ANZSBT Position statement on prevention of transfusion transmitted CMV

Summary of findings and recommendations

Cytomegalovirus (CMV) is prevalent in the Australian and New Zealand communities and once infection occurs, it remains latent within the host. For most people CMV infection is a mild, serious illness and death can result in immunocompromised individuals, and it is now the most common serious congenitally acquired infection. Given that CMV is transmitted within monocytes in cellular blood products, blood components for at-risk populations are commonly selected from serologically CMV-negative donors.

Reduction in CMV transmission was one of the specific objectives when introducing universal leucocyte filtration of cellular blood products. There is a lack of consensus on whether it is sufficient to use CMV serology unselected units in high-risk patients, with wide variation in practice, prompting a revision of ANZSBT guidance.

The Clinical Transfusion Practice Committee (CTPC) undertook a systematic review of CMV infection rates with and without selection for CMV seronegative units and performed a meta-analysis of comparative studies; reviewed haemovigilance data and correlated with preclinical studies. International guidelines were reviewed and CTPC developed evidence-based recommendations to prevent transmission of CMV through the blood supply and referenced broader CMV harm reduction strategies for at risk populations.

Key findings

- CMV infection is common in the population, including blood donors and difficulties are sometimes seen in sourcing appropriate blood from CMV seronegative donors
- Detectable CMV in clinically well, longstanding CMV positive donors is rare
- Detectable CMV in CMV negative donors may rarely be seen within a primary infection window period and poses a theoretical risk of transmission
- Modern leucocyte depletion processes routinely reduce CMV to levels below those shown to result in transmission in murine models. Leucocyte failure rates are low.
- CMV infection rates are equivalent following transfusion of leucodepleted blood products irrespective of whether it has been sourced from CMV positive or CMV negative donors
- No confirmed case of transmission of CMV with leucocyte depleted blood was found, including documented cases of transfusion of viral DNA positive units
- Pathogen reduction technologies are an effective alternative to leucodepletion to prevent CMV transmission
- International guidelines have gradually reduced the populations for which CMV negative blood should be given

Recommendations

- **Leucodepletion, pathogen inactivation or selection of CMV negative donors are each independently suitable methods to reduce transfusion transmitted CMV to negligible levels.**
- **Selection of blood from serologically negative donors is not recommended when transfusing leucodepleted blood.**
- **Seronegative donors must be used where possible for transfusion of cellular products that are not leucodepleted to at risk recipients. At risk recipients are people with conditions known to be associated with severe consequences of CMV including all women who are pregnant or likely to become pregnant within 12 months, people who are seronegative and have immunosuppression following stem cell or solid organ transplant, primary or secondary immunodeficiencies where CMV infection is a known risk.**
- **Where transfusion of non-leucodepleted cellular products from CMV positive donors occurs, clinical follow with consideration of pre-emptive therapy should be considered in recipients at risk of clinically serious CMV infection.**
- **Effective strategies to limit consequences of CMV include primary prevention public health strategies, monitoring with pre-emptive therapy and provision of blood products with a negligible risk of transmission (Leucodepletion, pathogen inactivation or selection of CMV negative donors) to at-risk populations. These include immunocompromised, neonatal or intrauterine patients and all women who are or may become pregnant.**
- **Suspected cases of Transfusion Transmitted Cytomegalovirus (TT-CMV) in immunocompromised, neonatal or intrauterine patients, and CMV-seronegative transplant recipients should be investigated using a standardised pathway. This should include assessment of maternal CMV serostatus and breast-milk exposure, donor and component tracing, residual component testing where available, and consideration of molecular sequencing to establish or refute donor–recipient linkage.**

Background

Cytomegalovirus (CMV) is a human herpes virus clinically relevant in transfusion medicine due to its transmission in transfused cellular products. Donor to recipient transmission has been confirmed with molecular characterisation of virus(1). Transmission is primarily through leucocytes in cellular products and leucodepletion was introduced with CMV transmission reduction specifically as one aim(2). Selection of blood from CMV serologically negative donors remains common.

