The Prevalence of Hepatitis C Virus Antibody in HIV-Negative Persons in Kenya, 2007

Kathleen N. Ly,^{1*} Andrea A. Kim,² Jan Drobeniuc,¹ Maja Kodani,¹ Joel M. Montgomery,³ Barry S. Fields,³ and Eyasu H. Teshale¹

¹*Division of Viral Hepatitis, National Center for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention, U.S. Centers for Disease Control and Prevention, Atlanta, Georgia;* ²*Division of Global HIV/AIDS, Center for Global Health, U.S. Centers for Disease Control and Prevention, Atlanta, Georgia;*

³*Division of Global Health Protection, Center for Global Health, U.S. Centers for Disease Control and Prevention—Kenya, Atlanta, Georgia*

Abstract. The prevalence of hepatitis C virus (HCV) infection in the Kenyan population has not been previously determined. We estimated the Kenyan HCV prevalence in HIV-negative persons aged 15–64 years. This is a retrospective cross-sectional study using data from the 2007 Kenya AIDS Indicator Survey—a nationally representative sample of 15,853 persons aged 15–64 years who completed a health interview and provided a blood specimen. Of the 1,091 randomly selected participants, 50 tested positive for HCV antibody using the automated chemiluminescence immunoassay, corresponding to a weighted HCV antibody positivity rate of 4.4% (95% confidence interval: 3.3–5.9%) or 848,000 (range: 634,000–1,100,000) persons. Hepatitis C virus RNA, a marker for current infection, was not detected in any of the tested antibody-positive specimens. The high HCV antibody prevalence together with no current infection suggests that some HCV antibody serologic testing in Kenya may result in false positives whereas others may be because of spontaneous viral clearance.

INTRODUCTION

Hepatitis C virus (HCV) infection is a major global public health problem with an estimated 2.8% of the world's population, or > 185 million HCV-infected persons.¹ In addition, HCV infection causes an estimated 499,000 deaths worldwide annually.² It is also estimated that 60–80% of persons with circulating HCV antibody (anti-HCV) are chronically infected.³ The most recent global burden of disease study estimated that the prevalence of anti-HCV for East sub-Saharan Africa, which included Kenya and 15 other countries, was approximately 2.0%.¹ However, the prevalence of HCV infection in Kenya at the national level was not documented. Like in many countries, data on the prevalence of HCV infection in Kenya are limited due to a number of challenges. First, the lack of access to highly sensitive and specific diagnostic tests means that cases go undetected. Second, because of the asymptomatic nature of most infections, infected individuals do not seek medical attention and are, therefore, not tested. Third, there is no surveillance system for HCV because of competing priorities from diseases that have more immediate adverse health outcomes and public health concerns.⁴ Finally, even when limited data on HCV antibody prevalence are available, data on HCV RNA status are lacking.

In addition to various clinical and administrative challenges in estimating HCV prevalence in Kenya, recent studies from sub-Saharan African countries have shown little to no HCV RNA-positive tests of samples previously tested positive for anti-HCV.^{5–8} Among female sex workers in Kenya, Andonov et al.⁷ found that 5% initially tested positive for anti-HCV; however, none were confirmed anti-HCV positive in follow-up tests nor were they HCV RNA positive. In another study, among 100 anti-HCV-positive samples collected from a blood donor center in Nairobi, Kenya, 16 were confirmed anti-HCV positive using a passive hemagglutination test, and of these, ten were confirmed HCV RNA positive.⁸ Similarly, in a Malawi study by Chasela et al.,⁵ recombinant immunoblot assay

(RIBA) confirmatory antibody testing was performed on HIV-positive pregnant women who were initially anti-HCV positive, and found that only two of the 109 specimens were confirmed anti-HCV positive; none of the initial anti-HCV reactive specimens were HCV RNA positive. The high-positive rate for anti-HCV in otherwise HCV RNA-negative samples appears to occur independent of the type of screening assay used or HIV coinfection status, suggesting that host characteristics may be contributing to the false anti-HCV positivity. Despite the known limitations of screening for HCV in counties in sub-Saharan Africa, the anti-HCV prevalence is high in these areas; thus, developing a HCV testing algorithm that can accurately detect true HCV-infected persons in these settings is critical, especially because highly effective and safe treatments are now available.^{9,10} This study aimed to estimate the prevalence of past or current HCV infection (anti-HCV positive) and prevalence of current HCV infection (anti-HCV and HCV RNA positive) in HIV-negative adolescents and adults at the regional and national levels in Kenya.

