IRON BALANCE REMAINS STABLE IN LIFE-LONG BLOOD DONORS

Frey B.M., Rüfer A., Hardegger K., Bachmann G., Kuhn C., Schmid I., Wägli F. and Ochmann O.
Stiftung Zürcher Blutspendedienst SRK, CH-8001 Zürich (www.zhbsd.ch)

Introduction:

Whole blood donation (WBD, 450ml) leads to additional iron (Fe) loss. Replenishment of plasma, proteins, leucocytes and platelets are achieved in a few hours to days by extra-intravascular flux of soluble components and release of cells from the splenic and marginal pool. Regeneration of red blood cells requires increase of erythropoiesis that takes two to three weeks to be accomplished and is limited by the accessible body Fe pool. Long-term and regular WBD might deplete Fe stores and may lead to Fe deficiency anemia if there is no or insufficient adaptation of Fe homeostasis.

Methods:

Active and frequent blood donors (FBD, 3-4 WBD/yr during the preceding two years) were recruited into two observational groups (<65 yrs; >65yrs, n>100 FBD/group), if they fulfilled donation criteria (males: cHb>135g/l, females: cHb>125g/l). Laboratory parameters measured: hemoglobin concentration (cHb, capillary method), serum ferritin (Ferr), soluble transferrin receptor (sTfR), C-reactive protein (CRP), all in venous blood samples. Evolution of cHb was assessed by retrospective analysis of FBD's files. Correlations of Fe metabolism' markers as well as ratio of sTfR/logFerr were calculated and graphically depicted.

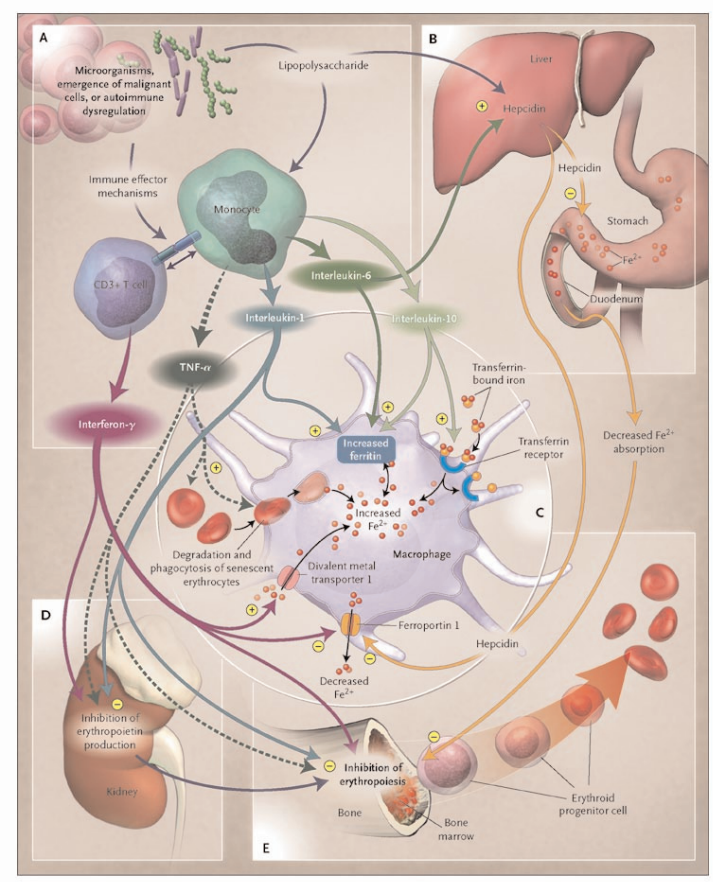
Results:

Characteristics and Outcome of FBD

Age group	≤ 65 yrs	> 65 yrs	overall
No of FBD	117	97*	214
Median Age (range)	50 (23 - 65)	69 (66 - 73)	62 (23 - 73)
Male : Female	102 : 15	75 : 22	177 : 37
Median No of Donations (range)	60 (9 - 142)	102 (19 - 198)	81.5 (9 - 198)
Hb at last donation (median, range, g/l)	153.7 (121 - 178)	151.6 (123 - 183)	152.8 (121 - 183)
Min Hb among last 13 donations (g/l)	120	114	114
Max Hb among last 13 donations (g/l)	186	185	186
CRP (mean, range, ng/ml)	3.9 (3-8)	3.9 (3-22)	3.9 (3-22)

*3 drop-outs due to insufficient data collection

Figure 5: Mechanism of Anemia of Chronic Disease by G. Weiss and L.T. Goodnough[1)]



References:

1) G. Weiss and L.T. Goodnough: Anemia of Chronic Disease. N Engl J Med, 2005: 352; 10: 1011-1023
2) R.E. Fleming and B.R. Bacon: Orchestration of Iron Homeostasis. N Engl J Med, 2005: 352; 17: 1741-1744
3) N.C. Andrews: Disorders of Iron Metabolism. N Engl J Med, 1999: 341, 26: 1986-1995
4) K. Punnonen, K. Irjala and A. Rajamäki: Serum Transferrin Receptor and Its Ratio to Serum Ferritin in the Diagnosis of Iron Deficiency. BLOOD, 1997, 89 (3): 1052-1057
5) C. Thomas and L. Thomas: Biochemical Markers and Hematologic Indices in the Diagnosis of Functional Iron Deficiency. Clinical Chemistry, 2002, 48 (7):1066-1076

