Rubella in pregnancy

Author: Laura E Riley, MD
Section Editors: Martin S Hirsch, MD, Charles J Lockwood, MD, MHCM
Deputy Editor: Allyson Bloom, MD

All topics are updated as new evidence becomes available and our peer review process is complete.
Literature review current through: May 2017. | **This topic last updated:** Feb 18, 2016.

INTRODUCTION — Rubella, also known as German measles, was a disease of childhood that has markedly declined in incidence in the United States since the introduction of routine childhood rubella vaccination. This virus causes a self-limited infection in most hosts, but can have potentially devastating effects on the developing fetus. Screening for antibodies to rubella is routinely performed by obstetricians.

Rubella virus is a member of the togavirus family, genus Rubivirus, and humans are the only reservoir for rubella infection. The virus is transmitted by direct droplet contact from nasopharyngeal secretions, replicates in the lymph tissue of the upper respiratory tract, and spreads hematogenously. Congenital infection occurs when maternal viremia allows hematogenous spread of the virus across the placenta.

Rubella in pregnancy will be reviewed here. The virology, pathogenesis, clinical manifestations, diagnosis, treatment, and prevention through vaccination are discussed separately. (See "Rubella".)

EPIDEMIOLOGY — Rubella and the congenital rubella syndrome (CRS) have largely been eliminated in the United States. The incidence of rubella has declined from 0.45 per 100,000 in 1990 to 0.1 per 100,000 in 1999 [1]. However, rubella outbreaks continue to occur in other parts of the world, and CRS remains a concern.

United States — Prior to the introduction of the rubella vaccine in 1969, epidemics of rubella occurred in six to nine year cycles, usually in the late winter and early spring. In 1964, a major worldwide pandemic spread to the United States resulting in approximately 12.5 million cases of rubella, as many as 11,000 fetal deaths, and approximately 20,000 cases of congenital rubella syndrome (CRS) [2].

Since the introduction of a comprehensive vaccine program, the epidemiologic evidence in 2004 suggests that rubella is no longer endemic in the United States [3]. During 1998 to 2000, the median number of reported rubella cases was 272, whereas, during 2001 to 2004, the median number reported was 13. Since 2004, CRS has rarely been reported in the United States (figure 1).

Other regions — The proportion of rubella susceptible women of childbearing age varies greatly among nations, especially the developing countries. As examples, an estimated 15 percent of women between the ages of 20 and 29 lack antibodies to rubella in Turkey [4], and 23 percent of women of childbearing age had negative titers in Nigeria [5]. In the Russian Federation, 16.5

percent of pregnant women were susceptible to rubella, and CRS occurred in approximately 3.5 cases per 1000 live births [6]. Fifteen percent of 342 infants with suspected congenital infections had detectable IgM against rubella in one study in India [7].

Rubella cases declined by 98 percent in the Americas between 1998 and 2006 (from 135,947 to 2998). In 2007, there were large outbreaks in Argentina, Brazil, and Chile, primarily in men, which prompted new vaccination campaign strategies to include men and women in an effort to eliminate rubella and CRS by 2010 [8]. Subsequent rubella outbreaks in Japan and Poland, with thousands of rubella cases and resultant congenital rubella cases, underscore the need to review the travel history and immunization history of pregnant women [9,10]. Immigrants have been the source of CRS in other developed countries, such as the United Kingdom [11] and Canada [12]. Among women living in or immigrating from developing countries, one should consider the possibility that a positive rubella screening test result at the first prenatal visit may be a sign of recent active infection, rather than evidence of immunity from old infection or vaccination [13].

Even in countries where rubella vaccination programs are available, the extent of vaccination is not always optimal. One group reported that although more than one-third of countries had a national vaccination policy, 9 percent reported a selective strategy (ie, protection to women or schoolgirls), and 31 percent reported only childhood immunization [14]. These selective strategies allow for some segments of the population to remain unvaccinated. Unvaccinated susceptibles can be a source of many preventable cases of CRS [15]. This theoretical concern was demonstrated to be of significance in Greece; vaccination coverage in the country was <50 percent and the number of susceptible pregnant women increased throughout the 1980s [16]. A major epidemic of rubella occurred in 1993; the mean age of affected patients was 17 years, and 64 percent were over the age of 15. The incidence of CRS in 1993 rose dramatically, from one to four cases per year before that time to 25 cases (24.6 cases per 100,000 live births). Similarly, in the United States, some areas note declining vaccination rates, which lead to more sporadic cases of rubella and provide a reservoir for transmission [17].