MATERIALS AND METHODS

Survey design. The data source for this study was the 2007 Kenya AIDS Indicator Survey (KAIS). The KAIS 2007, conducted by the Kenyan Ministry of Public Health and the U.S. Centers for Disease Control and Prevention (CDC), collected sera and interview data, including demographic, risk, and medical history information from a nationally representative household sample of persons aged 15–64 years. The sampling frame consisted of 1,260 rural and 540 urban clusters, of which 294 (23%) rural and 121 (22%) urban clusters were sampled using a stratified, two-stage cluster sample design that selected 25 households per cluster for a total of 10,375 households. More detailed information on the survey design for KAIS 2007 is available from the KAIS 2007 final report.¹¹

The survey, conducted from August to November 2007, included a standard questionnaire and blood draw, mainly for HIV testing and eventually for viral hepatitis testing. Because the survey was conducted primarily to test for HIV-associated biomarkers, all HIV-positive serum samples were exhausted and only HIV-negative samples with ≥ 1 mL of sera were eligible for HCV testing. From the 3,180 HIV-negative

* Address correspondence to Kathleen N. Ly, Division of Viral Hepatitis, U.S. Centers for Disease Control and Prevention, 1600 Clifton Rd. NE, Mailstop G-37, Atlanta, GA 30333. E-mail: kathleenly@cdc.gov

specimens that had ≥ 1 mL of stored sera, an equal probability sampling method was applied that randomly selected 1,091 specimens for hepatitis B, C, and E testing stratified by Kenyan province and then gender to ensure good representation from each province. Hepatitis C virus testing was performed at the CDC Hepatitis Reference Laboratory in Atlanta, GA.

Laboratory testing. Anti-HCV was tested by the automated chemiluminescence immunoassay (CIA; VITROS anti-HCV; Ortho-Clinical Diagnostics, Inc., Rochester, NY). Because HCV laboratory testing and analysis took place in 2014, the production of RIBA anti-HCV confirmatory testing had already been discontinued in the United States, and therefore, confirmatory testing from RIBA could not be performed on any of the positive anti-HCV by CIA specimens. Participants who were positive for anti-HCV by CIA were defined as having past or current HCV infection. Signal to cutoff ratios were assessed to isolate high-positive anti-HCV specimens, that is, ≥ 8.0 .¹² All anti-HCV-positive specimens by CIA were further tested for HCV RNA by a laboratory-developed (LDT) quantitative polymerase chain reaction (qPCR) with primers and probe from a highly conserved region of the 5'UTR, as previously described.¹³ The LDT qPCR accurately detected and measured the viral load when validated against the quantitative COBAS Ampliprep/Taqman HCV v2.0 IVD assay (Roche, Indianapolis, IN) and showed similar analytic performance in both serum and plasma. In addition, this qPCR assay was used in a previously published study examining HCV RNA positivity in US-bound refugees from parts of Africa, including Kenya.¹³ When compared with an FDA licensed assay with lower limit of detection of 15 IU/mL, the analytical sensitivity of the LDT qPCR assay was slightly lower (100 IU/mL), but the clinical sensitivity of both assays was 100%. The additional benefit of using a low-volume total nucleic acid isolation LDT was availability of residual extra sample in case the need arose to retest for viral genomic sequencing. The LDT qPCR required only 200 μ L for total nucleic acid isolation, whereas the commercial assay required almost 1 mL of sera. Participants who were positive for both anti-HCV and HCV RNA were defined as having current HCV infection. The KAIS 2007 study was approved by the Kenya Medical Research Institution Ethical Review Board and the CDC Institutional Review Board.

Data analysis. The anti-HCV positivity by CIA estimates were weighted and adjusted using the appropriate weighting, clustering, and stratification variables, which were designed specifically for the viral hepatitis laboratory sample to represent the overall HIV-negative population aged 15–64 years in Kenya and to account for nonresponse to the questionnaire and serologic testing. Population extrapolations were calculated using the 2007 HIV-negative Kenya population aged 15–64 years of 19.23 million, which was based on statistics from the World Bank¹⁴ and the KAIS 2007 final report.¹¹ All analyses were performed using SAS-callable SUDAAN (version 11.0) (Research Triangle Institute, Durham, NC).

