Hepatitis C Virus Infection

Updated: January 28, 2026
Reviewed: January 28, 2026

Panel's Recommendations

I. Do children with HIV/hepatitis C virus (HCV) coinfection warrant any specific surveillance or monitoring beyond what is recommended for HIV mono-infection and HCV mono-infection?

Children with HIV/HCV coinfection should receive the same routine care and monitoring that is recommended for children with HIV mono-infection and children with HCV mono-infection (strong, moderate).

II. Should infants born to women known to have hepatitis C be tested for HCV infection, and if yes, when and how?

Testing for HCV infection should be performed on any infant or child whose mother is known to have hepatitis C (strong, high).

As per Centers for Disease Control and Prevention guidelines, all pregnant women should be tested for hepatitis C with each pregnancy.

All children perinatally exposed to HCV should receive a nucleic acid test (NAT) for HCV RNA at age 2–6 months. If not performed earlier, NAT for HCV RNA can be performed at age 7–17 months (strong, high).

A negative NAT for HCV RNA at age 2–6 months is highly suggestive (>99% negative predictive value) of no perinatal transmission. For those not tested earlier, negative anti-HCV immunoglobulin G testing at 18 months is confirmatory of no HCV (strong, low).

In consultation with a health care provider with expertise in pediatric hepatitis C management, children who test positive for HCV RNA should have follow-up care through age 3 years to assess eligibility for HCV treatment (strong, high).

III. For infants and children born to women with HIV/HCV coinfection, do specific obstetric or infant feeding practices reduce the risk of perinatal transmission of HCV?

Recommendations on route of delivery and intrapartum management are the same for HIV/HCV coinfection and HIV mono-infection. Unlike HIV, available data suggest breastfeeding does not pose an appreciable risk of transmission to infants of mothers with HCV. Risk of HCV transmission via breastfeeding in the setting of HIV/HCV coinfection is likely similar to HCV mono-infection, although no data currently exist. Decisions regarding appropriate feeding practices in infants born to mothers with HIV/HCV coinfection should be determined on a case-by-case basis guided by shared decision making and driven by maternal HIV virological status. Mothers should abstain from breastfeeding from nipples that are cracked or bleeding or breasts affected by mastitis (strong, moderate).

IV. For women with HIV/HCV coinfection, does treatment of HCV infection with sustained virologic response prior to pregnancy (as opposed to no treatment or treatment failure) reduce the risk of HCV perinatal transmission?

Women with HIV/HCV coinfection who are of childbearing potential and wish to become pregnant should be evaluated for treatment of HCV infection prior to conception. They should be treated for HCV infection to reduce their risk of liver disease progression and perinatal transmission of HCV (strong, high).

Ribavirin-containing regimens are no longer recommended for treatment of HCV infection given the availability of safer and more efficacious treatment options (strong, high).

There are no large-scale clinical trials evaluating the safety of pangenotypic direct-acting antiviral (DAA) regimens during pregnancy. Treatment can be considered on an individual basis after a discussion of the potential risks and benefits (strong, low).

See details in the HCV/HIV Coinfection section of the Perinatal Guidelines and the American Association for the Study of Liver Diseases (AASLD)/Infectious Diseases Society of America (IDSA) HCV guidance for pregnancy.

V. What counseling should an adolescent with HIV receive to reduce the risk of HCV acquisition/transmission?

All adults and adolescents with HIV should be counseled to avoid injection drug use. If using drugs, they should—

- Avoid reusing and/or sharing needles, *and*
- Be tested for HCV and hepatitis B virus, *and*
- Receive appropriate referral and therapy for substance use disorder (**strong**, **high**).

Unprotected sex has been linked to HCV transmission, so all adolescents—including those with HIV, multiple sex partners, or sexually transmitted infections—should be advised to use barrier protection (**strong**, **moderate**).

VI. Among children with HIV/HCV coinfection, which vaccinations to reduce risk of liver disease are available?

Hepatitis A and hepatitis B vaccines are recommended for all children, including those with hepatitis C with or without HIV coinfection, with follow-up serologic confirmation of vaccine response (**strong**, **low**).

VII. For all children with HCV mono-infection or HIV/HCV coinfection, what are the indications for HCV treatment?

Any child aged 3 years or older with HCV mono-infection or HIV/HCV coinfection should receive treatment with a pangenotypic DAA regimen (**strong**, **moderate**).

VIII. For all children with hepatitis C, what U.S. Food and Drug Administration (FDA)–approved medications are available for those requiring treatment?

Either of the FDA-approved pangenotypic DAA regimens (sofosbuvir/velpatasvir or glecaprevir/pibrentasvir) can be used for treatment (**strong**, **high**).

For more information about evaluation for DAA treatment, visit the AASLD/IDSA HCV in Children webpage.

IX. When treating children with HIV/HCV coinfection, are there significant drug–drug interactions between HCV and HIV treatment regimens that require consideration?

Clinically relevant drug–drug interactions have been identified between current FDA-approved pediatric DAA regimens and multiple classes of antiretroviral drugs that may warrant alternate therapy, dose adjustment, or extra monitoring. Reference to current AASLD/IDSA HCV guidance on patients with HIV/HCV coinfection is recommended (**strong**, **high**).

Rating of Evidence

Strength of Recommendation: Strong; Weak

Quality of Evidence: High; Moderate; Low; Very Low

Epidemiology

The prevalence of hepatitis C virus (HCV) infection among children in the U.S. National Health and Nutrition Examination Survey was 0.2% (aged 1–11 years) to 0.4% (aged 12–19 years) in the 1990s[1,2] and fell to less than 0.1% in the subsequent decade.[3] A recent modeling study estimated a prevalence of 0.03% for children aged 0 to 11 years and 0.05% for children aged 12 to 18 years.[4] Centers for Disease Control and Prevention (CDC) modeling predicts that approximately 1,700 new cases of perinatally acquired pediatric HCV infection occur annually.[5] The prevalence of HCV infection among children with HIV may be significantly higher, with HCV coinfection documented among 1.5% of 535 children with HIV in U.S. pediatric HIV clinical trials in 2003.[6] Higher rates of HCV infection have been documented in some international cohorts of children with HIV, likely due

to increased risk of HCV perinatal transmission in pregnant women with poorly controlled HIV infection.[7,8]

Perinatal transmission is the predominant mode of HCV acquisition in infants and younger children.[9,10] Injection drug use is the predominant route of infection in older children; less common modes of transmission include noncommercial body piercing or tattooing, unintentional needle stick injury, household contact or sharing of potentially contaminated personal items, and sexual exposure.[11,12] Before 1992, blood transfusion was a key route of HCV transmission in the United States.