CLINICAL MANIFESTATIONS — Acquired rubella is generally a mild, self-limited disease associated with a characteristic exanthem (see "Rubella"). Symptoms appear approximately 14 to 21 days after inoculation with the virus. Though asymptomatic in 25 to 50 percent of cases, affected individuals may experience mild, prodromal symptoms consisting of low-grade fever, conjunctivitis, coryza, sore throat, cough, and occasionally headache and malaise. These symptoms usually last one to five days before the onset of the rash. Rubella may also be associated with generalized, tender lymphadenopathy, particularly involving suboccipital, postauricular, and cervical nodes, which often becomes pronounced during the rash. Just prior to the onset of the rash, approximately 20 percent of those infected will develop discrete rose spots on the soft palate (Forchheimer spots) that may later expand and coalesce.

The typical rash of rubella is an erythematous maculopapular eruption, which may be mildly pruritic and evolves into pinpoint papules similar to scarlet fever. The rash characteristically begins on the face and spreads to the trunk and extremities within hours. It lasts approximately one to three days and resolves first from the face and then from the body.

Polyarthritis and polyarthralgia are potential sequelae. Rheumatologic symptoms may develop about one week after the rash and are more commonly seen in female adolescents and adults, occurring in as many as 60 to 70 percent of adult women [18]. Classically, the hands, knees, wrists, and ankles are affected in a symmetric pattern with pain and morning stiffness for one to four weeks. Chronic arthritis rarely develops. Tenosynovitis and carpal tunnel syndrome may also

be associated with rubella. Other rare complications include: thrombocytopenia associated with purpura or hemorrhage, postinfectious encephalitis, myocarditis, pericarditis, hepatitis, hemolytic anemia, and hemolytic uremic syndrome [19,20].

Congenital rubella syndrome — Rubella infection can have catastrophic effects on the developing fetus, resulting in spontaneous abortion, fetal infection, stillbirth, or intrauterine growth restriction. This area is discussed in detail separately. (See "Congenital rubella syndrome: Clinical features and diagnosis" and "Congenital rubella syndrome: Management, outcome, and prevention".)

Maternal-fetal transmission occurs via hematogenous spread and varies with gestational age. There is considerable pathologic evidence that suggests that the rubella virus spreads through the vascular system of the developing fetus after infecting the placenta. The resulting defects stem from cytopathic damage to blood vessels and ischemia in affected organs [21]. In the first trimester, fetal infection rates as high as 81 percent have been observed, dropping to 25 percent in the late second trimester and increasing again in the third trimester from 35 percent at 27 to 30 weeks to nearly 100 percent for fetuses exposed beyond 36 weeks [22].

However, the risk of congenital defects after maternal infection is essentially limited to maternal infection in the first 16 weeks of pregnancy [23]. Little, if any, risk of CRS is associated with infection after 20 weeks' gestation, and intrauterine growth retardation may be the only sequelae of third trimester infection [22,24-26]. Similarly, there is no evidence that rubella infection immediately prior to pregnancy increases the risk of congenital infection [27].

In general, maternal immunity, either vaccine or naturally derived, is protective against intrauterine rubella infection. However, there have been CRS cases resulting from maternal reinfection [28-30]. None of these cases occurred in women infected after 12 weeks' gestation [31].

DIAGNOSIS — Serology is widely available and may be used to screen for rubella infection [32]. Enzyme linked immunoassays (ELISA) are sensitive, easy to perform, and measure rubella-specific IgG and IgM. Immunofluorescent antibody assays are also sensitive and rapid; commercial IgG and IgM assays are available. Other serologic tests include passive hemagglutination antibody (PHA), latex agglutination, complement fixation, and hemagglutination inhibition (HI). Most laboratories use ELISA due to its convenience, sensitivity, and accuracy. The clinical diagnosis of rubella should only be made in typical cases occurring during an epidemic.

