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Disparities in HIV/AIDS in black men who have sex with men

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The devastating effect of the HIV epidemic in Canada, the UK, and the USA is nowhere more evident than in men who have sex with men (MSM), and, particularly, in young black MSM. Distressing—but not well recognised—is that these ethnic disparities were present in the USA as early as 1984–89.¹ Similar patterns were present throughout the 1990s and remain today.^{2,3} Exacerbating these disparities is evidence of early unequal access by ethnicity to new antiretroviral treatments,⁴ contributing to the classic pattern of a growing social disparity with preferential uptake of interventions in more powerful social groups.⁵

In *The Lancet*, Gregorio A Millett and colleagues⁶ identify factors that are important in explaining the differential effects of HIV on black MSM, specifically sexual networks and partnering patterns (black and older partners), social structural barriers (low income, low education, lifetime incarceration, and unemployment) and barriers to health-care system accessibility and use in HIV-positive men.⁶ Based on a meta-analysis of studies done in the UK, Canada, and the USA, they found that black MSM reported fewer sexual risk behaviours than other MSM. However, black MSM were more likely to be infected with HIV in the UK (odds ratio [OR] 1.86, 95% CI 1.58–2.18) and USA (3.00, 2.06–4.40), and to have a diagnosis of sexually transmitted infection (UK 2.66, 1.53–4.64; USA 6.01, 5.39–6.72). Black MSM in the USA were more likely to report any structural barriers (2.28, 1.92–2.72) and to have black sexual partners (11.47, 6.02–21.88) than white MSM. Black MSM infected with HIV were less likely to report antiretroviral treatment than other MSM infected with HIV in the UK (0.78, 0.69–0.88) and USA (0.40, 0.26–0.62).

Millett and colleagues' synthesis provides some of the strongest evidence yet that neither sexual risk behaviour nor drug and alcohol use can explain the over-representation of black MSM in men infected with HIV. The investigators conclude that behavioural interventions focused on sex and drugs or alcohol should not be emphasised any longer; rather programmes to test and medically treat HIV in black MSM ought to be the focus. They also suggest that, based on findings about sexual partnering patterns of young black MSM with older black MSM, suppression of viral load in older HIV-positive black MSM is also an important priority.

We have much to learn about the details of how social and sexual networks are related to each other and influence HIV risk.⁷ The interventions proposed by Millett and co-workers related to networks (eg, promoting HIV testing in sex partners and encouraging HIV-positive partners to get medical care and initiate antiretroviral treatment) are important steps that could lead to lowering of community viral load and reduction of HIV incidence. These interventions rely on the implementation of many changes in health-care systems to address disparities in access. These solutions cannot ignore the role of government programmes that constrain and shape health-care provider behaviour, as well as the resistance to universal health-care coverage in the USA.

The structural factors identified by Millett and colleagues can be seen in sharpest relief in young black MSM, who experience structured risks associated with being black, young, and gay.⁸ Owing to overlapping systems of discrimination, young black MSM—

particularly men raised in urban areas—currently face unemployment levels of 30–50%.⁹ Young black men disproportionately experience strict criminal justice policies, such as stop-and-frisk and mandatory penalties associated with drug possession,¹⁰ which increase their risk of arrest, incarceration, and the cascade of negative effects that encountering the criminal-legal system has on black men. Young black MSM often grew up in urban neighbourhoods characterised by residential racial segregation and poverty, attending segregated public schools with fewer resources and lower levels of academic achievement compared with districts with lower poverty rates and lower concentrations of ethnic minorities.¹¹ Such urban neighbourhoods can be isolating, through restrictions of movement due to dangerous local environments; these environments are also typically characterised by scarce access to gay-identified space in which young black MSM might explore their emerging sexuality safely and with age-mates.¹² Here intersecting socially structured environments constitute the social and physical context in which young black MSM engage in lower levels of sexual risk behaviour than non-black MSM, yet acquire HIV more often.

How could we intervene to stem the spread of HIV in the black MSM community? Millett and colleagues⁶ provide a comprehensive list of recommended interventions to address factors driving the racial or ethnic disparities in HIV infection. To address the immediate urgency of the epidemic in black MSM, these recommended interventions must be implemented. At the same time, long-term changes are needed to address structural effects on the lives of black MSM. Structural interventions must begin at birth (and some say before) and include paid parental leave, high-quality and free infant-child care and preschool, and desegregated, high quality, equitably, and adequately resourced public schools, as well as schools and neighbourhoods accepting of gay youth. Criminal legal policy changes, such as ceasing stop-and-frisk and the arrest of young black men for low-level drug offences, can reduce lifetime arrest and incarceration rates, which act as barriers to full employment later in life.¹³ Increasing the visibility and acceptance of gay, lesbian, transgender, and bisexual people in all neighbourhoods and communities is important.



People complete paperwork before free HIV testing in Washington, DC, USA, on National Black HIV/AIDS Awareness Day, Feb 7, 2012

Mustering the political will to move government to support and enable implementation of any of these immediate and long-term interventions, let alone those involving significant policy changes, will be difficult. These difficulties are especially apparent in a political climate characterised by austerity and, at least in the USA, ongoing battles about access to and financing of health care.¹⁴ But governmental policies promise the strongest form of multilevel intervention with the greatest potential both to address the root causes of, and prevent the further widening of, the racial disparity in HIV acquisition and health-related consequences in MSM.

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How effective is citicoline for acute ischaemic stroke?

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In *The Lancet*, Antoni Dávalos and colleagues¹ report the results of the International Citicoline Trial on acUte Stroke (ICTUS) trial, which aimed to find out whether neurovascular protection and repair with citicoline would improve recovery from acute ischaemic stroke.

Although the main aim of treatment for acute ischaemic stroke is to restore brain perfusion rapidly with recombinant tissue plasminogen activator (rt-PA),² a complementary aim is to protect surviving neurons in the ischaemic penumbra from the adverse effects of hypoxia.³ In preclinical models, many neurovascular-protective agents have been shown to preserve membrane integrity, block the influx of calcium, and inhibit cell pathways that lead to neuronal injury and death. However, none of these agents have proved effective in phase 3 clinical trials.⁴ The most promising is citicoline, an exogenous form of cytidine-5'-diphosphocholine (CDP-choline), which is an essential intermediate in the generation of phosphatidylcholine, a phospholipid that is degraded during brain ischaemia to fatty acids and free radicals.⁵

Citicoline accelerates resynthesis of phospholipids and enhances neuroregenerative processes after experimental stroke in rats.⁶ Moreover, citicoline seems to be safe, well tolerated, and effective in human beings. An individual patient-data meta-analysis of four randomised, placebo-controlled, double-blinded trials of citicoline (500–2000 mg daily) in 1652 patients with moderate-to-severe ischaemic stroke showed that citicoline significantly increased recovery at 3 month

follow-up (odds ratio [OR] 1.33, 95% CI 1.10–1.62).⁷ Similar results were produced by an updated meta-analysis of tabulated data.⁸

The ICTUS trial randomly assigned 2298 patients with moderate-to-severe ischaemic stroke of less than 24 h onset to double-blinded treatment with citicoline (2000 mg daily) or placebo for 6 weeks.¹ Follow-up of the intention-to-treat population at 90 days revealed no significant difference in the primary outcome of recovery (OR 1.03, 95% CI 0.86–1.25; $p=0.364$), as measured by a global test combining the National Institutes of Health Stroke Scale (NIHSS) ≤ 1 , modified Rankin score (mRs) ≤ 1 , and Barthel Index ≥ 95 . No difference in the distribution of mRs occurred between treatment groups as assessed by shift analysis (OR for improvement over the six scores was 1.02, 0.88–1.19). These results were consistent among the secondary and safety outcomes. Analysis of prespecified subgroups showed significant heterogeneity in the effect of citicoline in participants older than 70 years of age, with moderate stroke severity (NIHSS <14), and those not treated with rt-PA. These results are hypothesis-generating rather than conclusive, especially in a trial with an overall neutral result.⁹

Dávalos and colleagues are to be congratulated for the manner in which ICTUS has been planned, conducted, and presented. The trial's results are likely to be internally valid, and double our collective experience with citicoline in clinical trials of acute ischaemic stroke. The main potential source of