The overall risk for perinatal HCV transmission from a woman with HCV mono-infection ranges from 4% to 10%.[9,13-21] The primary risk factor for perinatal HCV transmission is maternal HCV viremia at delivery, although an absolute threshold for HCV transmission has not been identified.[14,22-27] HCV genotype does not appear to affect the risk of perinatal HCV transmission.[14,20] Although a few studies have suggested that vaginal delivery increases risk of HCV transmission[13,15,17,22] and that HCV can be transmitted during the intrapartum period,[28] most studies have found that mode of delivery does not appear to influence overall perinatal HCV transmission.[10,15,16,18,29-33] In addition, even though HCV RNA can be detected in breast milk, studies of infants born to women with HCV have not demonstrated a higher risk of HCV transmission in breastfed infants than in those who are formula fed.[10,13-16,18,25,28,29,34]

Early studies demonstrated that maternal HIV/HCV coinfection increased the risk of perinatal transmission of HCV, with perinatal HCV transmission rates of 6% to 23% reported for infants born to women with HIV/HCV coinfection.[9,13-15,19,26,30-32,35-40] Furthermore, a few studies suggested that children who have HIV during the perinatal period may be more likely than those who do not have HIV to acquire HCV from mothers with HIV/HCV coinfection.[30,31,37,38] Dual virus transmission was reported in 4% to 10% of children born to mothers with HIV/HCV coinfection.[13,30,35,37,39] HCV RNA levels are hypothesized to be higher among women with HIV/HCV coinfection than in those with HCV mono-infection, which could account, in part, for the increased risk of perinatal HCV transmission from pregnant women with HIV/HCV coinfection. However, not all studies have found higher levels of HCV viremia in mothers with HIV/HCV coinfection.[24,31,36] Several recent studies in the era of routine combination antiretroviral therapy (ART) during pregnancy found that the risk of perinatal HCV transmission is far lower than rates found in historical studies when few or no antiretrovirals (ARVs) were available, suggesting that perinatal transmission of HCV may be significantly reduced in women with HIV who are receiving ART.[40,41]

Recommendations on route of delivery, intrapartum management, and infant feeding are the same for HIV/HCV coinfection and HIV mono-infection. Further details are available in the Perinatal Guidelines and Pediatric Antiretroviral Guidelines.

The incidence of HCV infection in adolescents and young adults has been increasing since 2010. Data from the CDC show an increase in the incidence of acute HCV infection among people aged 20 to 29 years from 0.75 cases per 100,000 population in 2010 to 2.2 cases per 100,000 population in 2022. The incidence rate of acute HCV infection in people aged 20 to 29 years peaked in 2018 at 3.0 cases per 100,000 population, which was the highest at the time but now surpassed by the rate in 30 to 39 year olds of 3.6 cases per 100,000 in 2022.[42] The incidence of HCV infection is higher in rural areas, and the majority of new infections are in people with a history of injection drug use (IDU).[43-47] HIV is a significant risk factor for HCV acquisition in men who have sex with men (MSM).[46,48] MSM with HIV who were followed in the Multicenter AIDS Cohort Study (MACS)

from 1984 to 2011 were almost six times more likely to acquire HCV than MSM who did not have HIV (incident risk ratio 5.98). The incidence rate for HCV infection in MSM with HIV recruited to MACS between 2005 and 2011 was 5.16 per 1,000 person-years.[49] The incidence of HCV infection was somewhat lower in a cohort of MSM with HIV in Boston tested between 2008 and 2009 who had an annualized incidence of 1.63 per 1,000 person-years. Risk factors for HCV acquisition included noninjection drug use and a history of sexually transmitted infections.[50]

Clinical Manifestations

The clinical course of HCV infection appears to be more benign in children with perinatal hepatitis C than in adults with newly acquired hepatitis C.[11,51,52] Most children with hepatitis C are asymptomatic; however, some may experience nonspecific symptoms—such as fatigue, myalgias, and poor weight gain—and develop hepatomegaly.[11,52,53] Intermittent asymptomatic elevations in transaminase levels are common during the first 2 years of life.[53-56] Across studies of children with hepatitis C, 20% to 65% of children had apparent clearance of HCV viremia; 50% had chronic asymptomatic infection, characterized by intermittent viremia, rare hepatomegaly, and usually normal liver transaminase levels; and 30% had chronic active infection with persistent viremia and abnormal transaminase levels.[57,58]

Histopathologic inflammatory changes of chronic hepatitis may be present in children with chronic hepatitis C despite a lack of symptoms, normal serum transaminases, and low HCV RNA levels.[53] Analysis of liver histology in 121 treatment-naive pediatric patients showed some degree of inflammation in all samples, mild fibrosis (Ishak stage 1–2) in 80% of specimens, and cirrhosis in 2% of specimens.[59] Most children with chronic hepatitis C who have undergone liver biopsy and are included in published studies typically have mild-to-moderate liver disease as determined by signs of structural alterations, inflammatory activity, and necrosis.[11,24,52,55] Similar proportions of children with perinatally and parenterally acquired HCV have signs of chronic hepatitis on liver biopsy.[56] A small subset of children may develop severe liver disease. In a study of 60 children with perinatally or transfusion-acquired hepatitis C for a mean duration of 13 years, 12% had significant fibrosis on liver biopsy.[52] Older age at time of infection and elevated serum gamma-glutamyl transpeptidase correlated with fibrosis; serum transaminase levels correlated with inflammation.[52]

In adults with HIV/HCV coinfection, the natural history of HCV infection appears to be accelerated, with more rapid progression to cirrhosis, decompensated liver disease, hepatocellular carcinoma, and death.[60,61] Reports on the effect of ART and immune reconstitution on liver-related mortality in adults with HIV/HCV coinfection are conflicting; some studies show decreases, but others show little difference in liver-related mortality.[62,63] Data are minimal on the effect of HIV/HCV coinfection on the natural history of HCV infection in children and insufficient to draw conclusions about HCV disease progression in children with HIV/HCV coinfection.[9] In a study from Spain in the early ART era comparing children with perinatal HIV/HCV coinfection to those with perinatal HCV mono-infection, HCV viremia and maximum transaminase levels were higher in the children with HIV/HCV coinfection than in those with HCV mono-infection.[64]

Data on the impact of HCV on HIV disease progression in adults are conflicting. Some studies suggest that HCV accelerates HIV progression, but others show no such effect.[9] The effect of pediatric HIV/HCV coinfection on HIV disease progression is also unclear because the number of children with HIV/HCV coinfection is small and only a few studies have evaluated any potential association. Two studies of children with perinatal HIV/HCV coinfection found no increase in HIV progression. In a study of older children with thalassemia who had HIV or HIV/HCV coinfection

through transfusion, disease progression was more rapid and mortality was higher in those with HIV/HCV coinfection than in those with HIV mono-infection.[30,39,65]

Diagnostic Assays

Both serologic assays for anti-HCV antibodies and nucleic acid tests (NATs) for HCV RNA are used to diagnose HCV infection. HCV RNA first becomes detectable 1 to 2 weeks after HCV acquisition, preceding the rather delayed antibody seroconversion that occurs an average of 7 to 8 weeks after acquisition.[66] Third-generation enzyme, chemiluminescent, and microparticle immunoassays for anti-HCV immunoglobulin G have high sensitivities and specificities, though sensitivities are lower in patients with HIV with severe immunosuppression and in acute infection prior to seroconversion. A U.S. Food and Drug Administration (FDA)–approved rapid saliva test is also available for screening adolescents and adults aged 15 years and older.[67,68]