Acute rubella syndrome is best diagnosed by:

- A fourfold rise in IgG titer between acute and convalescent serum specimens
- The presence of rubella specific IgM
- A positive rubella culture

Serum should be obtained within 7 to 10 days after the onset of the rash and repeated two to three weeks later. Rubella virus may be isolated from nasal, blood, throat, urine, or cerebrospinal fluid (CSF) specimens. The virus is generally isolated from the pharynx one week before to two weeks after the rash. Viral isolates can be typed to facilitate surveillance during outbreaks [33].

If rubella IgM is incidentally detected in a pregnant woman in the absence of a history of rubella-like illness or contact, further investigation is required. In persons with no or low risk of exposure to rubella, the reactive IgM is likely falsely positive due to rheumatoid factor or other antibodies to infection which can cross react with the assay. Use of rubella specific avidity assay may be useful

in these situations. Because of issues of false-positivity, the Centers for Disease Control and Prevention in the United States discourages the use of rubella IgM for rubella screening in pregnancy [34].

Prenatal diagnosis — Polymerase chain reaction (PCR) is another option for providing presumptive diagnosis of rubella infection. This method has been used extensively in the United Kingdom for detection of rubella virus in clinical specimens [35]. There are small series reporting the usefulness of PCR for prenatal diagnosis [36,37]. A reverse transcription-nested PCR assay has been used in small studies where it detects rubella virus in chorionic villous samples (CVS) and amniotic fluid samples of affected pregnancies. The largest study to date reported 34 cases where PCR detection of rubella was better in CVS samples than amniotic fluid samples [38]. A case report of maternal primary rubella in the second trimester also showed that amniotic fluid was negative by PCR at both 19 and 23 weeks, while fetal blood was positive at 23 weeks [39]. Furthermore, rubella IgM on fetal blood was negative in eight infected cases, but PCR was positive in all eight. Although the numbers are small, it would appear that rubella specific PCR on CVS samples may be superior to standard serologic testing on fetal blood. In addition, CVS sampling ideally done at 10 to 12 weeks' gestation would allow for earlier detection than other samples, such as fetal blood obtained at 18 to 20 weeks' gestation.

Ultrasound diagnosis of an affected fetus would be extremely difficult given the nature of the malformations seen with CRS, although, the workup of any fetus with intrauterine growth restriction should include evaluation for congenital viral infections including rubella.

TREATMENT — Treatment for acute rubella infection may include acetaminophen for symptomatic relief. Glucocorticoids, platelet transfusion, and other supportive measures are reserved for patients with complications such as thrombocytopenia or encephalopathy. The prognosis for pregnant women with rubella is generally excellent.

However, because of the potentially devastating effects on the fetus, women should be counseled about maternal-fetal transmission and offered pregnancy termination, especially prior to 16 weeks' gestation. After 20 weeks' gestation, management should be individualized, and parents should be counseled about the potential for delayed consequences of rubella infection (see "Congenital rubella syndrome: Clinical features and diagnosis", section on 'Late manifestations'). There is no definitively beneficial in utero treatment available for exposed or affected fetuses.

The use of immune globulin for pregnant women with acute infection is controversial. There are no data to suggest that IgG has a beneficial effect on the fetal response to disease. Thus, the Centers for Disease Control and Prevention (CDC) recommends limiting the use of immune globulin to women with known rubella exposure who decline pregnancy termination [40].

PREVENTION — The first live-attenuated rubella vaccines were introduced in 1969. RA 27/3, a live-attenuated vaccine, is the current vaccine used in the United States (see "Rubella"). A single dose of this vaccine given at one year of age or older results in measurable antibody in almost 95 percent of susceptible persons [41-43].

Vaccination is recommended for all children at 12 to 15 months and 4 to 6 years in conjunction with measles and mumps (MMR). All other persons should be vaccinated unless immunity is documented by serology.

