

## Transferrin

### IRON AND TRANSFERRIN

Iron is a mineral with several essential functions in the body. It constitutes the core of both hemoglobin, the molecule that carries oxygen from the lungs to the rest of the body, and myoglobin, a protein that provides oxygen to the muscles. Iron is also essential for growth, normal cell function, and the production of connective tissue and some hormones (1).

Transferrin is the main protein that binds to and transports iron around the body.

### TRANSFERRIN RANGES

Healthy transferrin levels vary slightly by age and gender. 174 - 364 mg/dL is considered normal for adult males aged 14 - 60 years, while the normal range for adult females is 180 - 382 mg/dL. The normal ranges for over 60 years are slightly lower, at 163 - 344 mg/dL (males) and 173 - 360 mg/dL (females) (2).

### SIGNS OF IRON DEFICIENCY

Low iron levels inhibit the production of hemoglobin, resulting in reduced red blood cells. When the body can't supply enough red blood cells to meet its demands, it manifests as anemia, which affects an estimated two billion people around the globe (3). Symptoms include tiredness, fatigue, pale skin, shortness of breath, headaches, and dizziness. These initial symptoms of deficiency can go unnoticed, but if left untreated, anemia can have serious repercussions, including impaired cognitive function, disturbances in the digestive system, and impaired immunity. Pregnant women, young children and frequent blood donors are at a much higher risk of iron deficiency (4).

### SIGNS OF EXCESS IRON

Increased iron concentrations occur in hemochromatosis and acute liver disease. Usually only 8-10% of iron from the diet is absorbed. However, individuals with hemochromatosis can absorb three to four times more iron than normal (5). This excess iron cannot be naturally excreted from the body, so it accumulates in organs and tissues, eventually causing serious health complications. The symptoms of hemochromatosis include fatigue, joint pain, abdominal pain, memory problems, depression, decreased sex drive, shortness of breath, and heart flutters. Further complications can occur in untreated individuals, including heart failure, liver cirrhosis and disease, and endocrine problems (6).

### TEST PROCEDURE

Correct specimen collection and handling is required for optimal assay performance.

This test requires a blood sample from a finger prick. All supplies for sample collection are provided in this kit. First wash and dry hands. Warm hands aid in blood collection. Clean the finger prick site with the alcohol swab and allow to air dry. Use the provided lancet to puncture the skin in one quick, continuous and deliberate stroke. Wipe away the first drop of blood. Massage hand and finger to increase blood flow to the puncture site. Angle arm and hand downwards to facilitate blood collection on the fingertip. Drip blood onto the blood collection card or into the microtainer tube.

Avoid squeezing or 'milking' the finger excessively. If blood flow stops, perform a second skin puncture on another finger, if more blood is required. Do not touch the fingertip.

Dispose of all sharps safely and return sample to the laboratory in the provided prepaid return shipping envelope.

Upon receipt at the laboratory, the blood sample is analyzed by the fully automated Alinity c Transferrin on the Alinity ci series analyzer.

### DISCLAIMERS/LIMITATIONS

Certain medications and supplements (e.g., birth control pills) can affect transferrin test results. In addition, low transferrin levels can be caused by various health issues, including chronic inflammation, infections, and malignancies.

These results should be interpreted in conjunction with other laboratory and clinical information.

Additional testing is recommended if transferrin levels are inconsistent with clinical evidence.

Correct specimen collection and handling is required for optimal assay performance.

Interferences from medication or endogenous substances may affect results.

### REFERENCES

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- (4) Camaschella C. (2015). Iron-Deficiency Anemia. *N Engl J Med.* 372, 1832-1843.
- (5) Witte DL, et al. (1996). Hereditary hemochromatosis. *Clinica Chimica Acta.* 245(2), 139-200.
- (6) Beutler E, Felitti V, Gelbart T, Ho N. (2001) Genetics of Iron Storage and Hemochromatosis. *Drug Metab Dispos.* 29(4):495-499.