A reactive HCV antibody test can represent current infection or past cleared infection, and the presence of HCV RNA, or HCV viremia, is indicative of current HCV infection. Diagnosis of hepatitis C requires a NAT for HCV RNA, and highly sensitive quantitative real-time polymerase chain reaction (PCR) or transcription-mediated amplification NATs have lower limits of detection of 10 to 15 IU/mL and broad quantitative range.[69]

Treatment

The goal of HCV treatment is to achieve sustained viral response (SVR12; absence of viremia 12 weeks after therapy is complete), which has been shown in adults to reduce the risk of progressive liver fibrosis and hepatocellular carcinoma. Secondary benefits of successful therapy include prevention of transmission of HCV and relief from HCV-related stigma. Because liver disease progresses slowly in most children, no rationale exists currently for urgent treatment of most cases of pediatric HCV. Many children previously included in HCV treatment studies had little or no fibrosis.[70] The relatively few children with more significant fibrosis and/or cirrhosis were often treated.[71]

The current standard of care for adults with hepatitis C is a combination of several direct-acting antivirals (DAAs) that target different HCV proteins. The DAAs are administered orally once a day, leading to rapid and consistent viral clearance, and have a very favorable side effect profile. Two pangenotypic DAA regimens are FDA approved for use in children with hepatitis C who are ages 3 years and older: sofosbuvir/velpatasvir and glecaprevir/pibrentasvir.[72-74] Each of these regimens has weight-based dosing and child-friendly formulations that make administration relatively simple.

Management Recommendations

Do children with HIV/HCV coinfection warrant any specific surveillance or monitoring beyond what is recommended for HIV mono-infection and HCV mono-infection?

Children with HIV/HCV coinfection should receive the same routine care and monitoring that is recommended for children with HIV mono-infection and children with HCV mono-infection ***(strong, moderate).***

Due to conflicting data about the rate of disease progression in children with HIV/HCV coinfection compared to that among children with either HIV mono-infection or HCV mono-infection, no

specific additional measures need to be implemented. Surveillance and diagnostic measures recommended for children with HIV/HCV coinfection include those recommended for children with HIV mono-infection and those recommended for children with HCV mono-infection. The involvement of a specialist with experience treating HCV is suggested.

Diagnosis and Testing Recommendations

Making the Diagnosis

Should infants born to pregnant women known to have hepatitis C be tested for HCV, and if yes, when and how?

Testing for HCV should be performed on any child whose mother is known to have hepatitis C ***(strong, high).***

As per CDC guidelines, all pregnant women should be tested for hepatitis C with each pregnancy.

All children perinatally exposed to HCV should receive a NAT for HCV RNA at age 2 to 6 months. Infants and children aged 7 to 17 months who have not previously been tested should receive a NAT for HCV RNA. Children aged ≥18 months who previously have not been tested should receive an anti-HCV test with reflex to NAT for HCV RNA when anti-HCV is reactive ***(strong, high).***

Undetectable HCV RNA at age 2 to 6 months is highly suggestive (>99% negative predictive value) of perinatal transmission. Perinatally exposed children aged ≥18 months with nonreactive anti-HCV test results do not have hepatitis C ***(strong, low).***

Infants and children with detectable HCV RNA should be managed in consultation with a provider with expertise in pediatric hepatitis C management and receive regular follow-up care until eligible for HCV treatment at age 3 years ***(strong, high).***

Passively transferred maternal HCV antibody can be detected up to age 18 months in infants born to women with hepatitis C. In a large cohort of children who were HCV exposed but uninfected, anti-HCV antibody was present in 15% of children at 12 months, 5% at 15 months, and 2% at 18 months.[24] Infants who are perinatally exposed to HCV should, therefore, not be tested for anti-HCV before age 18 months. Only the detection of HCV viremia can be used to diagnose HCV infection in at-risk infants aged <18 months.[75]

HCV infection can be diagnosed in infants with perinatal exposure by a NAT to detect HCV RNA after age 1 month. The sensitivity of the HCV RNA testing was low (22%) at birth but increased to 79% at age 1 month and 85% at 6 months in a multisite European study published in 2006.[76] A more recent single-site study utilizing high-sensitivity HCV RNA reverse transcription PCR assays to diagnose perinatal HCV infection at age 2 to 6 months reported a sensitivity of 100% (95% confidence interval, 87.5% to 100%).[77] The use of early NAT at 2 to 6 months is recommended by the CDC to identify infants with hepatitis C, given concerns about loss to follow-up before HCV antibody testing can be done at age ≥18 months.[76]

When testing children age ≥18 months an HCV antibody test when reactive should always be followed automatically by a NAT for HCV RNA. HCV antibody testing without reflex to HCV RNA when antibody is reactive, is incomplete testing. The detection of HCV RNA confirms current HCV infection, and detected HCV RNA should be managed in consultation with a provider with expertise

in pediatric hepatitis C management until eligible for HCV treatment at age 3 years. Children aged 3 years or older should receive HCV treatment.

Prevention Recommendations

Primary Prevention

Preventing Exposure

For infants and children born to women with HIV/HCV coinfection, do specific obstetric or infant feeding practices reduce the risk of HCV perinatal transmission?

Recommendations on route of delivery and intrapartum management are the same for HIV/HCV coinfection and HIV mono-infection. No strategy to prevent perinatal HCV transmission has been studied. Elective cesarean delivery is not associated with reduced perinatal transmission of HCV and is not recommended for this purpose for pregnant women with current hepatitis C. Maternal HIV/HCV coinfection does not alter the current recommendation for scheduled cesarean delivery for people with HIV who have HIV RNA levels >1,000 copies/mL near delivery to prevent perinatal HIV transmission.

Observational studies inconsistently associate prolonged duration of ruptured amniotic membranes, internal fetal monitoring, and perineal lacerations/episiotomy with increased risk of HCV transmission.[24,41,78]

Available data suggest breastfeeding does not pose an appreciable risk of transmission to infants born to mothers with HCV.[79] Risk of HCV transmission via breastfeeding in the setting of HIV/HCV coinfection is likely similar to that of HCV mono-infection, although no data currently exist. Decisions regarding appropriate feeding practices in infants born to mothers with HIV/HCV coinfection should be determined on a case-by-case basis guided by shared decision making and driven by maternal HIV virological status. Mothers should abstain from breastfeeding from nipples that are cracked or bleeding or breasts affected by mastitis. **(strong, moderate).**

In the setting of HIV/HCV maternal coinfection, testing the infant's HCV RNA PCR one additional time within 6 months after cessation of breastfeeding can be considered, regardless of the age of the infant at the time of breastfeeding cessation. Risks of HCV transmission and HIV transmission via breastfeeding in the setting of HIV/HCV coinfection are likely similar to risks in HCV mono-infection and HIV mono-infection, respectively, although no data currently exist. Infant feeding recommendations for people with HIV/HCV do not differ from recommendations for people with HIV who do not have HCV. For guidance about infant feeding for people with HIV,[80] see the Preventing HIV Transmission During Infant Feeding section of the Perinatal Guidelines.