Postpartum vaccination programs have been shown to significantly reduce rubella susceptibility in pregnant seronegative women [44]. However, a study showed that in 2004, one-third of pregnant

women were also susceptible to rubeola or mumps [45]. The recommendation of the Centers for Disease Control and Prevention (CDC) and American Congress of Obstetricians and Gynecologists (ACOG) is that rubella susceptible women should receive MMR vaccine postpartum for protection against all of these viral pathogens [1,46].

Rubella vaccine virus may cross the placenta and infect the fetus. However, there have been no cases of CRS reported in women inadvertently vaccinated during early pregnancy [47-49]. Therefore, pregnancy termination is **not** recommended for these women [46]. Given the theoretical risks to the fetus, women had been advised to avoid pregnancy for three months following vaccination. The Advisory Committee on Immunization Practices (ACIP) has now shortened the recommended interval to 28 days after reviewing data from the United States, United Kingdom, Sweden, and Germany, which again confirmed no CRS cases in infants born to mothers who were vaccinated between two weeks before and six weeks after conception [50].

Contraindications to rubella vaccination include febrile illness, immunodeficiency disorder, history of anaphylaxis to neomycin, and pregnancy. Postpartum vaccination should be performed in all women known to be susceptible. Breastfeeding is not contraindicated. A small percentage of individuals will have side effects, such as arthritis, arthralgia, rash, adenopathy, or fever.

The effectiveness of efforts to control and prevent rubella in the United States relies upon vaccination of susceptible individuals in all clinical settings with particular attention to persons born in countries lacking a comprehensive vaccination program.

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

- Basics topic (see "Patient education: Rubella (The Basics)")

SUMMARY AND RECOMMENDATIONS

- Since the introduction of a comprehensive vaccine program, rubella and congenital rubella syndrome (CRS) have largely been eliminated in the United States. However, in other parts of the world, particularly resource-limited settings, a considerable proportion of women of childbearing age remain susceptible to rubella, and thus CRS remains a concern. (See 'Epidemiology' above.)

- Acquired rubella in the pregnant woman is generally a mild, self-limited disease characterized by an erythematous maculopapular eruption that is sometimes preceded by prodromal symptoms of low-grade fever, conjunctivitis, coryza, sore throat, and cough. Rheumatologic symptoms, including polyarthritis and arthralgias of the hands, wrists, knees, and ankles, commonly occur in women about a week following the rash. (See 'Clinical manifestations' above and "Rubella", section on 'Clinical manifestations'.)

- Rubella infection of the fetus can be catastrophic, resulting in spontaneous abortion, stillbirth, congenital defects, and intrauterine growth retardation. Maternal-fetal transmission occurs via hematogenous spread and is highest in the first trimester and after 36 weeks' gestation. However, the risk of congenital defects after maternal infection is essentially limited to maternal infection in the first 16 weeks of pregnancy. (See 'Congenital rubella syndrome' above and "Congenital rubella syndrome: Clinical features and diagnosis".)

- Immunity to rubella in pregnant women should be documented as part of initial prenatal care. In susceptible pregnant women with suggestive clinical features, acute rubella syndrome can be diagnosed by one of the following (see "Prenatal care: Initial assessment", section on 'Documentation of rubella immunity' and 'Diagnosis' above):

  - A fourfold rise in IgG titer between acute (within 7 to 10 days of rash onset) and convalescent (two to three weeks later) serum specimens

  - The presence of rubella-specific IgM

  - A positive rubella culture (from nasal, blood, throat, urine, or cerebrospinal fluid samples)

- In women with documented acute rubella infection during pregnancy, fetal infection is diagnosed by testing chorionic villous samples (CVS) and amniotic fluid samples with a rubella-specific polymerase chain reaction (PCR) assay. Ultrasound findings are not specific for the prenatal diagnosis of CRS, but finding intrauterine growth retardation should prompt evaluation for congenital viral infections, including rubella. (See 'Prenatal diagnosis' above.)