RESULTS

Of the 10,375 households sampled for the 2007 KAIS study, 9,691 households (93.4%) and 17,940 persons aged 15–64 years completed the interview, and 15,853 (88.4%) of those interviewed provided a blood specimen. Of blood specimens collected, 1,104 (7.0%) were HIV positive and 11,569 (73.0%) had < 1 mL of stored sera available. There were 3,180

(20.1%) remaining specimens that met the eligibility criteria for HCV testing, which required HIV negativity and ≥ 1 mL of sera, and 1,091 (34.3%) of these specimens were randomly selected, stratified by province and gender, and tested for HCV infection.

In 2007, 50 of the 1,091 HIV-negative persons aged 15–64 years in Kenya tested positive for anti-HCV. The weighted anti-HCV positivity rate was 4.41% (95% confidence interval [CI]: 3.30–5.89%), corresponding to approximately 848,000 (CI: 634,000–1,100,000) persons (Table 1). Of the anti-HCV-positive

TABLE 1
Characteristics of HIV-negative study participants aged 15–64 years who were HCV antibody positive*, Kenya AIDS Indicator Survey, 2007

Category	HCV antibody positive	
	n/N	Wt. % (95% confidence interval)
Overall	50/1,091	4.41 (3.30–5.89)
Residing province		
Nairobi	2/153	1.63 (0.39–6.55)
Central	2/149	1.81 (0.37–8.32)
Coast	8/129	6.87 (3.19–14.18)
Eastern	2/112	2.67 (0.63–10.69)
North Eastern	5/140	3.42 (1.32–8.57)
Nyanza	18/154	10.60 (7.11–15.52)
Rift Valley	4/118	2.58 (0.75–8.44)
Western	9/136	6.16 (3.32–11.17)
Rural/urban		
Rural	46/785	5.52 (4.10–7.40)
Urban	4/306	1.58 (0.54–4.51)
Gender		
Male	21/431	5.22 (3.24–8.31)
Female	29/660	3.92 (2.64–5.78)
Age group (year)		
15–24	13/378	3.31 (1.88–5.77)
25–34	13/272	4.78 (2.59–8.65)
35–44	9/191	5.24 (2.62–10.24)
45–54	7/153	2.82 (1.22–6.39)
55–64	8/97	8.71 (3.98–17.99)
Education†		
No primary	16/223	7.77 (4.23–13.84)
Incomplete primary	14/295	4.21 (2.41–7.27)
Complete primary	11/212	5.40 (2.97–9.61)
Secondary or higher	9/361	2.63 (1.28–5.31)
Marital status		
Never married/cohabitated	12/328	4.05 (2.28–7.08)
Formerly married/cohabitated	5/130	3.28 (1.39–7.56)
Currently monogamous	32/554	5.43 (3.67–7.97)
Currently polygamous	1/79	1.11 (0.15–7.57)
Blood transfusion		
Yes	3/63	2.50 (0.70–8.56)
No	47/1,028	4.59 (3.41–6.15)
Lifetime number of sex partners		
No partner/never had sex	5/148	2.95 (1.16–7.32)
1 partner	11/337	2.64 (1.45–4.78)
2+ partners	33/579	5.82 (4.05–8.31)
Lifetime number of medical injections		
0 injections	39/724	5.26 (3.73–7.36)
1 injection	5/95	3.77 (1.55–8.91)
2–3 injections	1/168	0.74 (0.10–5.09)
4+ injections	5/104	4.83 (1.69–13.03)
Had sex partner in last 12 months?		
Yes	28/756	3.57 (2.39–5.29)
No	16/284	5.10 (3.16–8.13)
Have you ever used a condom?		
Yes	10/268	3.67 (1.86–7.12)
No	33/612	5.09 (3.49–7.38)

HCV = hepatitis C virus.

* There were 48 specimens with insufficient quantity and, therefore, were not presented in this table.

