving population-level impact for biomedical prevention approaches. In this paper we address what social and behavioural research approaches can contribute beyond well-known behaviour change and counselling interventions. We organise work into five areas. Adherence and disinhibition research is primarily into individual-level constructs pertaining to maximising intervention effectiveness. Coverage research represents a population-level construct germane to maximising efficient prioritisation for prevention. Research covering social determinants, a second population-level construct, contributes to both prioritisation and effectiveness. Finally, disparities and social inequities need to be incorporated into prevention, given the pervasive and persistent disparities found in rates of HIV and STIs and in their antecedents.

Additional keywords

adherence; disinhibition; disparities; intervention coverage; social determinants

Introduction

Sexually transmitted infection (STI) prevention still faces many challenges, including antimicrobial resistance and substantial prevalence disparities across the globe.[1,2] The World Health Organization estimated that there were 357 million cases of curable STI per year worldwide as of 2012, approximately 1 million per day.[3] Syphilis and its sequelae remain a significant global burden, and gonorrhoea rates, stable or otherwise, require extra attention due to the prospects for resistance to current treatments.[4–7] In this review we examine social and behavioural aspects of STI prevention in the context of current advances in biomedical HIV and STI prevention.

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Conflicts of interest

The authors declare no conflicts of interest.

Decades of social and behavioural research and programmatic experience have yielded a substantial portfolio of individual-level interventions to prevent and control STI prevalence and sequelae, including among vulnerable or at-risk populations.[8–12] However, two related factors have affected recent prospects for successful STI prevention, namely successes in biomedical interventions that: (1) prevent HIV or selected STIs, such as human papillomavirus (HPV); or (2) ensure contraception without condoms. Randomised trials of voluntary male circumcision (VMC) have shown substantial protective efficacy for men with regard to HIV acquisition, as has pre-exposure prophylaxis (PrEP) and antiretroviral treatment (ART) for discordant couples.[13–17] These and other biomedical advances are sometimes shown to be efficacious against viral STIs (e.g. HPV vaccine and herpes simplex virus type 2 via vaginal applications of tenofovir gel).[18,19]

Against the backdrop of advances in biomedical approaches to contraception and the prevention of HIV and viral STIs, social and behavioural approaches can contribute essential value in five areas. Two are construed primarily at the individual level: adherence and disinhibition. Another two are principally population-level constructs: coverage/prioritisation and social determinants. In this paper we address disparities among subpopulations (Fig. 1; Table 1) and, for each of these areas, describe relevant observational and intervention research specific to that area in light of recent biomedical advances. We conclude with a summary of research prospects.

Individual-level constructs

Adherence

Inadequate adherence can degrade the efficacy of biomedical interventions such as screening, diagnosis and treatment for infected people, as well as preventive interventions such as vaccination, condom use, PrEP and long-acting reversible contraception (LARC). When adherence is inadequate, individuals become or remain vulnerable to STI acquisition or transmission, which then further transfers vulnerability to members of their sexual networks. Moreover, the risk for pathogen resistance to medication may increase. In trials of PrEP, those with low adherence (<40%) had no effect on HIV acquisition rates,[20,21] whereas those with higher adherence (≥80%) showed reductions in HIV acquisition.[15] For microbicides, most studies appear to have had insufficient adherence to demonstrate an effect; however, in the Centre for the AIDS Program of Research in South Africa 004 (CAPRISA) trial, the protective effect of the gel was greater when adherence was high.[22]

Medication qualities, such as pill size, and method of administration (e.g. oral, injected, vaginal application) affect adherence. STIs treated on the spot in clinical settings overcome some of these issues because many treatments are single dose and can be administered at the time of diagnosis or as prophylaxis. For HIV, topical microbicides and ART require more extensive adherence because they require action from individuals on an ongoing basis (in contrast to, say, male circumcision, which requires adherence to post-circumcision instructions for a relatively brief time).

Beliefs and fears about side effects (e.g. fatigue, taste disturbances) also affect adherence, as do beliefs about safety (as opposed to actual safety risks).[23,24] A systematic review of

European populations' perceptions and concerns about the HPV vaccine found vaccine safety as the first concern overall, including long-term adverse consequences for HPV and fears of sexual promiscuity subsequent to vaccination.[25] A similar range of fears has been shown in systematic reviews of US studies and of studies conducted across 10 African nations.[26,27] Medical professionals' concerns about the HPV vaccine present another barrier to vaccination.[28] Thus, interventions to reduce vaccine hesitancy need to include healthcare providers and young adolescents and their caregivers.