- Women should be counseled about maternal-fetal transmission and offered pregnancy termination, especially when infection is diagnosed prior to 16 weeks' gestation, due to the high risk for CRS. Otherwise, there is no definitively beneficial in utero treatment available for exposed or affected fetuses. (See 'Treatment' above.)

- Rubella vaccination can effectively protect against subsequent infection and is the best strategy to eliminate cases of CRS. The vaccine is live and may cross the placenta and theoretically infect the fetus; thus, it is contraindicated during pregnancy, and women are advised to avoid pregnancy for one month following vaccination. However, there have been no cases of CRS associated with the rubella vaccine, and thus pregnancy termination is not recommended for women who are inadvertently vaccinated during pregnancy. (See 'Prevention' above.)

REFERENCES

1. Reef SE, Frey TK, Theall K, et al. The changing epidemiology of rubella in the 1990s: on the verge of elimination and new challenges for control and prevention. JAMA 2002; 287:464.
2. Orenstein WA, Bart KJ, Hinman AR, et al. The opportunity and obligation to eliminate rubella from the United States. JAMA 1984; 251:1988.
3. Reef SE, Redd SB, Abernathy E, et al. The epidemiological profile of rubella and congenital rubella syndrome in the United States, 1998-2004: the evidence for absence of endemic transmission. Clin Infect Dis 2006; 43 Suppl 3:S126.

4. Aksit S, Egemen A, Ozacar T, et al. Rubella seroprevalence in an unvaccinated population in Izmir: recommendations for rubella vaccination in Turkey. Pediatr Infect Dis J 1999; 18:577.
5. Onyenekwe CC, Kehinde-Agbeyangi TA, Ofor US, Arinola OG. Prevalence of rubella-IgG antibody in women of childbearing age in Lagos, Nigeria. West Afr J Med 2000; 19:23.
6. Semerikov VV, Lavrentyeva IN, Popov VF, et al. Rubella in the Russian Federation: epidemiological features and control measures to prevent the congenital rubella syndrome. Epidemiol Infect 2000; 125:359.
7. Ballal M, Shivananda PG. Prevalence of rubella virus in suspected cases of congenital infections. Indian J Pediatr 1997; 64:231.
8. Centers for Disease Control and Prevention (CDC). Progress toward elimination of rubella and congenital rubella syndrome--the Americas, 2003-2008. MMWR Morb Mortal Wkly Rep 2008; 57:1176.
9. http://wwwnc.cdc.gov/travel/notices/alert/rubella-poland (Accessed on March 04, 2014).
10. http://wwwnc.cdc.gov/travel/notices/alert/rubella-japan (Accessed on March 04, 2014).
11. Sheridan E, Aitken C, Jeffries D, et al. Congenital rubella syndrome: a risk in immigrant populations. Lancet 2002; 359:674.
12. McElroy R, Laskin M, Jiang D, et al. Rates of rubella immunity among immigrant and non-immigrant pregnant women. J Obstet Gynaecol Can 2009; 31:409.
13. Mehta NM, Thomas RM. Antenatal screening for rubella-infection or immunity? BMJ 2002; 325:90.
14. Robertson SE, Cutts FT, Samuel R, Diaz-Ortega JL. Control of rubella and congenital rubella syndrome (CRS) in developing countries, Part 2: Vaccination against rubella. Bull World Health Organ 1997; 75:69.
15. Cutts FT, Vynnycky E. Modelling the incidence of congenital rubella syndrome in developing countries. Int J Epidemiol 1999; 28:1176.
16. Panagiotopoulos T, Antoniadou I, Valassi-Adam E. Increase in congenital rubella occurrence after immunisation in Greece: retrospective survey and systematic review. BMJ 1999; 319:1462.
17. Louie JK, Shaikh-Laskos R, Preas C, et al. Re-emergence of another vaccine-preventable disease?-Two cases of rubella in older adults. J Clin Virol 2009; 46:98.
18. JOHNSON RE, HALL AP. Rubella arthritis; report of cases studied by latex tests. N Engl J Med 1958; 258:743.
19. Ozsoylu S, Kanra G, Savaş G. Thrombocytopenic purpura related to rubella infection. Pediatrics 1978; 62:567.
20. Bayer WL, Sherman FE, Michaels RH, et al. Purpura in congenital and acquired rubella. N Engl J Med 1965; 273:1362.
21. Webster WS. Teratogen update: congenital rubella. Teratology 1998; 58:13.
22. Miller E, Cradock-Watson JE, Pollock TM. Consequences of confirmed maternal rubella at successive stages of pregnancy. Lancet 1982; 2:781.
23. Morgan-Capner P, Miller E, Vurdien JE, Ramsay ME. Outcome of pregnancy after maternal reinfection with rubella. CDR (Lond Engl Rev) 1991; 1:R57.