For women with HIV/HCV coinfection, does treatment of HCV with sustained virologic response prior to pregnancy (as opposed to no treatment or treatment failure) reduce the risk of HCV perinatal transmission?

Women with HIV/HCV coinfection who are of childbearing potential and wish to become pregnant should be evaluated for treatment of HCV ***prior*** *to conception. They should be treated for HCV to reduce their risk of liver disease progression and the risk of perinatal transmission of HCV* ***(strong, high).***

Ribavirin-containing regimens are no longer recommended for treatment of HCV given the availability of safer and more efficacious treatment options ***(strong, high).***

There are no large-scale clinical trials evaluating the safety of pangenotypic DAA regimens in pregnancy. Treatment can be considered on an individual basis after discussion of the potential risks and benefits ***(strong, low).*** *For more information, see the HCV/HIV Coinfection section of the Perinatal Guidelines and the AASLD/IDSA guidance for HCV in pregnancy.*

No clinical trials have specifically assessed the efficacy of the treatment of maternal HCV to prevent future perinatal transmission. However, in observational studies in pregnant women who are anti-HCV reactive, those who lack detectable viremia do not transmit HCV to their children, with only rare exceptions that may relate to low-level or fluctuating maternal viremia. Thus, therapy to eradicate HCV viremia prior to pregnancy is expected to eliminate the risk of perinatal transmission. This option was not previously readily feasible because of the safety concerns for interferon and ribavirin in pregnancy, particularly the teratogenic side effects of ribavirin that preclude its use starting 6 months prior to conception and its potential transfer to infants by a breastfeeding mother.[81] The availability of highly effective interferon- and ribavirin-free combinations of all-oral DAA therapies for HCV infection that are safe in animal models of pregnancy offer a safe option for treating people prior to conception to both cure the infection and prevent future perinatal transmission of HCV.

A small study evaluating the pharmacokinetics of ledipasvir/sofosbuvir in pregnancy demonstrated 100% SVR12 and no safety concerns. Similarly, an international case series of 15 pregnant women treated with ledipasvir/sofosbuvir reported 100% SVR12 and no early safety concerns in the women or their infants.[82,83] Currently, data on the use of pangenotypic regimens during pregnancy are limited, with clinical trials in progress.[84] However, treatment can be considered on an individual basis after a patient–physician discussion about potential risks and benefits.

What counseling should an adolescent with HIV receive to reduce the risk of HCV transmission?

All adults and adolescents with HIV should be counseled to avoid injection drug use. If using drugs, they should—

- Avoid reusing and/or sharing needles,
- Be tested for HCV infection and hepatitis B virus (HBV) infection at a frequency aligned with their ongoing exposure, *and*
- Receive appropriate referral and therapy for substance use disorder **(strong, high).**

Unprotected sex has been linked to HCV transmission, so adolescents with HIV and those with multiple sex partners or sexually transmitted infections should be advised to use barrier protection ***(strong, moderate).***

Other potential exposures to HCV, such as tattooing and body piercing, should also be avoided ***(weak, low).***

No HCV preventative or prophylactic vaccine is available. IDU is the most reported risk factor for HCV infection in the United States. Increased IDU among rural and urban adolescents and young adults has fueled increases of new infections among individuals aged <40 in the United States since

2006, with the highest rates in nonurban communities. This change in epidemiology prompted the current recommendation for one-time screening of all adults 18 years and older and for pregnant women during each pregnancy.[85] Although adolescents were not included in the recommendation, adolescents are among those with IDU and often have been diagnosed with hepatitis C when they are admitted to the hospital after an overdose.[86] Given these circumstances, it is appropriate to adapt HCV screening and testing to younger populations or more frequent intervals based on ongoing risk of HCV exposure.

Multiple outbreaks of HCV infection have been reported among MSM with HIV who do not use injection drugs, linked most strongly with inconsistent condom use and other high-risk practices, including having sex while intoxicated on drugs and having multiple sexual partners. In the era of the message "Undetectable = Untransmittable" or U=U, education about the need to continue to use barrier protection to protect against sexual acquisition of HCV, regardless of HIV status, is important.

Preventing the First Episode of Disease

For children with HIV/HCV coinfection, which vaccinations to reduce risk of liver disease are available?

Hepatitis A and hepatitis B vaccines are recommended for all children, including those with hepatitis C with or without HIV coinfection, with follow-up serologic confirmation of vaccine response ***(strong, low).***

Patients with chronic liver disease, HIV, or both can develop fulminant hepatitis from hepatitis A virus (HAV) or HBV infection.[87] Patients with advanced HCV-related liver disease, HIV, or both may not mount an appropriate immune response to vaccines.[88] Therefore, measurement of HAV and HBV antibody titers 1 to 2 months after completion of the vaccination series is recommended. For more information, see the Hepatitis B Virus section of the Pediatric Opportunistic Infections Guidelines.

Treatment Recommendations

For all children with hepatitis C and children with HIV/HCV coinfection, what are the indications for HCV treatment?

All children aged ≥3 years should receive treatment with an approved pangenotypic DAA regimen regardless of disease severity ***(strong, moderate).***

With prior interferon-based HCV treatment or early versions of DAA regimens, concerns about cost, side effects, and complicated administration prevented these agents from being used for widespread treatment. Currently available pangenotypic DAA regimens can cure virtually all patients with HCV mono-infection and HIV/HCV coinfection; therefore, treatment should be offered to all eligible patients regardless of whether they were previously treated. Evaluation for DAA treatment has been simplified with fewer laboratory tests required prior to initiation.

For more information about evaluation for and monitoring on DAA treatment, visit the AASLD/IDSA HIV guidance on HCV in children.

For all children with hepatitis C, what FDA-approved medications are available for those requiring treatment?

Either of the FDA-approved pangenotypic DAA regimens (sofosbuvir/velpatasvir or glecaprevir/pibrentasvir) can be used for treatment ***(strong, high).***

Two pangenotypic DAA regimens are now approved for use in children as young as 3 years old: sofosbuvir/velpatasvir and glecaprevir/pibrentasvir. Both agents offer similar efficacy in curing HCV infection, and preference for one versus the other should be based on whichever agent is most easily obtained from public or private insurers for the individual patient.

For more information about dosing by age and weight, visit the AASLD/IDSA HIV guidance on HCV in children.

When treating children with HIV/HCV coinfection, are there significant drug–drug interactions between HCV and HIV treatment regimens that require consideration?

Clinically relevant drug–drug interactions have been identified between current FDA-approved pediatric DAA regimens and multiple classes of ARV drugs that may warrant alternate therapy, dose adjustment, or extra monitoring. Referral to current AASLD/IDSA HCV guidance is recommended ***(strong, high).***

Because treatment of HIV/HCV coinfection includes the challenge of dosing separate multidrug regimens, many studies have examined potential drug interactions. The Patients With HIV/HCV Coinfection section of the AASLD/IDSA HCV Guidance offers a detailed discussion of these potential interactions and should be consulted when considering treatment of any kind. This section includes a summary table to quickly screen for potential drug interactions between HCV DAAs and HIV ARVs. In addition, a summary table on the same webpage offers a quick resource for screening for potential drug interactions.