Stigma affects adherence as a direct stressor upon stigmatised individuals and as a contributor to structural inequities.[29–31] For HIV, several studies have demonstrated that stigma, including internalised stigma, affects adherence to ART and remaining in care more generally.[32–34] Stigma is also compounded when the stigma attached to an STI *per se* (e.g. gonorrhoea is 'morally suspect' in a way that a sports injury is not) and generically to people infected with an STI (e.g. gonorrhoea is a marker for poor individual character) is correlated with the perception that HIV and STI rates are disproportionately high among members of minority groups. The reciprocal reinforcement whereby STI stigma reinforces general stigmatisation of minorities, which, in turn, reinforces stigma attached to STI is an example of a contributor to 'minority stress'.[35,36]

For many people, mere awareness of infection leads to behaviour change without substantial intervention. During a counselling randomized control trial (RCT) enrolling people with acute HIV infection in Malawi, multiple sexual partnerships and condomless sex decreased regardless of the intensity of counselling at the point of diagnosis.[37] Yet, patterns of adherence to prevention are complex. In one analysis of correlates of adherence from a microbicide trial, perceptions of partner riskiness were associated with adherence in bivariate analyses, whereas in multivariate analyses partner age was positively associated and an increasing number of vaginal sex episodes was inversely associated with adherence (i.e. the odds of a woman adhering to microbicide use while having sex three or more times a week were 6% the odds of a woman who had sex once a week).[38] For condom use, a systematic review based on data from 12 African countries revealed that trust between partners reduces condom use in that perceptions of risk decrease as feelings of trust grow.[39] Finally, risk perception patterns for STI can persist in the face of other prevention. Among a Colorado cohort of female adolescents and young women (aged 13–24 years) who initiated either LARC or short-acting reversible contraception showed that those who acquired a new partner over 6 months increased their condom use.[40]

Efforts to increase user acceptability support adherence to biomedical interventions. New microbicide design is aimed at making the microbicides easier to insert and less noticeable to the user when inserted.[41] However, this approach contains its own threat to efficacy in that simply diminishing awareness of a user-mediated intervention may increase the odds of forgetting to use it (intervention efficacy and ease of use may be an inverted U-shaped function rather than linear). Consequently, behavioural research aimed at increasing adherence to medication regimens and prevention protocols (e.g. retesting after diagnosis and treatment of an STI) include cognitive approaches, such as reminder systems and reinforcing skills and habits through interactive interventions.[42,43] One recent RCT using an electronic platform to deliver risk management and HIV and STI testing behavioural

interventions demonstrated a 40% reduction in STI incidence over 12 months.[43] To combat stigma and encourage adherence, researchers have promoted provider–patient relationship quality, including through communication for HPV vaccination.[44] Social support in networks is correlated with adherence,[45] and a recent RCT showed that eliciting and reinforcing networks of care and social support increase retention in care among young men who have sex with men (MSM).[46]

Another approach to increasing acceptability with more overt behavioural components is addressed by framing interventions in terms of what the potential recipients of intervention already value and in venues they are likely to frequent (both part of communications and social marketing). For example, the GYT: Get Yourself Tested campaign in the US presented STI testing for adolescents and young adults to that population as a normal part of looking after their own health and as something to discuss with friends and partners.[47] Evaluation data showed exposure to the campaign was correlated with STI testing in multiple settings.[47,48] Social and behavioural research to support the HPV vaccine is often focused on increasing parent acceptance by remediating parents' fears for the effect of the vaccine on their children's health and prospective sexual behaviour.[25,26]

Risk compensation and disinhibition

Even high levels of adherence can be compromised by disinhibition and risk compensation, two terms that overlap in meaning and that have often been used interchangeably. With disinhibition, we refer to lack of restraint or self-regulation, characterised by impulsive behaviour and frequent disregard for social conventions. To explain disinhibition, Hirsh *et al.*[49] posited that intoxication, anonymity and social power all constrain activation of the brain's inhibitory mechanism in the hippocampal system (see Gray[50] for details on the brain's inhibitory system) and thus explain how these conditions are connected to behaviours that predict STI acquisition or transmission. Alcohol and the use of other drugs are correlates of risky sexual behaviours and cluster with other risks to health.[51] Situations that confer high levels of anonymity (e.g. music and other festivals) are associated with reports of unwanted sexual touching and assault.[52,53] A recent review synthesised randomised experiments and concluded alcohol use contributed causally to increased sexual risk decision making, both directly via expectancy and arousal and indirectly through delayed condom availability.[54] However, lack of protective action for oneself or others can also be caused by perceptions of being unable to influence one's future at all,[55] implying that the disinhibiting effect of social power is another U-shaped function, with increased disinhibition at both ends of the spectrum of social power. Moreover, lack of restraint and self-regulation, along with lack of empathy, can be influenced by social norms and expectations (e.g. the interaction between norms about sexual behaviours and gender).[56] These factors can also be influenced by personality traits, which are difficult to modify (and typically outside the scope of STI prevention).