There is variation in clinical practice and in practice guidelines(3-9). Expert opinion-based guidelines have increasingly limited the target populations where CMV negative blood is recommended and some centres no longer recommend CMV seronegative units in addition to leucodepletion, including the 2025 Canadian guidelines(8).

CMV seronegative blood products add no additional risk for recipients however lack of compliance with recommendations to transfuse only CMV seronegative products can provoke anxiety in recipients and staff involved in transfusion decisions where guidelines have not been followed or there are supply constraints. In addition, maintaining CMV negative inventories adds complexity in blood banks and transfusion laboratories and may become increasingly difficult if donor seroprevalence rises(10).

Epidemiology of CMV

CMV is common in the community, including in blood donors(11) . The seroprevalence of CMV in Australians up to the age of 60 years was estimated at 57% in 2006. Mother-to-child transmission (antepartum, intrapartum and postpartum including via breastmilk) is common in infants, with further substantial acquisition through community exposure across childhood and adolescence, such that 53% of individuals were seropositive by 20 years of age in one study(12). Age-weighted seroprevalence in blood donors was 76.1% in 2012 with a disproportionate number of seropositive females(10). New Zealand data from 2006 estimated CMV seroprevalence of 60.1% in over 9000 first time donors(13). It is posited that women have higher rates of infection due to increased contact with small children with exposure to bodily excretions or contaminated objects(14, 15).

Relatively low levels of plasma CMV DNA are detectable from several days to weeks following primary infection. The window period between infection and serological conversion can last up to several weeks. CMV DNA rises during the window period with peak plasma levels shortly after the appearance of anti-CMV IgG, which then persist lifelong. IgM antibodies are detected prior to IgG antibodies or shortly after. Peak IgM antibody titres occur during the first 3 months after infection then rapidly decline(16, 17).

Lifelong latency is established within cells of the myeloid lineage, particularly monocytes, and these are considered the major source of CMV transmission in blood products(18, 19). Cell free CMV DNA may be seen in plasma, but there is at most a low possibility of transmission. Of 221 (39 CMV seronegative) immunocompetent recipients

of blood with detectable CMV in the plasma, none developed a CMV-like illness or confirmed seroconversion(20). Plasma products are not currently issued based on CMV serology results. Reactivation at times of immunocompromise due to immunosuppression, medical conditions or pregnancy (24) can release cell-free CMV into the blood stream, breast milk, urine and saliva(21). Reinfection with a new strain can also occur.

Transfusion-transmitted CMV infection (TT-CMV) has been reported since the 1960s(22) First reported after cardiac surgery, clinically significant infections are particularly seen in immunocompromised populations including premature infants, haematology, including bone marrow transplant recipients and solid organ transplant patients. High rates of community transmission confound attribution of a post transfusion infection to blood. Secretion in saliva, urine and breast milk are common, may be prolonged after infection and may re-emerge during otherwise latent infection(23).

Clinical significance

CMV can lead to diverse clinical manifestations. Asymptomatic infection, respiratory symptoms or a mononucleosis-like illness are common in immunocompetent people. Seroconverting blood donors reported compatible symptoms in 85% of cases in one study, however 69% of persisting CMV negative controls experienced similar symptoms(17).

In immunocompromised patients, retinitis, pneumonitis, hepatitis, enterocolitis and marrow suppression are common and have been common causes of death, particularly in the post-transplant setting, both from primary infection and reactivation.

Congenital CMV (cCMV) is acquired from the mother during pregnancy and is more likely in maternal primary infection during or leading up to pregnancy than with secondary infection or reactivation. While transmission rates appear lower during first trimester, the resulting disease is more severe, with few longer-term sequelae if acquired in second and third trimesters(25). Deafness and neurodevelopmental delay can develop during childhood, even in children asymptomatic at birth. The incidence of cCMV in Australia is estimated to be at least 3.85 per 100 000 live births(15). The case fatality rate is estimated at 20% from intrauterine or neonatal death. Additional morbidity includes sensorineural hearing loss (12%) and cerebral palsy (10%)(26).