24. Grillner L, Forsgren M, Barr B, et al. Outcome of rubella during pregnancy with special reference to the 17th-24th weeks of gestation. Scand J Infect Dis 1983; 15:321.
25. Peckham C. Congenital rubella in the United Kingdom before 1970: the prevaccine era. Rev Infect Dis 1985; 7 Suppl 1:S11.
26. Peckham CS. Clinical and laboratory study of children exposed in utero to maternal rubella. Arch Dis Child 1972; 47:571.
27. Enders G, Nickerl-Pacher U, Miller E, Cradock-Watson JE. Outcome of confirmed periconceptional maternal rubella. Lancet 1988; 1:1445.
28. Bullens D, Smets K, Vanhaesebrouck P. Congenital rubella syndrome after maternal reinfection. Clin Pediatr (Phila) 2000; 39:113.
29. Paludetto R, van den Heuvel J, Stagni A, et al. Rubella embryopathy after maternal reinfection. Biol Neonate 1994; 65:340.
30. Aboudy Y, Fogel A, Barnea B, et al. Subclinical rubella reinfection during pregnancy followed by transmission of virus to the fetus. J Infect 1997; 34:273.
31. Robinson J, Lemay M, Vaudry WL. Congenital rubella after anticipated maternal immunity: two cases and a review of the literature. Pediatr Infect Dis J 1994; 13:812.
32. Best JM, O'Shea S, Tipples G, et al. Interpretation of rubella serology in pregnancy--pitfalls and problems. BMJ 2002; 325:147.
33. Frey TK, Abernathy ES. Identification of strain-specific nucleotide sequences in the RA 27/3 rubella virus vaccine. J Infect Dis 1993; 168:854.
34. Manual for the Surveillance of Vaccine-Preventable Diseases, 5th, 2012.
35. Bosma TJ, Corbett KM, O'Shea S, et al. PCR for detection of rubella virus RNA in clinical samples. J Clin Microbiol 1995; 33:1075.
36. Bosma TJ, Corbett KM, Eckstein MB, et al. Use of PCR for prenatal and postnatal diagnosis of congenital rubella. J Clin Microbiol 1995; 33:2881.
37. Ho-Terry L, Terry GM, Londesborough P. Diagnosis of foetal rubella virus infection by polymerase chain reaction. J Gen Virol 1990; 71 (Pt 7):1607.
38. Tanemura M, Suzumori K, Yagami Y, Katow S. Diagnosis of fetal rubella infection with reverse transcription and nested polymerase chain reaction: a study of 34 cases diagnosed in fetuses. Am J Obstet Gynecol 1996; 174:578.
39. Tang JW, Aarons E, Hesketh LM, et al. Prenatal diagnosis of congenital rubella infection in the second trimester of pregnancy. Prenat Diagn 2003; 23:509.
40. Rubella prevention. Recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR Recomm Rep1990; 39(RR-15):1-18.
41. Chang TW, DesRosiers S, Weinstein L. Clinical and serologic studies of an outbreak of rubella in a vaccinated population. N Engl J Med 1970; 283:246.
42. Davis WJ, Larson HE, Simsarian JP, et al. A study of rubella immunity and resistance to infection. JAMA 1971; 215:600.
43. Greaves WL, Orenstein WA, Hinman AR, Nersesian WS. Clinical efficacy of rubella vaccine. Pediatr Infect Dis 1983; 2:284.
44. Watson JC, Hadler SC, Dykewicz CA, et al. Measles, mumps, and rubella--vaccine use and strategies for elimination of measles, rubella, and congenital rubella syndrome and control of

