2025 AABB Annual Meeting Registration
October 25-28, 2025 | San Diego, CA

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The AABB Annual Meeting brings together the latest research and practice-changing resources for the fields of blood and biotherapies. Colleagues from throughout the world attend to connect, network, learn and advance the field.

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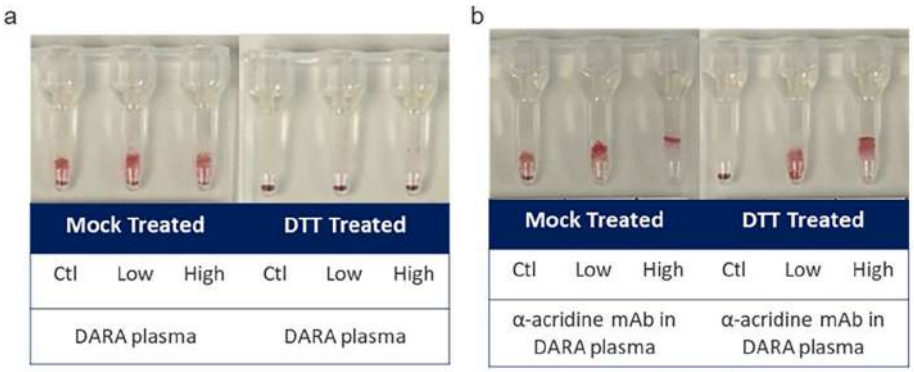

Figure 1: Representative Results of Antibody Screen Using Mock or DTT-treated Cells

designed to reduce the risk of transfusion-transmitted infections and to replace irradiation for the prevention of transfusion associated-graft-versus-host disease. The PR process leaves residual amustaline-derived acridine adducts on the RBC surface.

In clinical trials of PR-RBCs, an indirect antiglobulin test (IAT) with reagent PR-RBCs is used to screen for baseline and treatment-emergent PR-RBC specific antibodies. Each screening panel includes cells with no (control), low and high amounts of surface acridine adducts.

Daratumumab (DARA) used in the treatment of multiple myeloma interferes with blood compatibility screening, including the PR-RBC antibody screen. Dithiothreitol (DTT) treatment of RBCs has been shown to resolve DARA interference in standard blood compatibility testing.

The objective of this study was to determine whether DTT treatment of reagent PR-RBCs resolved DARA interference while still allowing the detection of PR-RBC antibodies.

Study Design/Methods: Nine PR-RBC reagent panels were treated to remove DARA interference (0 or 0.2 M DTT, 35 min, 37°C). The panels were screened with control anti-acridine antibody in an IAT assay (Ortho ID-Micro Typing System IgG plus C3d gel card) using plasma from DARA-treated patients, with or without added anti-acridine monoclonal antibody (2S-197M1). Surface acridine adducts on panel RBCs were quantitated by flow cytometry using QuantiBrite calibrated beads (BD Biosciences).

Results/Findings: Mock treated cells (no DTT) behaved as expected in 9 panels of reagent PR-RBCs. DARA interference was noted in all cells tested with DARA plasma including the control, while the DARA plasma and control anti acridine antibody combination displayed DARA interference with the negative control showing a positive result (Figure 1). DTT treatment eliminated DARA interference in all panels, including the PR-RBC reagent controls (Figure 1a) and in the DARA plasma and control anti acridine antibody combination, with some modulation of the acridine signal observed (Figure 1b). Surface acridine levels were quantitated by flow of PR-RBC panel cells and showed an average reduction in PE molecules/cell of 52.2% (37.7%–58.4%).

Conclusions: The standard use of DTT to resolve DARA interference was applied to the PR-RBC antibody screening assay and resulted in the elimination of DARA interference. Although acridine levels were reduced following DTT treatment, the PR-RBC IAT assay was able to detect the presence of positive control anti acridine antibody following DTT treatment, both in the absence and presence of DARA patient plasma.

P-CB-11 | Effect of Leukoreduction on Energy and Redox Metabolism in Red Blood Cells Stored for Transfusion

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Background/Case Studies: Stored red blood cells (RBCs) undergo various modifications that impair their function, collectively referred to as storage lesions. Available glucose is used as a substrate for the production of ATP and NADPH, serving as energy and reducing power sources, respectively. These lesions include alterations in the redox state of the cells, increased production of reactive oxygen species (ROS), and weakening of antioxidant defenses. In circulation, oxidative damage arises from hemoglobin autooxidation, extracellular agents such as the vascular endothelium, and the inflammatory context.

Study Design/Methods: We propose that residual leukocytes in the leukoreduced (LR) or non-leukoreduced (NLR) blood units exacerbate oxidative stress during storage. We analyzed the impact of pre-storage leukoreduction compared to non-leukoreduced units on various metabolites affected during storage. Glucose concentration was quantified using the Cobas 6000 analyzer (Roche), ATP was measured by a luminescence-based assay, and NADPH via an enzymatic cycling method.

