Blood transfusion has provided life-saving support for the last 80 years. Transfusions are necessary in a variety of circumstances, which include medical care, surgical treatment, and acute trauma cases. It is estimated that annual need for transfusions exceeds 20 million blood products annually in the United States. There are little long-term effects on recipients, and the risk of infection is low for currently screened infectious agents.1,2 However, there remains an unknown risk of new emerging blood borne pathogens for which donor screening is currently not available. Since the early days of blood transfusion there has been a steady progression in the identification of transmissible infectious diseases, with consequent improvement in blood safety. Historically, blood donor screening used health histories, a self-deferral process and pre-selected donor groups to enhance safety. The number of transfusion-transmitted infectious diseases (TTIDs) has increased since the beginning of routine blood transfusion in the late 1930s.3 Syphilis was the first TTID for which prospective blood donors were tested in the 1950s.4 Although post-transfusion hepatitis was recognized in the 1940s, it was thought to be transient and a minor infection. Awareness of TTIDs became more significant, along with the public demand for increased blood safety, with the introduction of human immunodeficiency virus (HIV) into the blood donor pool. The first acquired immunodeficiency syndrome (AIDS) case was reported in 1981, with the first reported AIDS transfusion related case in 1982.5,6 It was not until 1985 that blood banks had a test available to screen blood for the AIDS virus. For the 4-year interim period, volunteer blood donors were screened based on high-risk behavior and self-exclusion. Hepatitis was the second most feared transfusion associated chronic infection, caused by hepatitis B and hepatitis C (previously known as non A/non B). The transfusion risk for contracting hepatitis was 25% before 1965.2 Immunoassays were first developed for the hepatitis B virus in the 1970s.7 Since that time, there have been unprecedented efforts by the transfusion medicine industry to make the blood supply safer. As a result, chronic post-transfusion infections have diminished significantly (Table 1). Reasons for improved blood safety include:

- Discontinued paying donors (moved to an all volunteer donor pool).
- Development of an antibody test for hepatitis B surface antigen (HBsAg) in 1976-1977.
- Instituted better donor screening methods including health history and demographics.
- Added surrogate testing for non-A-non-B hepatitis, including alanine aminotransferase in the late 1980s.

<table>
<thead>
<tr>
<th>Infectious Disease</th>
<th>Estimated Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syphilis</td>
<td>Negligible</td>
</tr>
<tr>
<td>Hepatitis C</td>
<td>1:2,000,000†</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>1:205,000††</td>
</tr>
<tr>
<td>HIV-1</td>
<td>1:2,000,000‡</td>
</tr>
<tr>
<td>HTLV-III</td>
<td>1:2,000,000‡</td>
</tr>
<tr>
<td>West Nile Virus (WNV)</td>
<td>Regional and seasonal risk§</td>
</tr>
<tr>
<td>Cytomegalovirus (CMV)</td>
<td>Infrequent¹</td>
</tr>
</tbody>
</table>

*Estimate for HCV and HIV-1 includes NAT blood screening data across multiple blood donor centers.⁴
†Estimate for HBV risk is prior to NAT HBV DNA blood screening.⁵
‡Estimate for HTLV-IV.⁶
§Estimate for WNV is based on 2003 blood screening data.⁶
¹Infrequent with leukocyte reduced components.⁹
• Introduced routine screening using an immunoassay for HCV antibodies in 1990 with ongoing HCV assay sensitivity improvements.
• Food and Drug Administration licensed EIA serology and new generation test kits specific for hepatitis B and C, HTLV-I/II, and syphilis.
• Developed and implemented nucleic acid test based technologies, currently used for HIV-1, HCV and West Nile Virus (WNV) screening; 1999 for HIV-1, HCV and 2003 for WNV.

