http://dx.doi.org/10.4172/scientificreports.367

Open Access Scientific Reports

Research Article Open Access

Keywords: Serum ferritin; Storage iron metabolism; Determination of ferritin and hemosiderin iron; Pathway of ferritin and hemosiderin iron; Relationships between storage iron; Erythropoiesis and hepcidin

## Introduction

Iron is an essential element for the living body. e human body stores iron mostly in liver, spleen, marrow and skeletal muscle in the form of ferritin and hemosiderin. Hemosiderin has been known as yellowish granules that can be stained by Prussian blue in the tissue cells. On the other hand, ferritin is invisible by photomicroscopy or may be faintly visible and stained di usely in the tissue cells by Prussian blue, if concentrated. Ferritin and hemosiderin are iron containing proteins with magnetic susceptibility. Ferritin is water-soluble and heat-resistant up to 80°C, but hemosiderin is water-insoluble and thermally denatured. e total amount of body iron stores is around 600 to 1000 mg in the normal adult male and around 200 to 300 mg in the normal adult female [1]. e ratio of iron de ciency anemia in the menstruating female is less than 10% [2-4], and that of iron de ciency without anemia is around 20 to 40% in the menstruating female [3,4]. e amount of storage iron in the normal female increases gradually a er menopause, but it is still lower [2] than the level of the normal male even a er 20 years [5].

In a negative iron balance, reserved iron will be exhausted sooner or later, and results in iron de ciency. On the other hand in a positive iron balance, iron will be accumulated in the body and results in iron overload caused by the increase of iron absorption or blood transfusion or mistreatment.

Storage iron behaves as if resisting the change in the iron density gradient [6,7]. is is a homeostatic tendency of the storage iron metabolism.

Iron produces hazardous free radicals, those causing various disorders not only in iron overload, but also in localized iron deposition [8-12]. e transformation of ferritin into hemosiderin [13,14] might be the second best evolutionary step to reduce iron toxicity, compensating for the lack of iron excretion function of the human body. An iron chelating agent, deferasirox [15], with iron removing e cacy comparable to that of phlebotomy [7] is now in use for the treatment of transfusional iron overload.

Knowledge of the storage iron metabolism seems essential not only for understanding the basis of the iron metabolism, but also for studies of the vast eld of medicine.

## Clinical Methods For Determining Iron Stores

### Quantitative determination of iron stores

Total amount of iron in the blood removed by phlebotomy [11,16-21], super conduction quantum interference device susceptometry and Magnetic Resonance Imaging (MRI) [22] were introduced. However, other errheren-AUded*-1.575hotappropriate examinations are needed cases of overestimation. Despite such disadvantages, serum ferritin has been evaluated highly for the diagnosis and treatment of patients with iron de ciency anemia and iron overload [2-7,24-27].

According to the report by Addison et al. [24], it suggested that the serum ferritin concentration might re ect the iron stores of the body, a rate [27] and a formula [2] were proposed for the conversion from serum ferritin into iron stores. However, such conversion methods do not always reect the amount of iron stores because serum ferritin cannot re ect hemosiderin iron.

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Received August 31, 2012; Published September 08, 2012

Citation: Saito H (2012) Storage Iron Metabolism.

## Determination of ferritin and hemosiderin Iron

Saito et al. [7] developed a clinical method for the simultaneous determination of ferritin and hemosiderin iron, by using a serum ferritin decrease curve, measured in the course of iron removal by phlebotomy and iron chelating. The method is based on the fact that the serum ferritin decrease curve is composed of the sum of two components, [28] a decreasing and recovering component. The decreasing component reflects the decrease in pre-existed tissue ferritin iron, and the recovering component reflects the increase of the tissue ferritin iron by removal of iron from hemosiderin, i.e. decreasing hemosiderin iron.

## Storage Iron and Erythropoiesis

Human body reserves iron probably because the supply of a sufficient amount of iron is difficult by iron absorption, when there is an

## Pathways of Ferritin and Hemosiderin Iron

### Pathways of ferritin and hemosiderin iron in iron deposition

Shoden et al. [28] proposed an iron pathway from plasma to ferritin, and from ferritin to hemosiderin in iron deposition. eir proposal seems to be supported by the transformation of ferritin into hemosiderin by various measures [14,15,28]. Shoden et al. [28] proposed an iron pathway from plasma to hemosiderin, glancing o ferritin in iron deposition. However, the nature of such a pathway seems unclear.

Shoden et al. [28] also proposed an iron pathway from hemosiderin to ferritin in iron deposition. However, such a pathway seems unlikely, because its direction is contrary to the iron ow in iron deposition [7]. e same investigators also proposed a direct iron pathway from plasma to hemosiderin, bypassing ferritin synthesis in iron deposition [28]. However, such a pathway seems unlikely because intracellular labile iron will be involved in very active ferritin synthesis, as seen by the prompt serum ferritin increase a er intravenous iron injection in patients with iron de ciency anemia. Furthermore, the detection of radioiron in hemosiderin fractions separated from the tissue homogenate soon a er a radioiron addition, does not always indicate direct radioiron incorporation into hemosiderin, since it proved di cult to distinguish adhesion from incorporation [7].

us, one iron pathway seems to exist in iron deposition, where iron ows in the numbered order of (1) hemosiderin (2) ferritin (3) labile iron pool in iron deposition.

### Pathways of ferritin and hemosiderin iron in iron mobilization

us, one iron pathway seems to exist in iron mobilization [7], where iron decreases in the numbered order of (1) hemosiderin (2) ferritin (3) labile iron pool in iron mobilization, t 9 56.6:7en-AUbus, on

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