† Primary school is defined as elementary school usually from the ages of about 5–11 years. Secondary school is middle and high school usually from the ages 10 or 11 to 18 or 19 years.

specimens, 10 did not have sufficient sera volume for HCV RNA testing, and the remaining 40 specimens were HCV RNA negative. Seven of the 50 anti-HCV-positive specimens had a signal to cutoff ratio > 8.0 , indicating a high likelihood of true anti-HCV positivity. Of these, five had insufficient sera volume and, therefore, could not undergo HCV RNA testing to determine current HCV infection, whereas the other two were HCV RNA negative. Although not statistically significantly different, the estimated anti-HCV point positivity rate was notably highest among those who resided in the Nyanza Province (10.60%; 95% CI: 7.11–15.52%), followed by persons aged 55–64 years (8.71%; 95% CI: 3.98–17.99%) and persons with no primary education (7.77%; 95% CI: 4.23–13.84%). Geographically, the estimated anti-HCV point positivity rate was lowest among those who resided in an urban region (1.58%; 95% CI: 0.54–4.51%) and those who resided in the Nairobi (1.63%; 95% CI: 0.39–6.55%) and Central (1.81%; 95% CI: 0.37–8.32%) provinces.

DISCUSSION

This is the first and largest nationwide survey that aimed to assess the HCV infection prevalence in Kenya. Based on HCV RNA testing of initial anti-HCV-positive specimens, this study did not detect any cases of current HCV infection, whereas the weighted anti-HCV positivity rate, indicative of past infection, was 4.4% (95% CI: 3.3–5.9%). With a relatively high anti-HCV positivity rate, the lack of detectable viremia in tested samples could indicate that some of the anti-HCV reactive specimens may have either resolved their HCV infection because of spontaneous viral clearance or be false anti-HCV positives because chronic infection develops in approximately 60–80% of HCV-infected persons.³ However, the fact that five specimens with high positive signal to cutoff ratios could not undergo HCV RNA testing because of insufficient sera volume leaves uncertainty in determining if any current HCV infection was present in the population. A recent study by Layden et al.¹⁵ found that 74–88% of HCV antibody-positive blood donors from Ghana had HCV RNA. However, in the national Kenya population and in subpopulations in Malawi, Uganda, and Kenya, there was a high HCV antibody positivity rate with low or no circulating HCV viremia among HIV-positive and HIV-negative persons.^{5–7} Although this study was not able to detect current HCV infection in the general Kenya population, there was one blood donor study that indicated a 1.08% HCV prevalence in 2008–2009 among blood donors in Kenya;¹⁶ at the time the data were collected for that study, blood donors in Kenya were likely primarily family replacement and other remunerated donors as opposed to voluntary donors. Therefore, the 1.08% HCV prevalence from this blood donor study may be more representative of the general population than other HCV prevalence studies in Kenya where the target groups are usually higher risk groups. The results from our study suggest that testing using the standard HCV antibody screening test in the absence of a RIBA confirmatory test in these settings may yield some false-positive anti-HCV results. One possible explanation discussed by other studies for the reason behind these discrepant test results may be that other pathogens that are prevalent in sub-Saharan Africa may result in antibodies that could mimic HCV antibody and cross-react, resulting in a false-positive anti-HCV result.^{6,17} In populations where the HCV antibody prevalence is $< 10\%$, HCV screening tests may yield a

higher false-positive rate than in populations where the HCV antibody prevalence is higher.¹⁸ In these types of populations, HCV RNA testing is needed to diagnose true current HCV infection. It is also possible that the PCR assays used to detect HCV RNA may also perform differently in this population. The study from Ghana used a PCR assay that was different from those used in the two studies in Malawi and Uganda.^{5,6,15}

This study found that the initial screening anti-HCV positivity rate, although not statistically significantly different, was disproportionately highest in the Nyanza Province at 10.6% (95% CI: 7.1–15.5%) and among persons aged 55–64 years at 8.7% (95% CI: 4.0–18.0%). The reason behind this disparity is not apparent based on the findings from this study but warrants future investigation to understand the link between local disease epidemiology and seropositivity with standard anti-HCV screening tests.