A few key considerations include the following:

- Velpatasvir can increase concentrations of tenofovir disoproxil fumarate and lead to tenofovir-related renal toxicity. It is unclear at this time whether this is also the case with tenofovir alafenamide. Nevertheless, care should be taken in those patients with reduced creatinine clearance.[89]
- Concentrations of velpatasvir, glecaprevir, and pibrentasvir are significantly reduced when co-administered with efavirenz.[90,91] Thus, both currently approved pediatric pangenotypic DAA regimens (sofosbuvir/velpatasvir and glecaprevir/pibrentasvir) are to be avoided in children taking non-nucleoside reverse transcriptase inhibitors, including efavirenz, etravirine, or nevirapine. No interactions are expected with rilpivirine with either pangenotypic regimen.
- Glecaprevir co-administration is contraindicated with atazanavir and not recommended with ritonavir-boosted ART regimens due to elevated glecepravir concentrations.

Dosing Recommendations and Important Considerations for HCV Antiviral Therapy in Children and Adolescents With HIV

- The American Association for the Study of Liver Diseases (AASLD) and the Infectious Diseases Society of America (IDSA) have developed a web-based process for the rapid formulation and dissemination of evidence-based, expert-developed recommendations for hepatitis C virus (HCV) management. See the AASLD/IDSA HCV Guidance: Recommendations for Testing, Managing, and Treating Hepatitis C for more details.
- For detailed dosing recommendations for HCV antiviral therapy in children and adolescents, refer to the section on HCV in Children.
- For more information on other important considerations in the management of HCV in children and adolescents with HIV—such as drug–drug interactions, alternate therapies, dose adjustment, and extra monitoring—refer to the section on Patients With HIV/HCV Coinfection.

References

1. Alter MJ, Kruszon-Moran D, Nainan OV, et al. The prevalence of hepatitis C virus infection in the United States, 1988 through 1994. *N Engl J Med*. 1999;341(8):556-562. Available at: http://www.ncbi.nlm.nih.gov/pubmed/10451460.

2. El-Kamary SS, Serwint JR, Joffe A, Santosham M, Duggan AK. Prevalence of hepatitis C virus infection in urban children. *J Pediatr*. 2003;143(1):54-59. Available at: http://www.ncbi.nlm.nih.gov/pubmed/12915824.

3. Denniston MM, Jiles RB, Drobeniuc J, et al. Chronic hepatitis C virus infection in the United States, National Health and Nutrition Examination Survey 2003 to 2010. *Ann Intern Med*. 2014;160(5):293-300. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24737271.

4. Schmelzer J, Dugan E, Blach S, et al. Global prevalence of hepatitis C virus in children in 2018: a modelling study. *Lancet Gastroenterol Hepatol*. 2020;5(4):374-392. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31954439.

5. Ly KN, Jiles RB, Teshale EH, Foster MA, Pesano RL, Holmberg SD. Hepatitis C virus infection among reproductive-aged women and children in the United States, 2006 to 2014. *Ann Intern Med*. 2017;166(11):775-782. Available at: https://www.ncbi.nlm.nih.gov/pubmed/28492929.

6. Schuval S, Van Dyke RB, Lindsey JC, et al. Hepatitis C prevalence in children with perinatal human immunodeficiency virus infection enrolled in a long-term follow-up protocol. *Arch Pediatr Adolesc Med*. 2004;158(10):1007-1013. Available at: http://www.ncbi.nlm.nih.gov/pubmed/15466691.

7. Abera B, Zenebe Y, Mulu W, Kibret M, Kahsu G. Seroprevalence of hepatitis B and C viruses and risk factors in HIV infected children at the Felgehiwot referral hospital, Ethiopia. *BMC Res Notes*. 2014;7:838. Available at: http://www.ncbi.nlm.nih.gov/pubmed/25421947.

8. Eze JC, Ibeziako NS, Ikefuna AN, Nwokoye IC, Uleanya ND, Ilechukwu GC. Prevalence and risk factors for hepatitis C and human immunodeficiency virus coinfection among children in Enugu, Nigeria. *Afr J Infect Dis*. 2014;8(1):5-8. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24653810.

9. England K, Thorne C, Newell ML. Vertically acquired paediatric coinfection with HIV and hepatitis C virus. *Lancet Infect Dis*. 2006;6(2):83-90. Available at: http://www.ncbi.nlm.nih.gov/pubmed/16439328.

10. Tajiri H, Miyoshi Y, Funada S, et al. Prospective study of mother-to-infant transmission of hepatitis C virus. *Pediatr Infect Dis J*. 2001;20(1):10-14. Available at: http://www.ncbi.nlm.nih.gov/pubmed/11176560.

11. Jara P, Resti M, Hierro L, et al. Chronic hepatitis C virus infection in childhood: clinical patterns and evolution in 224 white children. *Clin Infect Dis*. 2003;36(3):275-280. Available at: http://www.ncbi.nlm.nih.gov/pubmed/12539067.

12. Murray KF, Richardson LP, Morishima C, Owens JW, Gretch DR. Prevalence of hepatitis C virus infection and risk factors in an incarcerated juvenile population: a pilot study. *Pediatrics*. 2003;111(1):153-157. Available at: http://www.ncbi.nlm.nih.gov/pubmed/12509569.

13. Tovo PA, Palomba E, Ferraris G, et al. Increased risk of maternal-infant hepatitis C virus transmission for women coinfected with human immunodeficiency virus type 1. Italian Study Group for HCV Infection in Children. *Clin Infect Dis*. 1997;25(5):1121-1124. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9402369.

14. Zanetti AR, Tanzi E, Romano L, et al. A prospective study on mother-to-infant transmission of hepatitis C virus. *Intervirology*. 1998;41(4-5):208-212. Available at: http://www.ncbi.nlm.nih.gov/pubmed/10213898.

15. Gibb DM, Goodall RL, Dunn DT, et al. Mother-to-child transmission of hepatitis C virus: evidence for preventable peripartum transmission. *Lancet*. 2000;356(9233):904-907. Available at: https://pubmed.ncbi.nlm.nih.gov/11036896.

16. European Paediatric Hepatitis C Virus Network. Effects of mode of delivery and infant feeding on the risk of mother-to-child transmission of hepatitis C virus. European Paediatric Hepatitis C Virus Network. *BJOG*. 2001;108(4):371-377. Available at: http://www.ncbi.nlm.nih.gov/pubmed/11305543.

17. Granovsky MO, Minkoff HL, Tess BH, et al. Hepatitis C virus infection in the mothers and infants cohort study. *Pediatrics*. 1998;102(2 Pt 1):355-359. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9685438.

18. Resti M, Azzari C, Mannelli F, et al. Mother to child transmission of hepatitis C virus: prospective study of risk factors and timing of infection in children born to women seronegative for HIV-1. Tuscany Study Group on Hepatitis C Virus Infection. *BMJ*. 1998;317(7156):437-441. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9703524.