We characterise risk compensation here as increases in risky behaviour as a function of reduced perceptions of risk due to prevention, whether or not these perceptions are accurate.[57,58] We also consider that the 'compensation' may actually affect risks not related to individuals' risk perceptions or even the goal of a given prevention intervention. For

example, reduced condom use among women using LARC should not affect conception rates, but may increase STI acquisition and transmission risk. Although risk compensation is not always a substantial risk at the population level (e.g. VMC did not appear to be correlated with greater subsequent risk across population surveys in sub-Saharan countries),[59] it appears present for at least subsets of vulnerable populations, such as MSM taking PrEP.[60] For risk compensation, some have suggested intervening to alter the amount of HIV or STI risk that people will tolerate: their 'risk equilibrium'.[61] A related approach is to identify long-term goals that resonate with the target populations, such as having children, ensuring a mutually supportive long-term relationship, taking the responsibility not to harm others; reviews show this approach facilitates adherence to prevention, including condom use.[39] Yet more nuanced interventions could test STI prevention efficacy for condom use matched to existing behavioural preferences and patterns and the highest-risk situations (new sex partners, non-monogamous sex partners, recent STI history).

A reason not to conflate risk compensation and disinhibition is that interventions designed to address the effects of disinhibition may not be as effective against the effects of risk compensation and vice versa. However, at least some approaches to reducing risk compensation are also largely relevant to mitigating disinhibition. In a Ghanaian study in which women receiving tenofovir (as prevention) also increased condom use (especially those at highest risk), qualitative research revealed that some women attributed trial participation as a reason to use condoms.[62] A cohort study in Uganda reported a 70% reduction in sexual risk behaviours after initiation of ART.[63] These results could indicate a shift in how much risk participants tolerated (i.e. their risk equilibrium)[61] and reductions in their sense of helplessness.

Cognitive behavioural interventions can play an important role in mitigating the effects of disinhibition. Impulsivity and self-control are associated with HIV and STI risk behaviours.[64,65] A systematic review of existing behavioural interventions targeting alcohol use among people living with HIV showed that interventions can reduce alcohol use and improve adherence,[66] so supportive interventions to increase self-regulation skills in conjunction with biomedical prevention may help mitigate disinhibition via intoxication or anonymity. Another potential avenue is to intervene to channel social power into prosocial action. A recent study from China showed that STI testing uptake among men who were offered free testing was much greater (54% vs 6%) if offered a chance to donate funds to support others' testing.[67] Finally, we note that HIV and STI stigma provide an incentive for anonymity, associated with disinhibition. Therefore, efforts to reduce stigma (and thereby increase social power) could attenuate disinhibition by reducing the incentive for anonymity, but also accentuate it via increases in social power. The likelihood that both very low and very high social power contribute to disinhibition adds complexity to this as yet untested hypothesis.

Population-level constructs

Coverage and prioritisation

Up to this point we have discussed social and behavioural aspects of STI prevention at the individual level. We now move to the population level. To achieve population impact, interventions must maximise the product of intervention efficacy and intervention scale

among populations that contribute directly or indirectly to transmission.[68] Moreover, interventions need to achieve sufficient scale rapidly enough to outpace infection spread (i.e. achieve a favourable 'prevalence dynamic').[67] Context for such efforts to scale-up interventions include cost and policy constraints, neither of which is likely to be static over time.[69,70]

Social and behavioural research germane to coverage and prioritisation includes efforts to uncover key populations and social structure.[71–73] With regard to identifying key populations, many case report surveillance systems[4] collect demographic and behaviour measurements among people diagnosed with STI, including HIV. Nationally representative surveys that collect behavioural data also have data on uninfected people and therefore provide 'denominators', useful for point estimates, trends and using behavioural surveillance to guide intervention coverage. For example, the US National Survey of Family Growth (NSFG) data have produced HIV testing estimates for young MSM and concentration of sexual behaviours across gender by levels of poverty, education, race and ethnicity, as well as trends across time.[74–76]