Moreover, the manufacturers of blood screening assay tests continue to improve their products with automation, as well as increased assay sensitivity and specificity. Currently, the FDA required infectious disease blood-screening test panel includes the assays listed in Table 2. There are several manufacturers that have licensed blood-screening assays. Detection of infectious diseases in the blood bank setting is generally based on serologic screening for anti-viral antibodies by enzyme immunoassay (EIA) and enzyme-linked immunosorbent assay (ELISA) with confirmation by supplemental antibody tests such as recombinant immunoblot assays (RIBA) for HCV and Western blot or immunofluorescence assays for HIV. Alternatively, in the case of hepatitis B surface antigen, the ELISA screening assay directly detects minute quantities of the viral protein in the blood; the confirmatory test uses an anti-HBs antibody to neutralize the viral protein. The more recent technology introduced in blood screening is NAT for the RNA (HIV-1, HCV) and DNA (HBV) viruses.

Blood screening laboratories are required to use these licensed tests and to follow the package insert stated test algorithm, whereby donation specimens that are initially reactive (IR) are retested in duplicate. For IR samples, a second positive result in either or both duplicate tests is considered “repeatedly reactive” and is a strong indication that the infectious agent is present. For nucleic acid testing (NAT), the initial test result (whether it is nonreactive or reactive) stands as test of record. If the serological and/or NAT assay test is reactive, the donated blood unit is discarded and the blood donor is deferred from further donation. For repeatedly reactive donor samples, confirmatory or supplemental tests are used to verify the presence of the infectious virus. The screening tests have a high degree of sensitivity to ensure infectious units are identified, whereas the confirmatory tests have enhanced specificity. It is the more specific confirmatory test result that is used in medical counseling of the blood donor. The following provides details on each of the infectious diseases and associated tests (Table 3) currently performed in blood donor screening laboratories.

### Syphilis: Treponema pallidum

In the 1930s the spirochete “Treponema pallidum” (T. pallidum), the causative agent of syphilis, was the most recognized transfusion transmitted infectious disease (TTID). Transmission of syphilis through blood transfusion today is extremely rare, as the organism does not survive well in greater than 72-hours of cold storage. Consequently, the transfusion risk is considered negligible. Wasserman developed the first syphilis tests in 1906, with the use of the non-treponemal complement fixation test. This test measures both IgG and IgM anti-‐lipid antibodies formed in response to lipoidal material released by damaged cells in early infection and to T. pallidum lipids. The assay’s antigen-antibody reaction is a suspended flocculation rather than an agglutination or precipitation commonly seen in serological tests. Although, a non-treponemal assay is useful in diagnosis of syphilis infection, it is not highly specific. In the mid-1960s, a hemagglutination test for T. pallidum was introduced and utilized as a blood-screening test. Subsequent to implementation, automation has been introduced using a microhemagglutination test for T. pallidum antibodies on an automated microplate system. The test principle is agglutination and pattern recognition. It is a 1-step process, which uses fixed chicken erythrocytes sensitized with T. pallidum. The donation sample is added to a sample diluent to prevent nonspecific reactions. Sensitized erythrocytes are then added and the reactants are allowed to settle in a terraced microplate well. If the donor has T. pallidum antibodies, hemagglutination occurs. Automation allows the instrument to read the settling patterns and differentiate agglutination from non-agglutination. A reactive test appears as a homogenous layer of cells. A nonreactive test presents as a compact dense button surrounded by a clear zone. A reactive result indicates the presence of IgG and/or IgM antibodies to T. pallidum, which may indicate an active, past or treated syphilis infection. A reactive or equivocal result on an initial treponemal-based assay requires a duplicate repeat screening test to distinguish active from non-active disease as well as rule out false positives. Repeatedly reactive results are further evaluated using a licensed confirmatory assay. An example is the CAPTIA Syph-G, an EIA that qualitatively detects IgG antibodies to T. pallidum. The assay principle uses microtitration wells coated with T. pallidum antigens and, when exposed to donor samples with specific antibodies, an enzymatic reaction results in a measurable spectrophotometric process.