19. Thomas SL, Newell ML, Peckham CS, Ades AE, Hall AJ. A review of hepatitis C virus (HCV) vertical transmission: risks of transmission to infants born to mothers with and without HCV viraemia or human immunodeficiency virus infection. *Int J Epidemiol*. 1998;27(1):108-117. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9563703.

20. Mazza C, Ravaggi A, Rodella A, et al. Prospective study of mother-to-infant transmission of hepatitis C virus (HCV) infection. Study Group for Vertical Transmission. *J Med Virol*. 1998;54(1):12-19. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9443104.

21. McMenamin MB, Jackson AD, Lambert J, et al. Obstetric management of hepatitis C-positive mothers: analysis of vertical transmission in 559 mother-infant pairs. *Am J Obstet Gynecol*. 2008;199(3):315 e311-315. Available at: http://www.ncbi.nlm.nih.gov/pubmed/18771997.

22. Okamoto M, Nagata I, Murakami J, et al. Prospective reevaluation of risk factors in mother-to-child transmission of hepatitis C virus: high virus load, vaginal delivery, and negative anti-NS4 antibody. *J Infect Dis*. 2000;182(5):1511-1514. Available at: http://www.ncbi.nlm.nih.gov/pubmed/11023474.

23. Dal Molin G, D'Agaro P, Ansaldi F, et al. Mother-to-infant transmission of hepatitis C virus: rate of infection and assessment of viral load and IgM anti-HCV as risk factors. *J Med Virol*. 2002;67(2):137-142. Available at: http://www.ncbi.nlm.nih.gov/pubmed/11992574.

24. Mast EE, Hwang LY, Seto DS, Nolte FS, Nainan OV, Wurtzel H, et al. Risk factors for perinatal transmission of hepatitis C virus (HCV) and the natural history of HCV infection acquired in infancy. *J Infect Dis*. 2005;192(11):1880-1889. Available at: https://www.ncbi.nlm.nih.gov/pubmed/16267758.

25. Ruiz-Extremera A, Salmeron J, Torres C, et al. Follow-up of transmission of hepatitis C to babies of human immunodeficiency virus-negative women: the role of breast-feeding in transmission. *Pediatr Infect Dis J*. 2000;19(6):511-516. Available at: http://www.ncbi.nlm.nih.gov/pubmed/10877164.

26. Polis CB, Shah SN, Johnson KE, Gupta A. Impact of maternal HIV coinfection on the vertical transmission of hepatitis C virus: a meta-analysis. *Clin Infect Dis*. 2007;44(8):1123-1131. Available at: https://www.ncbi.nlm.nih.gov/pubmed/17366462.

27. Shebl FM, El-Kamary SS, Saleh DA, et al. Prospective cohort study of mother-to-infant infection and clearance of hepatitis C in rural Egyptian villages. *J Med Virol*. 2009;81(6):1024-1031. Available at: http://www.ncbi.nlm.nih.gov/pubmed/19382251.

28. Mok J, Pembrey L, Tovo PA, Newell ML, European Paediatric Hepatitis C Virus Network. When does mother to child transmission of hepatitis C virus occur? *Arch Dis Child Fetal Neonatal Ed*. 2005;90(2):F156-160. Available at: http://www.ncbi.nlm.nih.gov/pubmed/15724041.

29. Conte D, Fraquelli M, Prati D, Colucci A, Minola E. Prevalence and clinical course of chronic hepatitis C virus (HCV) infection and rate of HCV vertical transmission in a cohort of 15,250 pregnant women. *Hepatology*. 2000;31(3):751-755. Available at: http://www.ncbi.nlm.nih.gov/pubmed/10706568.

30. Papaevangelou V, Pollack H, Rochford G, et al. Increased transmission of vertical hepatitis C virus (HCV) infection to human immunodeficiency virus (HIV)-infected infants of HIV- and HCV-coinfected women. *J Infect Dis*. 1998;178(4):1047-1052. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9806033.

31. Marine-Barjoan E, Berrebi A, Giordanengo V, et al. HCV/HIV co-infection, HCV viral load and mode of delivery: risk factors for mother-to-child transmission of hepatitis C virus? *AIDS*. 2007;21(13):1811-1815. Available at: http://www.ncbi.nlm.nih.gov/pubmed/17690581.

32. European Paediatric Hepatiis C Virus Network. A significant sex--but not elective cesarean section--effect on mother-to-child transmission of hepatitis C virus infection. *J Infect Dis*. 2005;192(11):1872-1879. Available at: https://www.ncbi.nlm.nih.gov/pubmed/16267757.

33. Ghamar Chehreh ME, Tabatabaei SV, Khazanehdari S, Alavian SM. Effect of cesarean section on the risk of perinatal transmission of hepatitis C virus from HCV-RNA+/HIV-mothers: a meta-analysis. *Arch Gynecol Obstet*. 2011;283(2):255-260. Available at: http://www.ncbi.nlm.nih.gov/pubmed/20652289.

34. Lin HH, Kao JH, Hsu HY, et al. Absence of infection in breast-fed infants born to hepatitis C virus-infected mothers. *J Pediatr*. 1995;126(4):589-591. Available at: http://www.ncbi.nlm.nih.gov/pubmed/7535353.

35. Thomas DL, Villano SA, Riester KA, et al. Perinatal transmission of hepatitis C virus from human immunodeficiency virus type 1-infected mothers. Women and Infants Transmission Study. *J Infect Dis*. 1998;177(6):1480-1488. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9607823.

36. Pappalardo BL. Influence of maternal human immunodeficiency virus (HIV) co-infection on vertical transmission of hepatitis C virus (HCV): a meta-analysis. *Int J Epidemiol*. 2003;32(5):727-734. Available at: http://www.ncbi.nlm.nih.gov/pubmed/14559740.

37. Hershow RC, Riester KA, Lew J, et al. Increased vertical transmission of human immunodeficiency virus from hepatitis C virus-coinfected mothers. Women and Infants Transmission Study. *J Infect Dis*. 1997;176(2):414-420. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9237706.

38. Giovannini M, Tagger A, Ribero ML, et al. Maternal-infant transmission of hepatitis C virus and HIV infections: a possible interaction. *Lancet*. 1990;335(8698):1166. Available at: http://www.ncbi.nlm.nih.gov/pubmed/1971901.

39. Nigro G, D'Orio F, Catania S, et al. Mother to infant transmission of coinfection by human immunodeficiency virus and hepatitis C virus: prevalence and clinical manifestations. *Arch Virol*. 1997;142(3):453-457. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9349291.

40. Checa Cabot CA, Stoszek SK, Quarleri J, et al. Mother-to-child transmission of hepatitis C virus (HCV) among HIV/HCV-coinfected women. *J Pediatric Infect Dis Soc*. 2013;2(2):126-135. Available at: https://academic.oup.com/jpids/article/2/2/126/915058#google_vignette.

41. Prasad M, Saade GR, Clifton RG, et al. Risk factors for perinatal transmission of hepatitis C virus. *Obstet Gynecol*. 2023;142(3):449-456. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37590978.

42. Centers for Disease Control and Prevention. Viral hepatitis surveillance report – United States, 2021. 2023. Available at: https://www.cdc.gov/hepatitis/statistics/2021surveillance/index.htm.