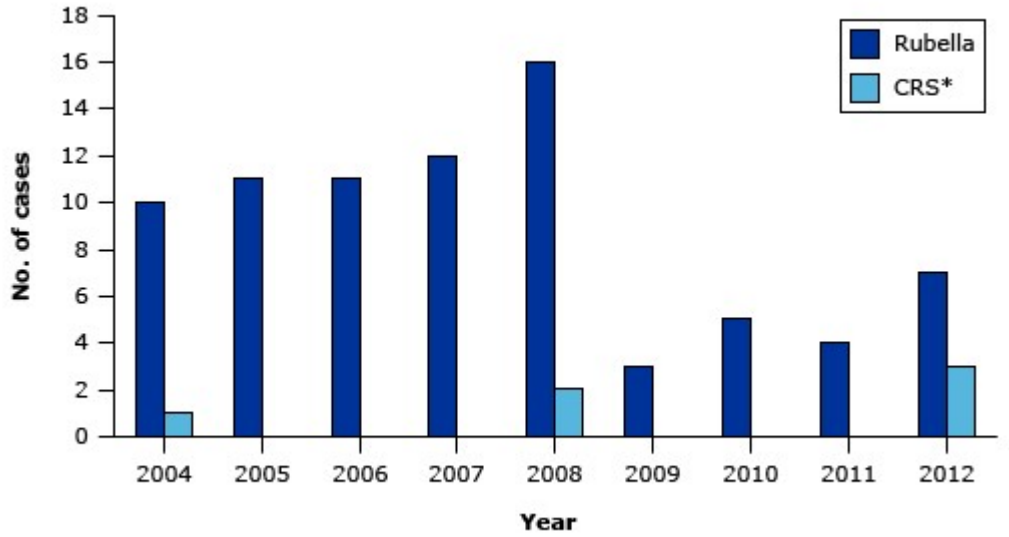
mumps: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep 1998; 47:1.

45. Haas DM, Flowers CA, Congdon CL. Rubella, rubeola, and mumps in pregnant women: susceptibilities and strategies for testing and vaccinating. Obstet Gynecol 2005; 106:295.
46. Immunization during pregnancy. American College of Obstetricians and Gynecologists Technical Bulletin, #160 October 1991. In compendium of selected publications. p. 528-537.
47. Bart SW, Stetler HC, Preblud SR, et al. Fetal risk associated with rubella vaccine: an update. Rev Infect Dis 1985; 7 Suppl 1:S95.
48. Centers for Disease Control (CDC). Rubella vaccination during pregnancy--United States, 1971-1988. MMWR Morb Mortal Wkly Rep 1989; 38:289.
49. Badilla X, Morice A, Avila-Aguero ML, et al. Fetal risk associated with rubella vaccination during pregnancy. Pediatr Infect Dis J 2007; 26:830.
50. Centers for Disease Control and Prevention (CDC). Revised ACIP recommendation for avoiding pregnancy after receiving a rubella-containing vaccine. MMWR Morb Mortal Wkly Rep 2001; 50:1117.

Topic 8312 Version 12.0

GRAPHICS

Reported cases of rubella and congenital rubella syndrome (CRS) — National Notifiable Diseases Surveillance System, United States, 2004-2012



* By year of birth.

Reproduced from: Three cases of congenital rubella syndrome in the postelimination era--Maryland, Alabama, and Illinois, 2012. MMWR Morb Mortal Wkly Rep 2013; 62:226.

Graphic 89568 Version 1.0

Contributor Disclosures

Laura E Riley, MD Nothing to disclose **Martin S Hirsch, MD** Nothing to disclose **Charles J Lockwood, MD, MHCM** Consultant/Advisory Boards: Celula [Aneuploidy screening (No current products or drugs in the US)]. **Allyson Bloom, MD** Nothing to disclose

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy