LETTERS

LIMITATIONS OF OPT-OUT HIV SCREENING AND MOTHER–CHILD HIV TRANSMISSION

We read with interest the article from Hughes et al. reporting their experiences with routine opt-out HIV screening in Alberta, Canada.[1] In Southern Alberta we have seen a similar success rate in this provincial antenatal opt-out HIV screening program, with 98% of 160 000 pregnancies in our region being screened between January 2002 and August 2009. Seventy-seven HIV-infected mothers were engaged into care. However, our experiences (illustrated by the following cases) have shown that even such a successful and comprehensive screening program does not completely remove the risk for mother–child HIV transmission. In the first case, a juvenile female unaware of her pregnancy presented to a rural hospital in the final stage of labor (28 weeks gestation). Delivery rapidly followed and the immediate medical and social crises, along with a negative HIV test 2 years prior, led to the recommended routine HIV screening being missed. Breastfeeding did not occur. One month later the infant developed signs of a viral infection. HIV infection was diagnosed (DNA based PCR testing positive and a plasma HIV viral load of 1.4×10^7 copies/mL).

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Iranian women protesting against the veil march in central Tehran on March 11, 1979, protected by young men. Printed with permission of Bettmann/Corbis.

In the second case, a woman aged 29 years (gravida 3, para 2, and known injection drug user) who had received no prenatal care delivered vaginally 20 minutes after arriving at the hospital. Maternal HIV infection was diagnosed postnatally and the newborn was started on a regimen of Zidovudine (GlaxoSmithKline, Research Triangle Park, NC) and Nevirapine (Boehringer Ingelheim Pharmaceuticals, Ridgefield, CT) 6 hours after birth.[2] No breastfeeding occurred. HIV infection was confirmed (HIV PCR DNA positive) in the infant at age 2 months despite 6 weeks of oral Zidovudine.

In the third case, a woman aged 20 years who tested HIV positive during prenatal screening but then disconnected from prenatal care arrived at the hospital in labor (33 weeks gestation). She was started on intravenous Zidovudine and delivered via caesarean section 8 hours later. The newborn received 1 dose of Nevirapine at birth and began a 6-week regimen of Zidovudine. No breastfeeding occurred. HIV infection in the infant was diagnosed after 2 months based on clinical signs and positive HIV serology but the infant died soon thereafter and an autopsy was not performed.

In the fourth case, as reported elsewhere,[3] a mother was diagnosed with HIV early in pregnancy and delivered an uninfected infant. She subsequently infected her child at 8 months through nonadherence to antiretroviral therapy and breastfeeding (despite medical advice) as a result of social and cultural pressures.

We fully agree with Hughes et al. that opt-out antenatal HIV testing is a major step in reducing mother–child HIV transmission. However, even with comprehensive opt-out testing, ongoing vigilance at all points of care is still vital for detection and minimization of possible HIV transmission. ■

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Contributors

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Human Participant Protection

All data were obtained through revisions of clinical charts; no identifiers are used in any of the cases. Approval from an institutional ethics review board was not needed.

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HUGHES ET AL. RESPOND

Siemieniuk et al. reported their experience with routine opt-out HIV screening in Southern Alberta, Canada, and the limitations of this approach. While we believe that routine opt-out testing has been a critical factor in reducing mother to child transmission (MTCT) of HIV in our region, we acknowledge that this approach does not eliminate the risk. Over the past 5 years (2004 to 2008 inclusive), approximately 3.5% of women have opted out of HIV prenatal testing in Alberta. Opportunities to prevent MTCT may be missed in women who decline testing. In addition, women may not seek prenatal care, as was the case for the single infant who was perinatally infected with HIV in our study. Following the completion of our study in February 2006, we have had 2 additional cases of MTCT in Northern Alberta. In the first case, a woman originally from an HIV endemic country who had been living in Europe for several years arrived in Canada near the term of her pregnancy. She was assumed to have negative prenatal serology (records were in a foreign language) and was not tested by her obstetrician. She developed *Pneumocystis jiroveci* pneumonia 7 months postpartum and she and her infant proved to be HIV positive. In the second case, an Aboriginal woman with no other risk factors requested testing 6 weeks postpartum, as she suspected her partner had recently acquired HIV; both she and her infant were found to be HIV positive. An HIV test that had been performed during the second trimester of her pregnancy was negative.

The cases presented by Siemieniuk et al., in addition to our own experience, confirm that further strategies to eliminate MTCT are needed. As recommended by the United States Centers for Disease Control and Prevention, strategies to further reduce the incidence of MTCT may include a second HIV test in the third trimester, either in all women or in identified high-risk groups. In addition, rapid HIV testing during labor is recommended for women with undocumented HIV status.[1] The acceptability and feasibility of rapid HIV testing during labor has been demonstrated in a multicenter trial.[2] We believe that approaches to eliminating MTCT should include development of more accessible, acceptable, and flexible antenatal services, including HIV testing for disadvantaged populations, to reduce the number of women not receiving prenatal care. In noncompliant pregnant women, minimizing the risk of MTCT will continue to require a flexible, multidisciplinary, and inevitably labor-intensive individualized response. ■

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Contributors

C.A. Hughes drafted the letter. All authors provided input into the content and critically reviewed the letter.

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ESTIMATED AUTISM RISK, OLDER REPRODUCTIVE AGE, AND PARAMETERIZATION

In their recent article, King et al.[1] seemed to dismiss the conclusion supported by previous epidemiologic studies (including 1 authored by us)[2,3] that increasing maternal age and paternal age are both independently associated with autism risk. Based on their analysis of California Department of Disabilities Services (DDS) data, King et al. argued that the previously observed paternal age effect is an artifact of pooling data across successive birth cohorts during a period in which both the prevalence of children receiving services for autism and the proportion of births to older parents have increased, essentially asserting that observed paternal age effects are confounded by birth cohort effects. Although King et al. acknowledge that our study,[2] based on a single birth year showing significant independent effects of both parents' ages on autism risk, is not subject to this artifact and, although the other study they reference found a paternal age effect after controlling for birth year,[3] they still conclude that "analyses that do not suffer