43. Suryaprasad AG, White JZ, Xu F, et al. Emerging epidemic of hepatitis C virus infections among young nonurban persons who inject drugs in the United States, 2006–2012. *Clin Infect Dis*. 2014;59(10):1411-1419. Available at: http://www.ncbi.nlm.nih.gov/pubmed/25114031.

44. Centers for Disease Control and Prevention. Hepatitis C virus infection among adolescents and young adults: Massachusetts, 2002–2009. *MMWR*. 2011;60(17):537-541. Available at: http://www.ncbi.nlm.nih.gov/pubmed/21544042.

45. Bravo MJ, Vallejo F, Barrio G, et al. HCV seroconversion among never-injecting heroin users at baseline: no predictors identified other than starting injection. *Int J Drug Policy*. 2012;23(5):415-419. Available at: http://www.ncbi.nlm.nih.gov/pubmed/22421554.

46. Wandeler G, Gsponer T, Bregenzer A, et al. Hepatitis C virus infections in the Swiss HIV Cohort Study: a rapidly evolving epidemic. *Clin Infect Dis*. 2012;55(10):1408-1416. Available at: http://www.ncbi.nlm.nih.gov/pubmed/22893583.

47. Williams IT, Bell BP, Kuhnert W, Alter MJ. Incidence and transmission patterns of acute hepatitis C in the United States, 1982–2006. *Arch Intern Med*. 2011;171(3):242-248. Available at: http://www.ncbi.nlm.nih.gov/pubmed/21325115.

48. Linas BP, Wong AY, Schackman BR, Kim AY, Freedberg KA. Cost-effective screening for acute hepatitis C virus infection in HIV-infected men who have sex with men. *Clin Infect Dis*. 2012;55(2):279-290. Available at: http://www.ncbi.nlm.nih.gov/pubmed/22491339.

49. Witt MD, Seaberg EC, Darilay A, et al. Incident hepatitis C virus infection in men who have sex with men: a prospective cohort analysis, 1984–2011. *Clin Infect Dis*. 2013;57(1):77-84. Available at: http://www.ncbi.nlm.nih.gov/pubmed/23532480.

50. Garg S, Taylor LE, Grasso C, Mayer KH. Prevalent and incident hepatitis C virus infection among HIV-infected men who have sex with men engaged in primary care in a Boston community health center. *Clin Infect Dis*. 2013;56(10):1480-1487. Available at: http://www.ncbi.nlm.nih.gov/pubmed/23386630.

51. Aach RD, Yomtovian RA, Hack M. Neonatal and pediatric posttransfusion hepatitis C: a look back and a look forward. *Pediatrics*. 2000;105(4 Pt 1):836-842. Available at: http://www.ncbi.nlm.nih.gov/pubmed/10742329.

52. Mohan P, Colvin C, Glymph C, et al. Clinical spectrum and histopathologic features of chronic hepatitis C infection in children. *J Pediatr*. 2007;150(2):168-174, 174 e161. Available at: http://www.ncbi.nlm.nih.gov/pubmed/17236895.

53. Davison SM, Mieli-Vergani G, Sira J, Kelly DA. Perinatal hepatitis C virus infection: diagnosis and management. *Arch Dis Child*. 2006;91(9):781-785. Available at: http://www.ncbi.nlm.nih.gov/pubmed/16923861.

54. Resti M, Jara P, Hierro L, et al. Clinical features and progression of perinatally acquired hepatitis C virus infection. *J Med Virol*. 2003;70(3):373-377. Available at: http://www.ncbi.nlm.nih.gov/pubmed/12766999.

55. Tovo PA, Pembrey LJ, Newell ML. Persistence rate and progression of vertically acquired hepatitis C infection. European Paediatric Hepatitis C Virus Infection. *J Infect Dis*. 2000;181(2):419-424. Available at: http://www.ncbi.nlm.nih.gov/pubmed/10669321.

56. England K, Thorne C, Harris H, Ramsay M, Newell ML. The impact of mode of acquisition on biological markers of paediatric hepatitis C virus infection. *J Viral Hepat*. 2011;18(8):533-541. Available at: http://www.ncbi.nlm.nih.gov/pubmed/21762285.

57. European Paediatric Hepatitis C Virus Network. Three broad modalities in the natural history of vertically acquired hepatitis C virus infection. *Clin Infect Dis*. 2005;41(1):45-51. Available at: http://www.ncbi.nlm.nih.gov/pubmed/15937762.

58. Ades AE, Gordon F, Scott K, et al. Spontaneous clearance of vertically acquired hepatitis C infection: implications for testing and treatment. *Clin Infect Dis*. 2023;76(5):913-991. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35396848.

59. Goodman ZD, Makhlouf HR, Liu L, et al. Pathology of chronic hepatitis C in children: liver biopsy findings in the Peds-C Trial. *Hepatology*. 2008;47(3):836-843. Available at: http://www.ncbi.nlm.nih.gov/pubmed/18167062.

60. Graham CS, Baden LR, Yu E, et al. Influence of human immunodeficiency virus infection on the course of hepatitis C virus infection: a meta-analysis. *Clin Infect Dis*. 2001;33(4):562-569. Available at: http://www.ncbi.nlm.nih.gov/pubmed/11462196.

61. Merchante N, Giron-Gonzalez JA, Gonzalez-Serrano M, et al. Survival and prognostic factors of HIV-infected patients with HCV-related end-stage liver disease. *AIDS*. 2006;20(1):49-57. Available at: http://www.ncbi.nlm.nih.gov/pubmed/16327319.

62. Mehta SH, Thomas DL, Torbenson M, Brinkley S, Mirel L, Chaisson RE, et al. The effect of antiretroviral therapy on liver disease among adults with HIV and hepatitis C coinfection. *Hepatology* 2005;41(1):123-131. Available at: https://www.ncbi.nlm.nih.gov/pubmed/15619237.

63. Qurishi N, Kreuzberg CL, Luchters G, et al. Effect of antiretroviral therapy on liver-related mortality in patients with HIV and hepatitis C coinfection. *Lancet*. 2004;362(9397):1708-1713. Available at: https://www.ncbi.nlm.nih.gov/pubmed/14643119.

64. Claret-Teruel G, Noguera-Julian A, Esteva C, et al. Impact of human immunodeficiency virus coinfection on the progression of mother-to-child transmitted hepatitis C virus infection. *Pediatr Infect Dis J*. 2011;30(9):801-804. Available at: http://www.ncbi.nlm.nih.gov/pubmed/21772231.

65. Shivraj SO, Chattopadhya D, Grover G, Kumar A, Baveja UK. Role of HCV coinfection towards disease progression and survival in HIV-1 infected children: a follow-up study of 10 years. *J Trop Pediatr*. 2006;52(3):206-211. Available at: http://www.ncbi.nlm.nih.gov/pubmed/16339160.

66. Dufour DR, Lott JA, Nolte FS, Gretch DR, Koff RS, Seeff LB. Diagnosis and monitoring of hepatic injury. I. Performance characteristics of laboratory tests. *Clin Chem*. 2000;46(12):2027-2049. Available at: https://www.ncbi.nlm.nih.gov/pubmed/11106349.