Hierarchical structure and social isolation (e.g. via stigma or prejudice) are key population-level attributes associated with transmission and that can be prioritised for intervention.[72] UK data show that household structure is associated with STI risk behaviour (e.g. young women living alone vs those living with parents were more likely to engage in unsafe sex).[77] In the US, racial segregation combines the effects of hierarchy and isolation: data from 257 US metropolitan areas showed that higher gonorrhoea case rates were particularly associated with isolation (the extent to which Black people in the US were unlikely to share neighbourhoods with members of other groups).[78,79] Global systematic reviews have shown that HIV rates among sex workers (a group typically isolated from other social structures and often highly stigmatised) were much higher than for women in the general population; the authors of those studies concluded that decriminalisation of sex work would avert a substantial proportion of HIV concentrated in this population.[80,81] Social structures, such as sex ratios, are also relevant: data from India's National Family and Health Survey have shown that India's sex ratio, which is biased towards males, is associated with early marriage and forced sex among women,[82] if plausibly mediated by social norms facilitating these conditions and behaviours. Finally, some evidence suggests that reduced isolation is associated with lower rates of STI risk. Adolescents in Baltimore who perceived higher levels of neighbourhood cohesion reported higher rates of condom use than adolescents who perceived their neighbourhoods as low cohesion.[83]

For STI, efficacious interventions must also be applied swiftly enough to avert transmission: delays in diagnosis, health care seeking, treatment and prevention can sabotage population-level prevention.[69,84] For example, HIV care continua are associated with timeliness goals (e.g. time from diagnosis to entry into care).[85] Timeliness is also a factor for individuals: an experiment embedded in a UK national survey found that minimal delays between testing and diagnosis were important to those aged 16–24 years.[86] The Person-Time of Infectiousness model categorises timeliness through delays in time to health care seeking, diagnosis, treatment and prevention.[84] Point-of-care tests can reduce delays in health care seeking (faster testing) and diagnosis: social and behavioural research supporting such

efforts in Australia include assessments of acceptability and capacity to manage test results in a short period of time.[87] Interventions in this area can have complex effects: in one RCT of Internet-facilitated STI testing, a gain in time to testing resulted in no difference in time to treatment.[88] The intervention group's extra time to seek care negated the gain in testing almost exactly, although overall uptake also increased (thus, an overall benefit to coverage).

Social determinants of health

Addressing coverage shows that social and behavioural science must incorporate variables that are characteristics of populations and social structure. Many existing frameworks link social determinants, behaviours and demographics to health conditions,[89,90] with some models specifically oriented to the areas of HIV, contraception, STI prevention or sexual health.[91–94] Empirical work drawing from these models and including a focus on STI, HIV and contraception includes broad analyses of STI and concepts such as social cohesion and economic status, poverty, education (attainment and access) and incarceration.[95–99] Data from the American Community Survey combined with gonorrhoea reports from Centers for Disease Control and Prevention (CDC) surveillance showed that factors such as population density and proportion of the population unemployed or below the poverty line were associated with gonorrhoea rates.[95] Rates of eviction from rental housing and rates of STI in the US are correlated at the county level.[100] Regarding incarceration, US National HIV Behavioural Surveillance data showed incarceration history and STI diagnoses were associated among women,[98] and a retrospective cohort study of over 200 000 people showed elevated risk for STI among people in their first year after release.[101]

Other studies use more specific constructs, such as neighbourhood-level characteristics and HIV and STI.[102–105] For example, one study of sex partner meeting places showed that core transmitters were more likely to be found in venues with drug and sex 'market' activity.[102] Another study examining social support and cohesion among city neighbourhoods in the US found that informal social control at the neighbourhood level was protective for young women, but unrelated for young men; in contrast, individual social support was associated with a large increase in the odds of having an STI, but only for young men.[103] The authors of that study speculated that these counterintuitive findings may be based on the connections of young men with social support (e.g. to high-risk social networks in which norms supported risk, not protective behaviours).