### Hepatitis

Hepatitis viruses target and damage human hepatic cells. Hepatitis virus B and hepatitis virus C are frequently associated with post-transfusion hepatitis and, therefore, are routinely screened in the blood testing laboratory.

#### Hepatitis C Virus (HCV)

Hepatitis C virus is a Flaviviridae RNA virus and has a very high prevalence in the United States with approximately 1.8% of the population positive for HCV antibodies, 2.7 million chronic carriers, and 25,000 new infections annually. It was first identified as a blood-borne non-A, non-B hepatitis (NANBH). The causative agent was discovered through genomic analysis in 1989. It is estimated that greater than 90% of transfusion-associated hepatitis infections are hepatitis C. Although HCV infection is most frequently a result of intravenous drug use, approximately 15% of infections were from blood transfusions.
before reliable HCV blood donor serological screening assays became available in May 1990. It was then that the risk of HCV transmission declined. Further reduction occurred with the implementation of HCV NAT in 1999. The current risk estimate for HCV transmission from a properly tested blood component is approximately 1:1,935,000 (Table 1).

Routine donor screening includes a test for antibodies to HCV. One example is the Ortho HCV version 3.0 qualitative ELISA. The assay method is a 3-step process that uses microwells coated with recombinant HCV protein antigens, as the solid phase. The assay uses 3 recombinant HCV encoded antigens (developed by the Chiron Corporation): (1) Recombinant protein c22-3 is derived from the core region of the HCV genome. Following infection, antibodies develop early to this region. (2) Recombinant protein c200 is derived from nonstructural regions of the HCV genome. (3) Recombinant protein SNS is derived from a nonstructural region of the genome that encodes for the viral polymerase, an HCV replication enzyme. In step 1, the donor antibodies specific for HCV protein antigens become bound to the solid phase. For step 2, the bound donor antibodies are then detected by complementary antibody, which is labeled with an enzyme capable of acting on a chromogenic substrate. In step 3, once the enzyme substrate is added, the donor antibody can then be detected colorimetrically, by measured light absorbance within the microwell. For donation samples found repeatedly reactive, the qualitative Chiron RIBA HCV 3.0 strip immunoblot assay (SIA) may be used as a confirmatory test for HCV.

**Hepatitis B Virus (HBV)**

Hepatitis B virus is a DNA virus in the Hepadnaviridae family. The virion “Dane particle” is the infective agent and has both surface and core components—HBs antigen (HBsAg) and HBe antigen (HBeAg), respectively. Approximately 1 to 1.3 million of the United States population has serologic evidence of HBV infection. Since 1987, there has been a significant reduction in HBV infection, in large part due to hepatitis B vaccination programs. Hepatitis B surface antigen is detectable approximately 4 weeks after infection. Subsequently, IgM anti-HBc antibodies appear, coinciding with symptom onset. The current HBV blood transmission risk of 1:205,000, does not include NAT. Early studies suggest that NAT single donation testing has the potential to reduce transmission-false positive NAT HBV infection even further, however, not when using the current NAT minipool testing strategy.

**Hepatitis B Surface Antigen (HBsAg)**

Routine donor screening for HBsAg is a 2-step process carried out in a microwell coated with antibody to HBsAg as a solid phase. An example is the Ortho ELISA test system version 3.0. In step 1, working conjugate (antibody conjugated to horse-radish peroxidase), diluted in conjugate diluent is added to the test wells along with the donor sample. After a specified period of time, if HBsAg is present in the donor sample, it will bind to the antibody coated on the well and simultaneously bind to the conjugate to form immobilized antibody-HBsAg-conjugate complexes. In step 2, an enzymatic detection system (O-phenylenediamine (OPD) and hydrogen peroxide) is added to the test well. If bound conjugate is present, there will be oxidization and an orange colored end product; the color intensity is measured with a microwell reader at 490 to 492 nm wavelength. Hepatitis B surface antigen confirmatory testing is a neutralization procedure using an anti-HBs (human) antibody. Confirmation of HBsAg is determined when the viral protein test well reactivity is reduced by at least 50%. Antibodies to hepatitis B surface antigen (anti-HBs) generally appear a few weeks after HBsAg has cleared. In cases where HBsAg has cleared and the appearance of anti-HBs is delayed, antibody to HBC may be the only serological marker of recent HBV infection. Antibodies to HBC antigen are also found in individuals with chronic HBV infection.