67. U.S. Food and Drug Administration. Oraquick HCV rapid antibody test. Premarket approval [press release]. 2021. Available at: https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfpma/pma.cfm?id=P080027

68. U.S. Food and Drug Administration. Summary of safety and effectiveness data. Antibody to Hepatitis C Virus (Anti-HCV) Rapid Assay [press release]. 2010. Available at: https://www.accessdata.fda.gov/cdrh_docs/pdf8/P080027b.pdf

69. Kamili S, Drobeniuc J, Araujo AC, Hayden TM. Laboratory diagnostics for hepatitis C virus infection. *Clin Infect Dis*. 2012;55 Suppl 1:S43-48. Available at: http://www.ncbi.nlm.nih.gov/pubmed/22715213.

70. Schwarz KB, Gonzalez-Peralta RP, Murray KF, et al. The combination of ribavirin and peginterferon is superior to peginterferon and placebo for children and adolescents with chronic hepatitis C. *Gastroenterology*. 2011;140(2):450-458 e451. Available at: http://www.ncbi.nlm.nih.gov/pubmed/21036173.

71. Rumbo C, Fawaz RL, Emre SH, et al. Hepatitis C in children: a quaternary referral center perspective. *J Pediatr Gastroenterol Nutr*. 2006;43(2):209-216. Available at: https://pubmed.ncbi.nlm.nih.gov/16877987.

72. Epclusa [product insert]. Gilead Sciences. 2022. Available at: https://hcp.epclusa.com.

73. Jonas MM, Squires RH, Rhee SM, et al. Pharmacokinetics, safety, and efficacy of glecaprevir/pibrentasvir in adolescents with chronic hepatitis C virus: part 1 of the DORA study. *Hepatology*. 2020;71(2):456-462. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31254392.

74. Jonas MM, Rhee S, Kelly DA, et al. Pharmacokinetics, safety, and efficacy of glecaprevir/pibrentasvir in children with chronic HCV: part 2 of the DORA study. *Hepatology*. 2021;74(1):19-27. Available at: https://www.ncbi.nlm.nih.gov/pubmed/33811356.

75. Dunn DT, Gibb DM, Healy M, et al. Timing and interpretation of tests for diagnosing perinatally acquired hepatitis C virus infection. *Pediatr Infect Dis J*. 2001;20(7):715-716. Available at: http://www.ncbi.nlm.nih.gov/pubmed/11465848.

76. Polywka S, Pembrey L, Tovo PA, Newell ML. Accuracy of HCV-RNA PCR tests for diagnosis or exclusion of vertically acquired HCV infection. *J Med Virol*. 2006;78(2):305-310. Available at: http://www.ncbi.nlm.nih.gov/pubmed/16372293.

77. Gowda C, Smith S, Crim L, Moyer K, Sanchez PJ, Honegger JR. Nucleic acid testing for diagnosis of perinatally acquired hepatitis C virus infection in early infancy. *Clin Infect Dis*. 2021;73(9):e3340-e3346. Available at: https://www.ncbi.nlm.nih.gov/pubmed/32640018.

78. Cottrell EB, Chou R, Wasson N, Rahman B, Guise JM. Reducing risk for mother-to-infant transmission of hepatitis C virus: a systematic review for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2013;158(2):109-113. Available at: https://www.ncbi.nlm.nih.gov/pubmed/23437438.

79. Garcia-Tejedor A, Maiques-Montesinos V, Diago-Almela VJ, et al. Risk factors for vertical transmission of hepatitis C virus: a single center experience with 710 HCV-infected mothers. *Eur J Obstet Gynecol Reprod Biol*. 2015;194:173-177. Available at: https://www.ncbi.nlm.nih.gov/pubmed/26409061.

80. Abuogi L, Noble L, Smith C, Committee On P, Adolescent HIV, Section On B. Infant Feeding for Persons Living With and at Risk for HIV in the United States: Clinical Report. *Pediatrics*. 2024;153(6). Available at: https://www.ncbi.nlm.nih.gov/pubmed/38766700.

81. U. S. Food and Drug Administration. Rebetol [package insert]. Available at: https://www.merck.com/product/usa/pi_circulars/p/pegintron/pegintron_5ml_pi.pdf

82. Yattoo GN. Treatment of chronic hepatitis C with ledipasvir/sofosbuvir combination during pregnancy. *Hepatol Int.* 2018;12:S292-S293. Available at: https://www.hcvguidelines.org/references/yattoo-2018.

83. Chappell CA, Scarsi KK, Kirby BJ, et al. Ledipasvir plus sofosbuvir in pregnant women with hepatitis C virus infection: a phase 1 pharmacokinetic study. *Lancet Microbe*. 2020;1(5):e200-208. Available at: https://www.hcvguidelines.org/references/chappell-2020.

84. Kushner T, Lange M, Sperling R, Dieterich D. Treatment of women with hepatitis C diagnosed in pregnancy: a co-located treatment approach. *Gastroenterology*. 2022;163(5):1454-1456 e1451. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35863531.

85. Schillie S, Wester C, Osborne M, Wesolowski L, Ryerson AB. CDC recommendations for hepatitis C screening among adults - United States, 2020. *MMWR Recomm Rep*. 2020;69(2):1-17. Available at: https://www.ncbi.nlm.nih.gov/pubmed/32271723.

86. Barritt ASt, Lee B, Runge T, Schmidt M, Jhaveri R. Increasing prevalence of hepatitis C among hospitalized children is associated with an increase in substance abuse. *J Pediatr*. 2018;192:159-164. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29106926.

87. Rubin LG, Levin MJ, Ljungman P, et al. 2013 IDSA clinical practice guideline for vaccination of the immunocompromised host. *Clin Infect Dis*. 2014;58(3):309-318. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24421306.

88. Kramer ES, Hofmann C, Smith PG, Shiffman ML, Sterling RK. Response to hepatitis A and B vaccine alone or in combination in patients with chronic hepatitis C virus and advanced fibrosis. *Dig Dis Sci*. 2009;54(9):2016-2025. Available at: http://www.ncbi.nlm.nih.gov/pubmed/19517231.

89. Osinusi A, Townsend K, Kohli A, et al. Virologic response following combined ledipasvir and sofosbuvir administration in patients with HCV genotype 1 and HIV co-infection. *JAMA*. 2015;313(12):1232-1239. Available at: http://www.ncbi.nlm.nih.gov/pubmed/25706232.

90. Kosloski MP, Oberoi R, Wang S, et al. Drug–drug interactions of glecaprevir and pibrentasvir coadministered with human immunodeficiency virus antiretrovirals. *J Infect Dis*. 2020;221(2):223-231. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31504702.

91. Mogalian E, Stamm LM, Osinusi A, et al. Drug–drug interaction studies between hepatitis C virus antivirals sofosbuvir/velpatasvir and boosted and unboosted human immunodeficiency virus antiretroviral regimens in healthy volunteers. *Clin Infect Dis*. 2018;67(6):934-940. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29522076