Infection rates following transfusion

Prior systematic reviews have not supported reliance on leucodepletion to prevent CMV(27), largely because they were based on historical evidence that does not reflect modern practice. Most included studies pre-dated universal, validated pre-storage leukodepletion and relied on small, heterogeneous, and underpowered cohorts, often using bedside or post-storage filters with inconsistent leukocyte removal. CMV-seronegative blood was already entrenched as the “gold standard,” setting a high evidentiary bar that leukodepletion studies could not meet, while theoretical concerns about cell-free CMV viraemia and the absence of mature haemovigilance systems

encouraged conservative interpretation. Despite a low estimated residual risk,(28) recommendations continued to support use of CMV seronegative blood in selected recipients. We therefore undertook a systematic review to evaluate the risk of TT-CMV in the context of universal pre-storage leucodepletion.

There were four comparative clinical studies (one randomised and three observational) that examined CMV infection rates following transfusion of leucodepleted blood with or without CMV serological donor selection. Meta-analysis demonstrated no difference in CMV infection rates between groups (relative risk 1.21, 95% CI 0.42–3.49). Pooled analysis of 19 observational studies reporting CMV incidence following leucodepleted transfusion found rates of 0.17% for leucodepletion alone and 0.22% for CMV-seronegative plus leucodepleted products, reinforcing the absence of a clinically meaningful difference. Haemovigilance reports were also reviewed and found no confirmed cases of TT-CMV attributable to leucodepleted blood products, despite inclusion of high-risk populations and decades of surveillance. Pre-clinical, translational, and modelling studies provided strong biological plausibility for the clinical findings.

Residual risk modelling has estimated the likelihood of TT-CMV with leucodepleted blood to be less than 1 in 13 million transfusions, a frequency below the detection threshold of population-based surveillance.(28) Collectively, these data indicate that CMV serological donor selection does not confer additional safety benefit when effective leucodepletion is in place, supporting rationalisation of CMV-negative inventory requirements in modern blood systems.

Haemovigilance

Over the past decade, international haemovigilance systems have shown no evidence of TT-CMV in jurisdictions using universal pre-storage leucodepletion. In the United Kingdom, the Serious Hazards of Transfusion (SHOT) scheme applies stringent attribution criteria for transfusion-transmitted infections, including exclusion of non-transfusion sources and, where possible, donor or component testing. Within this framework, SHOT has continued to report only very small numbers of suspected CMV cases, with no confirmed cases to suggest ongoing TT-CMV risk (SHOT Annual Reports 2015–2024, available from https://www.shotuk.org/shot-reports).

Canadian haemovigilance and policy outputs similarly describe TT-CMV as an exceptionally rare event since the implementation of universal leucodepletion, with National Advisory Committee (NAC) guidance consistently framing leucodepleteded components as “CMV safe” and clinically equivalent to CMV-seronegative products. The Canadian Blood Service moved to a single CMV prevention strategy in 2017 with the provision of leucodepleted blood component for all indications except IUT. To date there have been no reported cases of TT CMV infection with this strategy, and very recently they have extended the single strategy of pre-storage leucodepletion to include IUT(29).

In the United States, haemovigilance data from AABB-aligned guidance, the CDC's National Healthcare Safety Network (NHSN), and Food and Drug Administration (FDA) surveillance demonstrate that transfusion-transmitted infections are uncommon overall, and that CMV is not a pathogen in reported cases. The lack of contemporary high-quality trials demonstrating superiority of CMV-seronegative over leucodepleted components reflects the near-elimination of TT-CMV rather than ongoing uncertainty. Collectively, these haemovigilance data support the conclusion that residual TT-CMV risk in modern blood systems is extremely low and largely theoretical.