**Hepatitis B Virus Core Antigen (HBeAg)**

Donor screening for HBeAg is performed with a qualitative ELISA specific for the antibody to HBC (anti-HBe). This antibody appears shortly after HBsAg is detected and remains detectable following the clearance of HBsAg. It is useful in monitoring the progress of HBV infection and can indicate both recent and past infection. Antibodies to hepatitis B surface antigen (anti-HBs) generally appear a few weeks after HBsAg has cleared. In cases where HBsAg has cleared and the appearance of anti-HBs is delayed, antibody to HBC may be the only serological marker of recent HBV infection. Antibodies to HBC antigen are also found in individuals with chronic HBV infection.

**Table 3_Routine Blood Screening Assays’ Technique and Specificity**

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<th>Manufacturer</th>
<th>Blood Screening Assay</th>
<th>Specificity</th>
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<tr>
<td>Syphilis – <em>Treponema pallidum</em></td>
<td>Olympus America</td>
<td>PK TP System Microhemagglutination Test for Detection of <em>Treponema pallidum</em> antibodies</td>
<td>99.8% (95% CI: 99.2–99.8%)</td>
</tr>
<tr>
<td>Hepatitis C</td>
<td>Ortho Clinical Diagnostics</td>
<td>ELISA Antibody to HCV (anti-HCV) Version 3.0</td>
<td>99.55%‡</td>
</tr>
<tr>
<td>Hepatitis C</td>
<td>Chiron/GenProbe</td>
<td>Procleix Nucleic Acid Test (NAT) for HIV-1/HCV RNA</td>
<td>100% (95% CI: 99.4–100%)</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>Ortho Clinical Diagnostics</td>
<td>Antibody to HIV core antigen (anti-HBC)</td>
<td>98.9%§</td>
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<tr>
<td>Hepatitis B</td>
<td>Ortho Clinical Diagnostics</td>
<td>Hepatitis B Surface Antigen (HBsAg)</td>
<td>99.97% (95% CI: 99.92–100%)</td>
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<tr>
<td>Human Immunodeficiency Virus-1 (HIV-1/HIV-2)</td>
<td>Bio-Rad Genetic Systems</td>
<td>Antibody to HIV-1/HIV-2 Peptide EIA</td>
<td>99.87% (95% CI: 99.82–99.92%)</td>
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<td>Human T-Lymphotropic Virus-1, II</td>
<td>BioMerieux</td>
<td>Vironostika HTLV-VII Microelisa system antibody to HTLV-VII (anti-HTLV-VII)</td>
<td>99.95% (95%CI: 99.89–99.98%)</td>
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<tr>
<td>West Nile Virus (WNV)</td>
<td>Chiron/GenProbe</td>
<td>Procleix Nucleic Acid Test (NAT) for WNV RNA</td>
<td>Not Applicable</td>
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<tr>
<td>Cytomegalovirus (CMV)</td>
<td>Olympus America</td>
<td>Passive particle agglutination test</td>
<td>99.3% (95% CI: 98.3–99.7%)</td>
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‡WNV is under IND, specificity data not available, pending assay licensure.
†95% Confidence Interval (CI) data was not provided.
*Specificity data represents routine blood donor population and laboratory testing sites.
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*Specificity data represents routine blood donor population and laboratory testing sites.
screening is the Ortho HBc ELISA test system. The assay format is a 3-step process, which is carried out in a microwell coated with recombinant-derived hepatitis B core antigen (rHBCAg). Step 1 begins with donor antibody (if present) binding and forming antigen-antibody complexes on the microwell surface. Step 2 is addition of antibody conjugate (murine monoclonal antibody specific for human IgG and IgM) to the test well, which binds to the antibody component of the antigen-antibody complex. Step 3 is a measurable colorimetric enzyme detection system (ODP and hydrogen peroxide) that is measured within the microwell reader.28 Unlike other serological blood-screening assays, there is no confirmatory test available. The blood-screening test algorithm allows the donor to be initially reactive and then reactive again (2 separate donation dates) before deferral.

