OP 35. A review of RHCE inferred phenotypes and red cell genotyping data in donors serologically confirmed as negative for the high frequency antigen RH:34

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Background
The HrB(Rh:34) and hrB(Rh:31) antigens were defined in the literature in the 1970's after tests confirming the presence of the corresponding antibodies were found in the serum of a black South African woman. Rh34 is a high frequency antigen that is significant in South Africa as it is negative or absent in <0.2% RhD+/RhD- black donors thus defining them as rare donors. Anti-Rh34 is also a clinically significant antibody capable of causing Haemolytic Disease of the Newborn. Red cell genotyping using the IDCOREXT assay is performed on selected rare donors where serology is limited due to lack of rare expensive antisera. The IDCORE^{XT} assay tests for the hrB (Rh:31), however samples that test negative for Rh:31 gene may also be negative for HrB (Rh:34) and this could be a surrogate test for detecting potential Rh:-34 donors. The objective of this study was to review the RHCE phenotypes and red cell genotyping on donors serologically confirmed as Rh:-34 to identify phenotypic and genotypic result patterns that can be used as triggers when screening for Rh:-34 rare donors.

Methods
Samples from four serologically confirmed Rh:-34 black male donors were sent for DNA extraction on the Maxwell AS2000 instrument. Red cell genotyping was completed using the IDCORE^{XT} kit on the Luminex platform. DNA was outsourced for DNA sequencing. The molecular genotyping results and inferred phenotypes of the 4 donors were analysed and reviewed.

Results
Two donors (donor 1 and 2) were RHD- and displayed partial C, E-, partial c, partial e, VS+V- and Rh:-31 phenotypes. The third donor was partial RHD+ and had C-, E-, partial c, partial e, VS+V- and Rh:-31. The fourth donor was RHD+, partial C, E+, partial e, VS+V- and Rh:-31. At a molecular level, the partial C antigen is encoded by alleles RHD*DIIIa-CE(3-7)-D which was present in all donors except the third donor which was C(Rh:02) negative. A silent polymorphism c.609A was identified on exon 4 of the RHCE gene in donor 2 however this has no impact on the predicted phenotype which is the same as compared to donor 1. The Rh:-31 phenotype is usually linked to RHD*DIIIa or hybrid RHD*DIIIa-CE-(3-7)-D and with homozygous 48C, 733G and 1006T polymorphisms which is present in all except the fourth donor where it is only at one allele. The fourth donor has an E+ phenotype with weak detection of Rh:-34 serologically. The third donor is an example of the RHD*IIIa hybrid with a c.150C polymorphism.

Conclusions
From the results above, the serological triggers identified will be partial or weak agglutination reactions with the C, e, and c phenotypes. VS will always be positive with V negative. At a molecular level, the RHD*DIIIa hybrids are evident and literature has supported that the presence of homozygous 733C>G in exon 5 and 1006G>T in exon 7 of the RHCE gene could replace serological phenotyping for Rh:-34. A strategic objective at SANBS is to introduce next generation sequencing to perform genomic analysis of Rh genes within the next 5 years.