Beyond population-level haemovigilance reporting, evidence addressing whether CMV-untested but universally leucodepleted blood components confer an acceptably low risk of TT-CMV is best drawn from prospective and observational studies in high-risk populations, in whom even rare transmission events would be most readily detected. CMV-seronegative allogeneic haematopoietic stem cell transplant (HSCT) recipients represent a particularly sensitive model, given their profound immunosuppression, frequent transfusion exposure, and routine post-transplant virological surveillance. In this setting, Bowden *et al.* demonstrated in a prospective randomised trial that leucocyte depleted components were equivalent to CMV-seronegative components in preventing CMV infection, despite the use of filtration techniques less effective than contemporary pre-storage leucodepletion(30, 31). Subsequent observational and prospective studies using CMV-unscreened leucodepleted components have shown similarly reassuring results. Narvios *et al.* reported no cases of CMV disease or clinically significant TT-CMV in 72 CMV-seronegative HSCT recipients monitored for at least 100 days post-transplant (32). Thiele *et al.*, using systematic CMV DNA nucleic acid testing alongside serology, observed no CMV DNAemia or disease across 1,847 transfused products from over 3,000 donors, corresponding to an estimated TT-CMV risk of 0% (33).

Parallel findings are observed in neonatal and very-low-birth-weight (VLBW) populations. Prospective studies consistently demonstrate that when leucodepleted (with or without CMV-seronegative selection) blood components are used, transfusion-transmitted CMV is effectively prevented, and postnatal CMV acquisition is instead dominated by non-transfusion sources, particularly breast-milk exposure and perinatal infection (14, 23, 34-38). Importantly, even in studies employing intensive virological surveillance, TT-CMV has not emerged as a measurable contributor to CMV infection in modern neonatal practice.

Finally, we acknowledge the inherent limitations of passive haemovigilance in attributing CMV acquisition to transfusion. To address this, any future suspected TT-CMV cases in immunocompromised, neonatal or intrauterine patients and CMV-seronegative transplant recipients should trigger a standardised investigation pathway, including assessment of maternal CMV serostatus and breast-milk exposure, donor and component trace-back, residual component testing where available, and consideration of molecular sequencing to establish or refute donor–recipient linkage.

Early notification of transfusion services and haemovigilance programs is essential to enable timely component tracing and maximise the feasibility of residual component testing and molecular analysis.

Recommendations:

- **Suspected cases of TT-CMV in immunocompromised, neonatal or intrauterine patients, and CMV-seronegative transplant recipients should be investigated using a standardised pathway. This should include assessment of maternal CMV serostatus and breast-milk exposure, donor and component tracing, residual component testing where available, and consideration of molecular sequencing to establish or refute donor–recipient linkage.**

Risk of transmission from CMV-negative donors

Although CMV-seronegative blood components have traditionally been considered the safest option, there remains an inherent limitation due to the window period following acute infection. During this period, donors may have circulating CMV DNA in plasma or latently infected leucocytes but have not yet seroconverted to detectable IgG antibodies. Several studies have documented the presence of CMV DNA in otherwise seronegative donors, albeit at low frequency(16, 39). The infectivity of such low-level viraemia remains uncertain, but its existence highlights that CMV-seronegative status may not provide absolute protection and challenges the perception of it as a gold standard.

Laboratory / preclinical evidence

Leucodepletion

The New Zealand Blood Service (NZBS) introduced universal leucodepletion in 2001, with the Australian Red Cross Lifeblood (ARCL) implementing the same practice nationally in Australia in 2008. Current leucodepletion filters result in < 5 x 10^6 residual leucocytes in RBC or platelet components. Filter failures are rare. Enumeration of residual leucocytes in select products is performed as part of routine quality control.

Prior to leucodepletion the estimated rate of TT-CMV ranged between 28-57%. Post leucodepleted the prevalence of primary CMV fell to 0.23-4% in stem cell transplant recipients with earlier generation filters(21).

Breakthrough infections occur due to failure of filtration mechanisms or due to the transfusion of cell free CMV. In a nationwide Swiss study of blood donors, Voruz et al found 0.009% (4 of the 42,240) of donations tested were positive for CMV DNA. However, viremia levels were low, and at levels where infectivity is unknown(21). Whilst all donors were CMV IgG positive, others have reported similar prevalence of CMV DNA in CMV seronegative donors in the window period of primary infection(40).