Structural interventions that directly or indirectly reduce STI risk support biomedical prevention by either facilitating access to prevention or reducing the need for it by mitigating the circumstances that produce risk.[106] In New York City, an evaluation of an effort to provide affordable housing to youth emerging from foster care showed that youth who were in the housing program (with health access support) had under one-third the rate of STI diagnosis than youth not in the program.[107] In India, ongoing interventions on social determinants of health are aimed at reducing HIV risk through supporting girls in low castes entering and staying in secondary education: the mediating factor is delaying the age of marriage and reducing the proportion entering into sex work.[108] Another study showed that promoting gender equity messages in a low-income and predominantly Muslim community in Mumbai could produce greater norms of equity, including among leaders of important

community organisations.[109] Messages and selection of dissemination channels (e.g. imams in area mosques) were based on extensive formative research to assess prior community attitudes and to find community organisations that could, together, reach the entire population.

Disparities and social inequities

Global disparities in STI are pervasive.[1,3] In the US, an analysis of Healthy People 2010 objectives placed HIV, AIDS, gonorrhoea (overall and among 15- to 44-year-old females) and syphilis (primary, secondary and congenital) as six of the top 10 largest health disparities.[110] Data typically reveal substantial disparities by racial or ethnic identification, sexual orientation, age, gender, measures of income level and various other sociodemographic variables.[111] Disparities are sometimes exacerbated for people who fall into multiple minority categories (i.e. intersectionality),[112] although these relationships are often complex. Although STI rate differences measured by some of these variables incorporate underlying biological differences in vulnerability to STI and HIV acquisition or transmission (e.g. heterosexual transmission risks),[113] disparities are, at best, incompletely explained by biology.

Disparities can be affected by any of the concepts we have described thus far; we suggest inequities in the distribution of social determinants are the most salient factors (sometimes mediated by individual-level concepts). Behavioural differences certainly fail to explain sociodemographic disparities (they often help explain differences within groups). Hallfors *et al.* organised respondents from the National Longitudinal Survey of Adolescent to Adult Health by 15 categories of sexual and drug use behaviour with varying levels of established HIV and STI risk (e.g. few sex partners and low alcohol and other drug use, multiple partners and use of multiple drugs).[114] In that study, Black respondents had substantially higher rates than White respondents of HIV and STIs in every one of the 15 categories, and the largest disparities existed at the lowest levels of risk. This finding means that ‘low-risk’ behaviours were much more protective for White respondents than for Black respondents. More recently, analyses from the National Health and Nutrition Examination Survey showed that the number of partners was unrelated to chlamydial prevalence among female Black participants, although prevalence was lower for those with fewer partners among the remaining women in the sample.[115] Thus, not only is a disparity in group prevalence a risk in its own right, it degrades the effectiveness of preventive intervention and requires increasingly potent combinations of prevention intervention to overcome. Analogous data demonstrate that: (1) US Black MSM at all levels of behavioural risk have higher rates of HIV than do other MSM; (2) HIV prevalence differs by educational level among pregnant women in Brazil; and (3) inter-related combinations of race and economic deprivation are associated with STI incidence in the UK.[116–118]

Disparities are also visible in the social determinants of STI and unintended pregnancy, including categories composed of variables measured at: (1) the individual or household level (e.g. race, ethnicity, sexual orientation, income, age, education and employment status); and (2) an area level (e.g. health care access and provision, poverty and segregation). The two categories are related in that area-level disparities often exacerbate individual

disparities. For example, in both India and Kenya, individuals' coping strategies for low government spending on sexual and reproductive health services are shown to be highly regressive: the poorest people spend a considerably higher proportion of income on health management and have to engage in riskier financial strategies, such as high-interest loans.[119] Segregation and caste-based data discussed in previous sections also indicate substantial racial disparities.[78,108] When segregation is absent, disparities diminish. In a cohort study of a racially integrated Baltimore community with similar socioeconomic status by race, health outcomes (e.g. obesity) were also similar across race.[120]

Social and behavioural research can also reveal the role and combinations of social divisions, prejudices and discrimination (e.g. racism, classism and intersectionality), and inform interventions.[120–124] For interventions, this translates to 'achieving health equity by design', building interventions specifically to incorporate and manage the 'first causes' of differential health care provision, access and use.[125] Thus, interventions conducted at a scale designed to produce population effect, whether or not they explicitly incorporate social determinants or are even designed to reduce disparities, are likely to have to address the effects of racism rather than control for the effects of race during analyses. Analogous work has been done in Europe; in both the US and Europe, the role of affected communities in intervention partnerships is emphasised.[123,126] As the relative dearth of intervention examples in this section suggests, reducing disparities in STI rates is hard to do. Large-scale demonstrations and models for reducing health disparities are more often built with chronic disease as outcomes,[127] and the movement of infection through transmission networks greatly complicates such models.[71] Finally, the examples in this section also show that disparities are not just an outcome. They can be a contributor to lack of adherence, disinhibition and determinants like segregation. For example, a caste- or class-based disparity in HIV could fuel learned helplessness via public stigma and prejudice. Reciprocal, self-reinforcing relationships can be difficult to define and measure in public health, but need to be addressed in prevention.