**Retroviruses**

There are 2 human retroviruses that are transfusion-transmissible pathogens: human immunodeficiency virus (HIV) and human T-lymphotropic virus (HTLV). Retroviruses have the reverse transcriptase enzyme, which allows transcription of viral RNA to DNA after infection of a host cell.6

**Human Immunodeficiency Virus**

Acquired immunodeficiency syndrome in hemophiliacs and transfusion recipients first appeared in 1982.30,31 Subsequent studies established transmissibility of the causative agent (HIV) by untreated coagulation factor concentrates and blood components.32 The Centers for Disease Control and Prevention (CDC) has reported that 9,352 AIDS cases in the United States were linked to transfusion through December 31, 2001.33 Once transmissibility was established, preventive measures were put into place to protect the blood supply. In the spring of 1983, a detailed blood donor history questionnaire with deferral of high-risk blood donors was implemented, followed by antibody testing for HIV-1 in 1985. As a result, very few documented cases of HIV-1 transmission by blood products occurred after 1986.54 During the 1990s, there was sequential implementation of improved highly sensitive EIA tests for detection of HIV-1 and HIV-2 antibodies. With the addition of testing for HIV-1 p24 antigen in 1995, HIV transmission-risk transmission risk was further reduced. However, in 1999, HIV-1 RNA testing, using NAT methodologies, reduced further the window period of infection and the risk of transmission to the lowest level ever, 1:2,000,000 (Table 1).6,8

The HIV donor-screening assay is a qualitative EIA test specific for the antibody to HIV (anti-HIV-1/HIV-2). An example is the Bio-Rad Genetic Systems HIV-1/HIV-2 peptide EIA. The assay uses synthetic peptides derived from highly conserved, immunodominant regions of the env (envelope) and pol (polymerase) gene products for HIV-1 and HIV-2. The microwells are coated with a mixture of 4 peptides: env and pol sequences for both HIV-1 and HIV-2. This 3-step assay begins with step 1, donor samples with a diluent are added to the microtiter wells. If the donor sample has HIV-1 and/or HIV-2 antibody it will bind to the absorbed antigen coated on the well. In step 2, working conjugate solution (peroxidase-labeled goat anti-human immunoglobulin) is added to the wells and binds to the antibody-antigen complex, if present. Step 3, a working chromogen solution is added to the wells and allowed to incubate. If HIV antibody is present a blue or blue-green color develops, which is in proportion to the amount of HIV antibody present in the sample. During step 3, color development stops with the addition of acid and the color changes from blue-green to yellow and optical absorbance is measured spectrophotometrically at 450 nm.35 Confirmatory testing is performed with an HIV-1 Western Blot qualitative assay, which uses disrupted proteins of HIV-1 separated by gel electrophoresis according to molecular weight. If virus-specific antibodies are present, they bind to their corresponding viral band.36