Pathogen inactivation

Currently there are no pathogen-inactivation (PI) technologies used in Australia or New Zealand, but they are used in other jurisdictions with- amotosalen/UV-A (INTERCEPT) most commonly used for platelets. This process reliably inactivates CMV in vitro and in platelet concentrates, with studies demonstrating ≥5–6 log reductions in CMV infectivity and prevention of transfusion transmission in animal models(41).

Contemporary policy and guidance increasingly recognise PI platelets as an acceptable CMV-risk–mitigation strategy alongside universal leucodepletion, with AABB materials explicitly listing PI as a CMV-risk–reducing option and Canadian services moving toward broader PI adoption within their inventories.

Recommendations

- **Leucodepletion, pathogen inactivation or selection of CMV negative donors are each suitable methods to reduce transfusion transmitted CMV to negligible levels.**
- **Selection of blood from serologically negative donors is not recommended when transfusing leucodepleted blood.**

Clinical contexts

Pregnancy

cCMV can occur from maternal CMV transmission, more likely in the context of primary rather than secondary infection. The rates of primary CMV infection in pregnancy vary depending on the setting with estimates of between 0.3 – 7% in high income settings internationally(42, 43). In the Australian setting, the most robust data comes out of South Australia, with an incidence of primary CMV infection estimated to be 6 per 1,000 pregnancies (44) These figures are well below the theoretical risks associated with leucodepleted cellular blood products. Recommendations to prevent cCMV have a strong focus on prevention of infection through contact with bodily fluids and do not mention transfusion(45).

Transfusion of CMV seronegative blood products continues to be the recommended practice in many countries. This approach fails to consider the risk of TT-CMV in the periconceptual period, where maternal infection remains active and can be transferred to the fetus if conceived within a 12-month period.(28, 29, 46)

Intrauterine and neonatal transfusion

Neonatal and intrauterine transfusion are of particular concern due to possible catastrophic outcomes. Fetal/neonatal immaturity and lack of effective treatments have resulted in significant apprehension when determining strategies to prevent TT-CMV in this vulnerable population.

Comparison of NAC (Canada), BSH (UK) CMV Recommendations for IUT

Aspect	NAC (Canada, 2025) (8)	BSH (UK, 2016)(47)
Position on CMV-safe vs CMV-seronegative	Pre-storage leucodepleted (CMV-safe) components are considered equivalent to	CMV-seronegative required; leucodepleted only acceptable if CMV-seronegative not available.

	CMV-seronegative, including for IUT.	
Rationale	Residual CMV transmission risk with modern pre-storage leucodepletion is extremely low; operational priority is extended phenotype matching for IUT.	Traditional concern about CMV transmission risk; default to CMV-seronegative where possible.
Other requirements for IUT RBC	Extended phenotype matching prioritised; irradiated as per local practice (not specified in this statement).	Must be irradiated; transfused within 24 h of irradiation; within 5 days of collection.

Neonatal/infant transfusion

Although CMV-seronegative, leucodepleted blood has historically been preferred for neonates—especially for very low birth weight (VLBW) infants—current evidence indicates that transfusion is not a significant source of CMV infection, particularly since the widespread use of leucodepleted blood products. The latest SHOT (UK) data show no confirmed TT-CMV, despite numerous process deviations where CMV-seronegative units were recommended and not provided(48). In VLBW infants in particular postnatal CMV is overwhelmingly linked to maternal milk, not transfusion(36).

CMV reactivation in mammary epithelial cells leads to viral shedding into milk in approximately 70–90% of seropositive mothers(49). Systematic reviews and cohort studies demonstrate that among very preterm and VLBW (<32 weeks or <1500 g), infection rates reach 16–26% with fresh milk versus 8–13% with frozen or mixed milk(49-51). The literature converges on a clear message: breast milk is both indispensable for neonatal health and the dominant vehicle for postnatal CMV transmission in high-risk preterm populations. Evidence supports heat treatment as the most effective preventive strategy, but practice must carefully balance infection risk against the loss of milk's bioactive properties(29, 48, 52-54).