There are limitations to consider. First, because the RIBA anti-HCV test was discontinued before the KAIS specimens underwent HCV testing, we could not confirm that the 50 initial anti-HCV-positive specimens were false positives. In addition, of the seven specimens with high anti-HCV signal to cutoff ratios, five did not have sufficient sera volume to test for HCV RNA; therefore, we could not confirm nor rule out current HCV infection in these high anti-HCV-positive specimens. However, HCV RNA negativity was found among the 38 anti-HCV positive specimens with signal to cutoff ratios < 8.0 and two specimens with signal to cutoff ratios ≥ 8.0 . Because most HCV-infected adults are expected to develop chronic HCV infection, it is likely that at least some of the anti-HCV-positive specimens were false positives. Third, because only leftover HIV-negative samples were available from KAIS 2007 for the testing of other infectious agents, there may be concern regarding the effect of excluding HIV-positive specimens on the anti-HCV and HCV RNA prevalence estimates. Next, KAIS 2007 samples were collected and stored for about seven years until viral hepatitis laboratory analyses were performed. Although samples were preserved in accordance with standard storage procedures, it could be possible that multiple freeze-thaws over time may have occurred, resulting in degradation of HCV RNA and reduction of viral loads to an undetectable level. In addition, the HCV prevalence in Kenya assessed for a period that was about 10 years ago may not reflect the current HCV prevalence in Kenya; however, this country, like the great majority of countries in sub-Saharan Africa, has never had a serosurvey for HCV prevalence until now. Therefore, this study is unique and is the first to establish a baseline estimate for HCV prevalence in Kenya. In one study that examined the World Health Organization's Global Database on Blood Safety data from 38 sub-Saharan African countries, including Kenya, the median percentage of blood donations that were anti-HCV-reactive, did not change drastically over time, from 1.4%; interquartile range (IQR), 0.6–3.1% in 2000 and 2004 to 0.9%; IQR, 0.5–1.7% in 2010 and 2011.¹⁹ Still, future studies are warranted to determine the extent to which the HCV prevalence in Kenya has changed since 2007. Finally, HCV RNA testing was performed on only 50 samples. Although this number may be large enough to achieve statistically significant results, these data could still be subject to biases even though the appropriate survey design variables were applied.

In conclusion, this study could not find any current HCV infection among the 50 sampled HIV-negative adolescents and adults in Kenya. However, using an anti-HCV screening

assay alone yielded an anti-HCV prevalence that was higher than the world HCV prevalence (4.4% versus 2.8%). Had HIV-positive samples been available for the analysis, the anti-HCV prevalence would likely have been even higher. These results correlate with previous findings of a high anti-HCV false positivity rate in select populations in sub-Saharan Africa.^{5–7} Understanding the prevalence of past and current HCV infection at a population level is important as HCV infection has emerged as a leading infectious disease cause of death, but is now curable. However, using results from anti-HCV screening assays in the absence of a RIBA confirmatory test may lead to overestimation of the burden of HCV infection for certain regions. In countries where a high rate of false anti-HCV positivity is observed, it is important to develop a standard HCV testing algorithm that can accurately detect true cases of HCV infection. Effectively identifying and treating HCV infections in a particular region require understanding the local prevalence of current infection rather than lifetime HCV infection. Special studies to determine the true rate of false-positive anti-HCV tests are warranted, and to determine whether antibody positivity in certain regions could be because of cross-reactivity with biologic agents other than HCV.

Received October 24, 2017. Accepted for publication February 23, 2018.

Published online April 23, 2018.

Acknowledgments: We thank John Williamson for his statistical support in the study design including development of the viral hepatitis laboratory sampling weighting variables, Anthony Waruru for support in the management of data from the KAIS 2007, and Tonya Hayden for testing the specimens for HCV RNA.

Financial support: The U.S. President's Emergency Plan for AIDS Relief funded the survey from which specimens were tested for this analysis.

Disclaimer: The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the U.S. Centers for Disease Control and Prevention (CDC).

Authors' addresses: Kathleen N. Ly, Andrea A. Kim, Jan Drobeniuc, Maja Kodani, Joel M. Montgomery, Barry S. Fields, and Eyasu H. Teshale, U.S. Centers for Disease Control and Prevention, Atlanta, GA, E-mails: kathleen.ly@cdc.gov, aakim@cdc.gov, jdrobeniuc@cdc.gov, mkodani@cdc.gov, jmontgomery@cdc.gov, bfields@cdc.gov, and eteshale@cdc.gov.