**Human T-Lymphotropic Virus Type I and Type II (HTLV-I/II) Infections**

Human T-lymphotropic virus type I and II are closely related to retroviruses in the Oncovirinae group. Unlike HIV, HTLV is rarely present in cell-free plasma and is associated with neoplastic conditions and a variety of demyelinating neurologic disorders.17 Routine blood screening for the HTLV-I antibodies began in the United States in 1988. In 1994, a more sensitive HTLV-I/II combination assay was implemented.6 An example is the Vironostika HTLV-I/II Microelisa system assay. This ELISA uses solid phase microwells coated with purified HTLV-I and HTLV-II viral lysate, and a recombinant HTLV-I p21E antigen.37 This 3-step assay begins with step 1, the addition of a diluted donor sample. If antibodies to HTLV-1 or HTLV-II are present, complexes are formed with the interaction of the donor antibodies with the solid phase antigens. Step 2, is the addition of anti-human immunoglobulin (goat) conjugated with horseradish peroxidase (HRP), which binds the antibody-antigen complex and a blue color is produced. Step 3 stops the enzymatic reaction with the addition of a sulfuric acid solution and the color changes to yellow; optical absorbance is measured spectrophotometrically at 450 nm. The amount of antibody present in the sample is proportional to color development.38 The blood-screening test algorithm, similar to that for anti-HBc, allows the donor to be initial reactive and then repeat reactive (2 separate donation dates) before deferral.

**Nucleic Acid Testing**

In 1994, the FDA urged the industry to develop the sensitive NAT tests to assure the public that the blood supply was as safe as possible. Another driving force for NAT implementation was the European plasma fractionators’ requirements, which required HCV NAT-screened plasma products. Studies have shown that there is a “window period” when the donor is infectious, but has not made detectable antibodies for HIV-1 and HCV. Since 1999, under an FDA investigational new drug (IND) application, NAT testing has been used to detect RNA from hepatitis C virus (HCV) and from human immunodeficiency virus (HIV-1). Nucleic acid testing assay licensure for HIV-1 and HCV occurred in 2002-2003. Implementation of these tests have significantly reduced the pre-seroconversion window period for HIV-1, HCV, and identified many RNA positive, antibody negative infectious units.39 It is important to note that NAT testing is very sensitive; however, the current technology is a semi-manual process. Because the technology is labor intensive, the blood industry has generally adopted a “minipool” test algorithm. Donation samples are tested with either 16 or 24 member pools, depending on the manufacturer, rather than testing individual donation samples. The minipool test algorithm allows for completion of all 16 or 24 donation sample test results...
if the minipool test result is negative for RNA. If the minipool is positive for RNA, further testing of the individual donation samples is required to determine each correct result. Currently, there are 2 manufacturers distributing NAT tests kits for routine blood screening. The Procleix (Chiron/Gen-Probe) qualitative nucleic acid assay system for the detection of HIV-1 and/or HCV RNA in minipools of 16 donation samples. This test is a multiplex NAT assay, which simultaneously detects HIV-1 and HCV RNA using transcription mediated amplification (TMA). The Roche Molecular Systems COBAS AmpliScreen uses PCR technology to separately detect HIV-1 and HCV RNA in minipools of 24 donor samples. Minipool nucleic acid amplification testing has helped prevent the transmission of approximately 5 HIV-1 infections and 56 HCV infections annually and has reduced the residual risk of transfusion-transmitted HIV-1 and HCV to approximately 1:2 million blood units.

As well as keeping the blood supply safe, NAT testing is useful for resolving true infectious status of seroreactive donors due to the enhanced specificity over traditional serology based EIA formats. For example, blood donors who are HCV confirmed positive might have a resolved or persistent infection. If the HCV RNA had dropped below the limits of detection, this may assist the clinician in pharmacological treatment of the individual. In addition, NAT test results reinforce diagnoses based on serology confirmation and aid in counseling donors who are HIV-1 or HCV antibody positive to their “true infectious state.”