Figure 1: Donors >65 years ordered by sTfR/logFerr

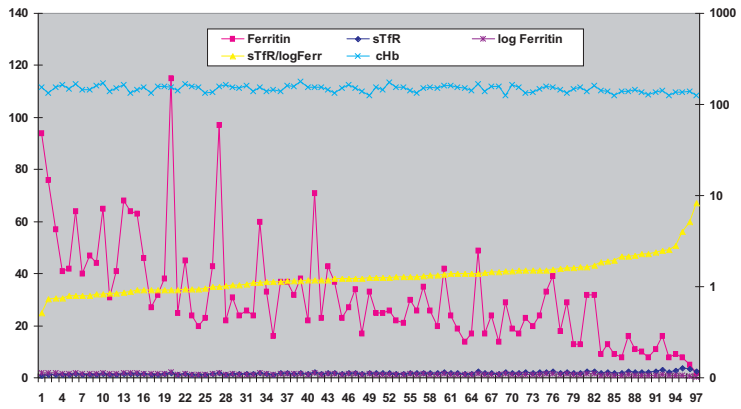


Figure 2: Donors <65 years ordered by sTfR/logFerr

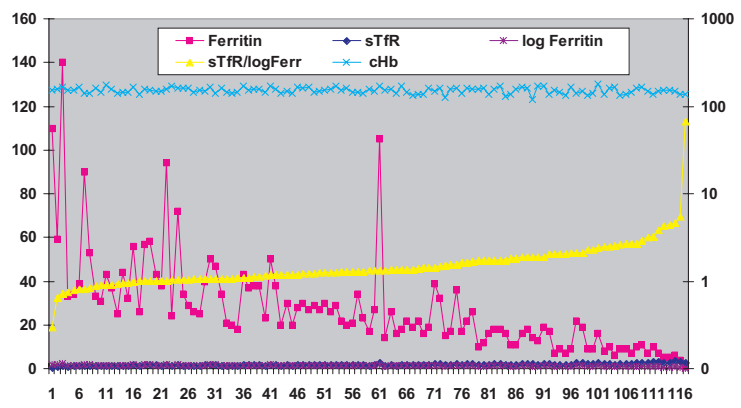


Figure 3: Donors >65 years ordered by Number of Donations

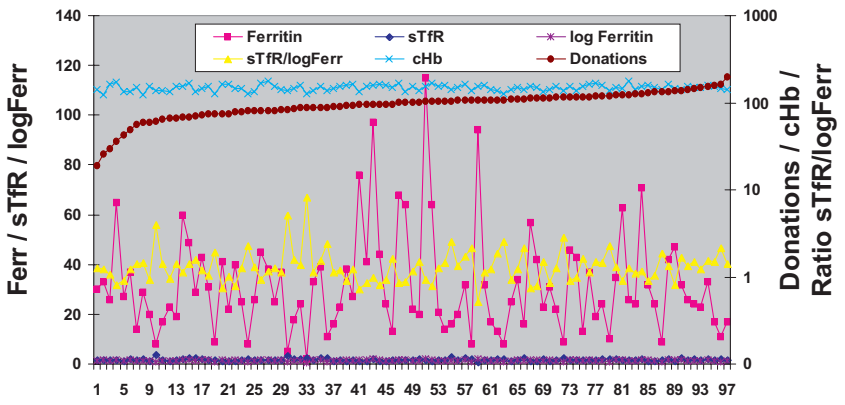
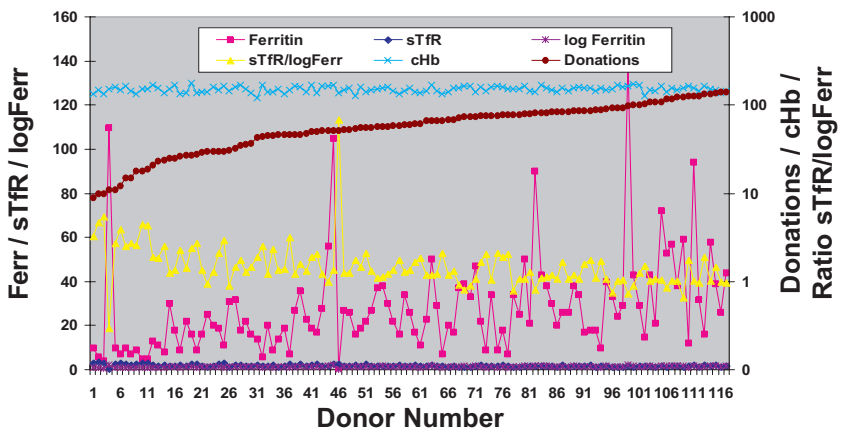


Figure 4: Donors <65 years ordered by Number of Donations



Conclusions:

1. **cHb measurement and adherance to donor selection criteria prevent development of clinically significant Fe deficienc in FBD.**
2. **Ferr alone does not provide sufficient information regarding Fe homeostasis in FBD. In contrast, regular check of ratio sTfR/logFerr (Ferritin Index) allows to recognize significant Fe depletion by WBD.**
3. **Decreasing Ferr in FBD may reflect shift of non-erythropoietic Fe into erythropoietic tissue.**
4. **Since it has been shown, that WBD decreases hepcidin secre tion, it is conceivable that increase of enteral Fe resorption, shift of body Fe into erythrpoietic tissue and releave of hepcidin mediated inhibition of erythropoiesis, all may be caused by low hepcidin concentration and contribute to efficient compensation of Hb loss by WBD.**
5. **Functional adaptation of Fe homeostasis in FBD by modulated hepcidin secretion works well even at advanced donor age.**

40. Jahreskongress DGTI, Friedrichshafen/N. Buffat, A. Havel