REFERENCES

1. Hanafiah KM, Groeger J, Flaxman AD, Wiersma ST, 2013. Global epidemiology of hepatitis C virus infection: new estimates of age-specific antibody to HCV seroprevalence. *Hepatology* 57: 1333–1342.
2. Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, Abraham J, Adair T, Aggarwal R, Ahn SY, 2012. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 380: 2095–2125.
3. Liang T, Rehmann B, Seeff L, Hoofnagle J, 2000. Pathogenesis, natural history, treatment, and prevention of hepatitis C. *Ann Intern Med* 132: 296–305.
4. Jehuda-Cohen T, Unal S, 2012. The challenges of hepatitis C diagnosis and the potential role of immunology to address them. *Antivir Ther* 17: 1415–1422.
5. Chasela CS et al., 2012. Prevalence of hepatitis C virus infection among human immunodeficiency virus-1-infected pregnant women in Malawi: the BAN study. *J Clin Virol* 54: 318–320.
6. Mullis CE et al., 2013. High frequency of false-positive hepatitis C virus enzyme-linked immunosorbent assay in Rakai, Uganda. *Clin Infect Dis* 57: 1747–1750.
7. Andonov A, Songok E, Ochwoto M, Osiowy C, Ball TB, 2015. HIV and hepatitis C virus co-infection in Kenya. *Lancet Infect Dis* 15: 1377–1378.
8. Mwangi J et al., 2016. Hepatitis C virus genotypes in Kenya. *Arch Virol* 161: 95–101.
9. AASLD/IDSA Guidance Panel; Chung RT et al., 2015. Hepatitis C guidance: AASLD-IDSA recommendations for testing, managing, and treating adults infected with hepatitis C virus. *Hepatology* 62: 932–954.
10. World Health Organization, 2016. *Guidelines for the Screening, Care, and Treatment of Persons with Hepatitis C Infection*. Available at: <https://www.ncbi.nlm.nih.gov/books/NBK362924/>. Accessed January 6, 2016.
11. National AIDS and STI Control Programme (Kenya), CDC Kenya, Centers for Disease Control and Prevention, (U.S.), Joint United Nations Programme on HIV/AIDS, United States Agency for International Development, U.S. President's Emergency Plan for AIDS Relief, World Health Organization, 2009. *Kenya AIDS Indicator Survey 2007: Final Report*. Available at: <http://stacks.cdc.gov/view/cdc/12122/Share>. Accessed June 22, 2015.
12. Centers for Disease Control and Prevention (U.S.), 2011. *Viral Hepatitis Laboratory Testing*. Available at: <https://www.cdc.gov/hepatitis/hcv/labtesting.htm>. Accessed January 10, 2018.
13. Mixson-Hayden T, Lee D, Ganova-Raeva L, Drobeniuc J, Stauffer WM, Teshale E, Kamili S, 2014. Hepatitis B virus and hepatitis C virus infections in United States-bound refugees from Asia and Africa. *Am J Trop Med Hyg* 90: 1014–1020.
14. The World Bank, 2015. *Population Ages 15–64 (% of Total)*. Available at: <http://data.worldbank.org/indicator/SP.POP.1564.TO.ZS/countries?page=1>. Accessed April 16, 2015.
15. Layden JE et al., 2015. High frequency of active HCV infection among seropositive cases in west Africa and evidence for multiple transmission pathways. *Clin Infect Dis* 60: 1033–1041.
16. Basavaraju SV et al., 2010. Reduced risk of transfusion-transmitted HIV in Kenya through centrally co-ordinated blood centres, stringent donor selection and effective p24 antigen-HIV antibody screening. *Vox Sang* 99: 212–219.
17. Seremba E, Ocamo P, Opio CK, Kagimu M, Thomas DL, Yuan HJ, Attar N, Lee WM, 2010. Poor performance of hepatitis C antibody tests in hospital patients in Uganda. *J Med Virol* 82: 1371–1378.
18. Mayer D, 2004. Bayes' theorem, predictive values, post-test probabilities, and interval likelihood ratios. *Essential Evidence-Based Medicine*, Chapter 24. Cambridge, United Kingdom: Cambridge University Press.
19. Apata IW, Averhoff F, Pitman J, Bjork A, Yu J, Amin NA, Dhirga N, Kolwaite A, Marfin A; Centers for Disease Control and Prevention (CDC), 2014. Progress toward prevention of transfusion-transmitted hepatitis B and hepatitis C infection—sub-Saharan Africa, 2000–2011. *MMWR Morb Mortal Wkly Rep* 63: 613–619.