Conclusions

The five areas we have discussed indicate some broad considerations for social and behavioural research. Under coverage, we suggest using surveillance and population-level epidemiological analyses to both derive social structures and uncover key populations (on an ongoing basis) deserves even greater emphasis than these approaches now have. This work also helps allocate resources such that most at-need groups receive the highest-intensity interventions. Methodologically, non-linear outcomes appeared plausible in several instances (e.g. the potential U-shaped function for adherence based on ease of intervention use). Different modelling approaches (e.g. mathematical modelling or structural equation modelling) help test such complex outcomes. More broadly, attention to non-linearity needs to be incorporated into conceptual thinking, as with the role of disparities as both cause and effect. Integrating prevention approaches is not a new idea; we suggest this point includes embedding many prevention efforts in the context of social determinants and measuring how remediating social determinants affects STI and HIV prevention, and perhaps even vice versa. Finally, we acknowledge much of our review has focused on interventions to manage

and mitigate risk. Alternative framing around principles like positive youth development[128] and sexual health can result in effective STI and HIV prevention.[8]

To this end, we conclude with a broad roadmap for applying social and behavioural research to support STI prevention (Fig. 2). The factors and strategies are drawn from the content in this paper. The precise combination of supportive prevention strategies will depend on the population mix, the STI(s) in question, the biomedical prevention context and the stage of epidemic.[129] We hope that this paper has illustrated how social and behavioural research is most valuable to STI prevention: integrated with biomedical HIV and STI prevention, incorporating social context and integrated with overall prevention. It is surely the first step in integrating all our efforts to address the full scope and scale of STI preventions.

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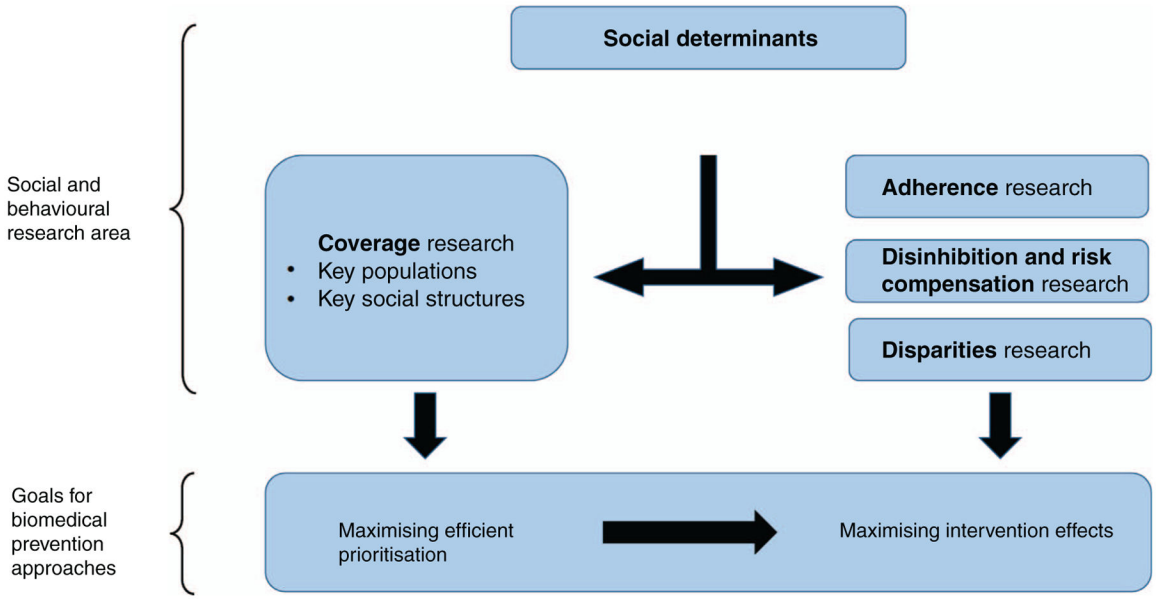

Fig. 1.