**West Nile Virus Infection**

West Nile virus is a single-stranded RNA arbovirus (ie, transmitted by mosquitoes and arthropod vectors) of the Flavivirus family with the potential to cause meningoencephalitis. It was first isolated in 1937 in the West Nile District of Northern Uganda and derives its name from that region. Mosquitoes are vectors with birds serving as the primary vertebrate hosts. Humans and other mammals (particularly horses) are incidental hosts with transmission through bites of infected mosquitoes. There were no cases of WNV infection in the United States prior to an outbreak in New York City, in the summer of 1999. There was a large epidemic in 2002, with 23 transfusion-transmitted WNV infections confirmed, including transmission from an organ donor to 4 recipients. All types of blood components were implicated. West Nile virus blood donor screening with NAT assays was implemented throughout the United States in June/July 2003 using a minipool NAT and individual donor screening protocols. From late June to December 2003, approximately 6 million donations were screened for WNV, yielding over 800 (0.01%) viremic donations. The NAT-based WNV screening assays are similar to the methods described for NAT HIV-1 and HCV testing.

**Cytomegalovirus (CMV) Infection**

Cytomegalovirus is a double-stranded DNA virus similar to members of the herpes virus family. Although this virus is not routinely screened in the blood donor population, CMV infection can lead to increased morbidity and mortality in the immunosuppressed patient, infants, and the pregnant mother’s unborn child. Therefore, the blood bank strategy is to selectively screen donor units for CMV for patients who need this additional measure of safety. Cytomegalovirus is worldwide in distribution with 20% to 82% of the adult population positive for CMV antibody. Most infections are asymptomatic or associated with nonspecific illness. Once initial infection occurs, the virus may remain in a latent state indefinitely or become active later on. There are a number of test methods available to detect CMV antibodies. One example is the PK CMV-PA system, which uses gelatin particles coated with CMV antigens to detect IgG and IgM antibodies. The assay is a 3-step process. With step 1, the donor sample is added to a sample diluent and then mixed with the sensitized particles in a terraced microplate well, whereby the particles settle. In step 2, antibody to CMV binds to the antigen-sensitized particles and agglutination forms (visually seen as a homogeneous blue layer of gelatin particles). Absence of antibody appears as compact dense blue button with a surrounding clear zone. For step 3, an automated instrument reads the particles settling patterns and differentiates between agglutination (positive) and non-agglutination (negative).

**Future Considerations for Blood Bank Donor Screening**

Emerging transmissible diseases vary depending on the geographic location of the donors and recipients. The following agents are currently being investigated or discussed as potential targets for blood donor screening assays. Parasitic infections including malaria, Lyme disease, Babesiosis and Chagas disease (T. cruzi) were previously thought to be non-existent in the United States blood donor population; however, recent reports indicate otherwise. Based upon results from select trials, indicating the presence of T. cruzi in the United States blood donor population, manufacturers are developing Chagas screening assays, which may implement in 2006. Immunocompromised patients and stem cell transplant recipients are vulnerable to the herpes viruses, cytomegalovirus and Epstein-Barr virus. Patients with chronic hemolytic anemia are at an increased risk if they become infected with Parvovirus B19, which may cause severe aplastic crises. This DNA virus can survive solvent-detergent viral inactivation treatment. Therefore screening for B19 has become an important cause for the plasma fractionators. As a result, it is expected within 2005 that most recovered plasma products will be screened for this infectious agent. Variant Creutzfeldt-Jakob disease (vCJD) (human form of mad cow disease caused by infectious prion proteins) is a fatal neurological disorder and thought to be a low risk in blood transfusion. However, there is recent interest in FDA’s Blood Products Advisory Committee to defer blood donors who were transfused in the United Kingdom since January 1980.

**Conclusion**

The benefits of blood transfusion, when indicated, are clear. The risk of infection due to blood transfusion has decreased substantially during the past 80 years in large part due to screening volunteer blood donations for transmissible infectious diseases. The safety of the blood supply has improved through ongoing implementation of new generation antibody and antigen assays, as well as nucleic acid testing. As new and emerging diseases are recognized, it becomes the responsibility of the assay manufacturers, blood-screening laboratories, accrediting agencies, and the FDA to develop, evaluate performance and/or require implementation of new screening tests to ensure that the blood banks have the safest blood supply attainable.


13. CAPTTA Syphilis (T. pallidum)-G. Trinity Biotech USA. Package insert 970-29, Rev. B.


