Summary/Conclusions: No significant differences were observed between the two groups of leuko-reduced packed RBCs stored in additive solution (study and control) as regards the mean fluorescence intensity of CD44, CD47 and Annexin V.

Blood products

Novel blood products/components

P213 | 1,2-α-fucosidase treatment effectively converts O group blood components into phenotypical Oh units compatible with Bombay patient plasma

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Background: Blood transfusion is a lifesaving intervention at the center of many routine medical procedures, including acute haemorrhages and cancer treatments. Yet transfusion is often out of reach for patients with the rare Bombay (O_h) and para-Bombay blood groups who have naturally-occurring haemolytic antibodies against carbohydrate determinants on common blood types.

Aims: The aim of this study was to investigate the ability of *1,2-α-fucosidase* from the probiotic bacteria *Bifidobacterium bifidum* to hydrolyze the H antigen on O type blood components and thus convert them to phenotypical Oh units.

Methods: The enzymatic reaction was performed at 4 degrees, directly in blood bags containing SAGM additive solution. The resultant H-antigen expression was evaluated using Ulex lectin and monoclonal anti-H antibodies in gel column based micro typing system cards, microscopy and flow cytometry. Furthermore, 20 sera from Bombay patients were crossmatched against ECOh RBCs and three of those sera were used for the Monocyte Monolayer Assay. We also cross-matched 1000 healthy blood donor plasmas against ECOh RBCs to screen for cryptic antigens. The physical properties of ECOh RBCs were evaluated using Ektacytometry.

Results: The enzymatic reaction effectively generated blood units phenotyped as Oh. Enzyme converted Oh (ECOh) red blood cells were crossmatch negative when tested against 20 unique Bombay patient sera. This compatibility was further confirmed through functional *ex vivo* testing using the Monocyte Monolayer Assay (MMA). *Fucosidase* treatment did not affect the physical membrane properties of the RBCs and it did not expose cryptic antigens.

Summary/Conclusions: Clinical availability of this easy-to-use *1,2-α-fucosidase* at Blood Banks could increase the chance to survive acute haemorrhage situations for the 100,000+ Bombay and para-Bombay individuals around the world as well as enable them to undergo modern medical treatments, e.g. chaemotherapy against cancer, with reliable access to transfusion support.

P214 | Abstract withdrawn

P215 | Differential expression of microRNAs in room temperature and cryopreserved platelets

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Background: Platelets contain an abundance of microRNAs (miRNAs) which are involved in the regulation of platelet function. The expression of miRNAs change during conventional platelet storage at room temperature, which is likely related to the platelet storage lesion and apoptosis. However, changes in expression of miRNAs in platelets stored under alternative conditions, such as cryopreservation, have not been examined. Analysis of apoptosis-related miRNAs in platelet components may provide new insight into the regulation of platelet function during component storage.

Aims: To characterise differences in the relative abundance of apoptosis-related miRNAs in fresh and cryopreserved platelets using a commercially available panel.

Methods: Apheresis platelets (day 1 post-collection) were cryopreserved with 5-6 % dimethyl sulfoxide (DMSO) before freezing at -80°C. Cryopreserved platelets were thawed and resuspended in a unit of plasma before sampling. Samples were taken before (fresh) and after cryopreservation ($n = 3$), and RNA was extracted using TRIzol reagent. RNA was reverse transcribed and PCR was performed with 25 ng cDNA using a miRCURY LNA miRNA Human Apoptosis Focus

P215 - Table 1: miRNAs with a relative increase in abundance in cryopreserved platelets compared to fresh platelets.

miRNA	p-value	Hypothesised role in platelets
miR-181c-5p	0.022	Inhibition of Bcl-2, inhibition of PI3K/Akt signalling pathway
miR-29a-3p	0.005	Inhibition of Bcl-2
miR-29c-3p	0.004	Inhibition of Bcl-2, inhibition of Bcl-w
miR-32-5p	0.032	Regulation of BIK
miR-365a-3p	0.047	Regulation of Bak and Bax
miR-497-5p	0.018	Inhibition of Bcl-2, inhibition of Bcl-w