Haematological malignancies

CMV is a concerning infection in immunocompromised haematology patients. Before leucodepletion, transfusion was a recognised pathway for TT-CMV. However, the introduction of universal pre-storage leucodepletion has drastically reduced this risk to undetectable levels(27, 48). A randomized trial, multiple comparative studies and subsequent systematic reviews and meta-analyses have confirmed this equivalence(27, 30, 31). Community transmission remains possible in immunocompromised groups, with monitoring of CMV and pre-emptive treatment for rising quantitative DNA levels now standard practice.

There are products typically only used in this population that cannot be leucodepleted. These include stem cells and granulocyte transfusions. Donor selection for allogeneic transplantation is complex, with CMV being a major but not the only factor in donor suitability. Granulocyte transfusions, (or any other cellular product not able to be leucodepleted) should preferably be from CMV negative donors when the recipient is

CMV negative. Clinical consideration of monitoring and pre-emptive therapy is likely to be required in most CMV negative recipients if CMV positive granulocytes are required.

Solid organ transplant recipients

Solid organ transplant (SOT) recipients, particularly CMV-seronegative recipients, are at high risk of CMV. Observational studies suggest that CMV infections in SOT are more commonly attributable to reactivation or community acquisition than to transfusion. Accordingly, CMV prevention in SOT is centred on antiviral prophylaxis or pre-emptive therapy, not transfusion strategy(55, 56).

Contemporary guidelines from the UK Advisory Committee on the Safety of Blood, Tissues and Organs (SaBTO), the Canadian National Advisory Committee (NAC), and the Australian National Blood Authority (NBA) all endorse leucodepletion as sufficient for SOT patients(29, 54, 57).

HIV-positive patients

In the anti-retroviral era, reports of TT-CMV in HIV are absent from the literature**.** The Viral Activation Transfusion Study(58), a randomized trial of leucodepleted versus standard red cells in HIV-infected patients, found no evidence of CMV activation or transfusion-attributable CMV DNA. Most participants were already seropositive and thus not at risk of primary TT-CMV.

Recommendations

- **Seronegative donors must be used where possible for transfusion of cellular products that are not leucodepleted to at risk recipients. At risk recipients are people with conditions known to be associated with severe consequences of CMV including all women who are pregnant or likely to become pregnant within 12 months, people who are seronegative and have immunosuppression following stem cell or solid organ transplant, primary or secondary immunodeficiencies where CMV infection is a known risk**
- **Where transfusion of non-leucodepleted cellular products from CMV positive donors occurs, clinical follow up with consideration of pre-emptive therapy should be considered in recipients at risk of clinically serious CMV infection.**

The collective evidence supports the use of pre-storage leucodepleted blood components as “CMV-safe” for immunosuppressed patients. CMV-seronegative components do not offer additional benefit over leucoreduction in these groups.

While TT-CMV has been a significant concern in the past, and vigilance must remain when transfusing non-leucodepleted products, the implementation of universal pre-storage leucodepletion has effectively reduced TT-CMV. A holistic approach to preventing harm due to CMV recognises that community acquisition is a far greater issue than iatrogenic infection through the blood supply. Strategies need to consider prevention of community infection as well as managing both primary infection and reactivation in populations at risk. Sourcing blood from CMV negative donors in

addition to universally applied leucodepletion, comes at additional cost, increases inventory management complexity and creates anxiety without additional benefit.

Recommendation

- **Effective strategies to limit consequences of CMV include primary prevention public health strategies, monitoring with pre-emptive therapy and provision of blood products with a negligible risk of transmission (leucodepletion, pathogen inactivation or selection of CMV negative donors) to at-risk populations. These include immunocompromised, neonatal or intrauterine patients and all women who are or may become pregnant.**

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