Relationship between social and behavioural research and biomedical prevention for sexually transmissible infection (STI), HIV and contraception. The top portion of this schematic identifies the five key areas of social and behavioural research covered in this paper. Biomedical prevention approaches include, but are not limited to, pre-exposure prophylaxis and antiretroviral treatment, long-acting reversible contraception, screening, testing and treatment.

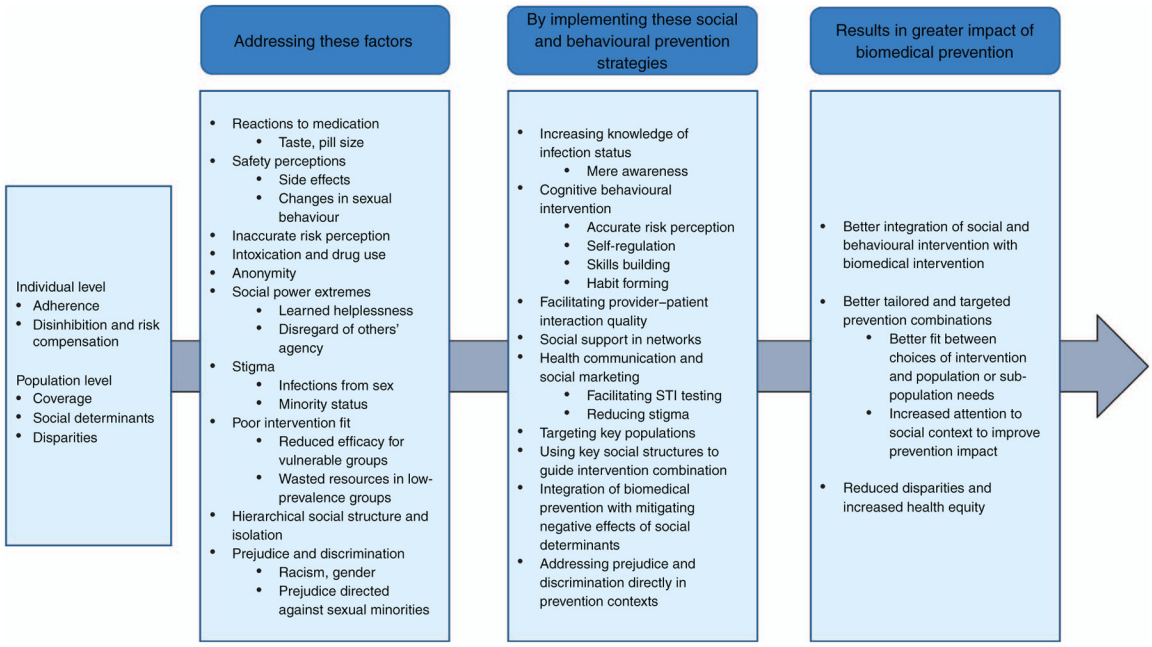

Fig. 2.

A broad roadmap for matching social and behavioural prevention research with factors inhibiting prevention impact for biomedical strategies. Factors and strategies in the second and third columns are drawn from the research covered in this paper. Biomedical prevention approaches include, but are not limited to, pre-exposure prophylaxis and antiretroviral treatment, long-acting reversible contraception, screening, testing and treatment. Estimating the mix of intervention strategies will vary according to sexually transmissible infection (STI) and HIV epidemiology and the mix of demographic and social determinants and disparities in populations.

Table 1.

Five categories of social and behavioural research as related to sexually transmissible infection (STI) prevention and care

	Definition
Individual Level	
Adherence to prevention	Fidelity (or lack of) to prevention protocols, including primary prevention, treatment and post-treatment prevention
Disinhibition and risk compensation	Disregard of risk or consequences due to intoxication, anonymity or extremes of social power or influence (disinhibition)
	Engagement in one risk after reduced susceptibility or severity of another risk (risk compensation)
Population level	
Coverage and prioritisation	Factors related to identifying key populations and aspects of social structure relevant to STI transmission dynamics to ascertain priorities for prevention
Social determinants of health	Characteristics of social structure (within or across societies) that affect access to and provision of prevention and health care
Disparities	Differences in rates or vulnerability to STI or sequelae, including differences in access to or provision of prevention and health care, as a function of sociodemographic characteristics or social position