Results/Findings: A decrease in glucose and ATP concentrations was observed with increasing storage time; however, no significant differences were found between NLR and LR units on day 42 in terms of glucose (NLR 198 ± 37 vs 176 ± 52 mg/dL $n = 7$) and ATP levels (NLR 71 ± 10 μM vs. LR 77 ± 43 μM, $n = 4$). Regarding NADPH quantification, no significant differences were detected between NLR and LR units on day 42 (NLR 14 ± 5 μM vs. LR 14 ± 7 μM, $n = 4$), nor in the NADPH/NADPtotal ratio (NLR 0.69 ± 0.09 vs. LR 0.67 ± 0.15, $n = 4$).

Conclusions: The decline in glucose concentration available for glycolysis may explain the reduction in ATP levels, as glucose is diverted toward NADPH production to sustain reducing power, maintaining its concentration throughout storage. The presence of leukocytes in the leukocyte-depleted blood unit does not appear to significantly impact redox balance, though it may limit ATP production, which is vital for erythrocyte functions. These results are relevant for public health policy decision-making and contribute to the discussion on selective versus universal leukoreduction strategies. Furthermore, they underscore the importance of continued research on the effects of oxidative stress on the metabolic pathways of red blood cells, particularly the pentose phosphate pathway, a key source of NADPH.

P-CB-12 | Emergence of Various Platelet Subpopulations with Specific Hemostatic Properties in Cold-Stored Amotosalen-UVA Pathogen-Reduced Platelet Concentrates

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Background/Case Studies: Current increase in the need for therapeutic *versus* prophylactic platelet transfusions leads to growing interest in cold-stored platelets (CSP), owing to their potentially advantageous characteristics as compared to standard room temperature platelets (RTP). We assessed the emergence of various platelet subpopulations and related functional properties in platelet concentrates (PCs) treated with amotosalen-UVA pathogen reduction and stored at 22°C or 4°C for 21 days (D).

Study Design/Methods: A pool-and-split strategy was used to obtain double-dose buffy-coat (BC)-PCs collected into PAS-C/plasma (55/45%) treated with amotosalen-UVA and stored at 22°C with constant agitation or at 4°C without agitation. Eight platelet subpopulations were identified using a combination of markers (P-selectin and phosphatidylserine (PhtdSer) exposure, DYm, PAC-1 binding for activated GPIIbIIIa) analyzed by multicolor flow cytometry (FCM). Functional testing included thrombin generation by calibrated automated thrombography, aggregometry, thrombus formation on a collagen-coated surface under flow conditions (1500 s^{-1}) and clot viscoelasticity of reconstituted whole blood (Quantra QPlus, HemoSonics LLC, Stago). Statistical comparisons were done by Pearson's correlation and two-way ANOVA followed by Tukey's post-hoc test ($n = 4$).

Results/Findings: Multicolor FCM analysis indicated that the subpopulation of resting platelets decreased from 78±3% to 45±2% at D7 at 22°C and to 8±1% at 4°C. In CSP, procoagulant (30±4% from D7 to D21) and apoptotic (53±3% by D21) platelets, both exposing PhtdSer, supplanted resting platelets. This activated pattern remained unaltered upon TRAP + convulxin stimulation as of D7, indicating maximally activated platelets in PCs. CSP displayed enhanced thrombin generation capacity as of D7 compared to RTP ($p < 0.01$), consistent with increased proportion of procoagulant platelets ($r = -0.7776$ $p < 0.01$). Aggregation to various agonists (collagen, TRAP, arachidonic acid) decreased progressively during storage and was lower in CSP *vs.* 7-day RTP (D21 $p < 0.01$), consistent with high procoagulant platelet content ($r = -0.6829$ $p < 0.05$) characterized by loss of aggregation capacity. Thrombus formation on collagen under flow with reconstituted hirudinated whole blood was best preserved at 4°C at least up to D14, compared to only D7 at 22°C. Platelet contribution to clot stiffness was lower with CSP as of D7 when compared with 7-day RTP ($p < 0.01$), consistent with a high proportion of procoagulant platelets ($r = -0.7227$ $p < 0.01$), lacking the ability to aggregate, contract and contribute to clot strengthening.

Conclusions: CSP is a promising strategy to prolong platelet storage, leading to phenotypic changes with resting platelets replaced by PhtdSer-bearing procoagulant and apoptotic platelet subpopulations with increased thrombin production, reduced aggregation and clot firmness.

P-CB-13 | Evaluation of a Calcein-Based Fluorescent Label for Tracking in Vivo Recovery and Survival of Human Platelets in a SCID Mouse Model of Transfusion

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