Comment to: Hepcidin: from discovery to differential diagnosis. Haematologica 2008; 93:90-7

In your journal there is a review article on hepcidin¹ summarizing the rapid (r)evolution of new knowledge in the field of iron metabolism gained during the last decade. New molecular actors along the iron pathways are presented in elegant figures easily available for teaching purposes by a little click on the computer.

Figure 1 reproduced from a previous review² is such a figure, illustrating the iron content of the tissues involved in the daily iron traffic. The erythrocytes have 1800 mg, bone marrow 300 mg, macrophages 600 mg, the figures for the major storage site, the liver, is not given. The numbers seem to be taken from a previous review article in N Engl J Med by Nancy Andrews.³ We wonder about the health of this person with more iron in his iron stores (1900 mg) than in the blood,1800 mg. As one gram of haemoglobin contains 3,4 mg of iron, 1800 mg corresponds to 529 gram of haemoglobin. Let us assume that he has a total blood volume of 5,5 L as that found in normal Swedish men.^{4,5} With a correction figure of 0.91 for the body/venous hematocrit ratio his hemoglobin will be 529/5.5×0,91=105,7 g/L.

He is anemic!

We previously performed vigorous phlebotomy studies to measure the iron stores in normal men. In one study⁴ of 11 normal men the iron stores measured

757±333 mg in another of 19 men⁵ 486±253 mg. None had a value exceeding 1400 mg of iron.

Thus the person presented in the reviews with anemia and elevated iron storage seems to suffer from anemia of chronic disease rather than being a healthy person.

Even if the focus of the reviews is to present new molecular events the reader should also be given correct information concerning more basal findings.

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