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Genetic Research of Iron Homeostasis and Related Diseases

Guest Editor:

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Message from the Guest Editor

Iron is an important micronutrient in the hematopoietic process and part of iron containing proteins, in the form of heme groups or Fe/S clusters. Iron is involved in many vital cellular processes and responses, such as oxidation–reduction reactions, mitochondrial respiratory chain, DNA/RNA synthesis.

In mammals, iron levels are regulated by the liver-secreted hepcidin. At a cellular level, iron homeostasis is controlled by IRP1 and IRP2, two proteins that control the expression of the genes involved in iron uptake, storage, and utilization.

Iron metabolism dysregulation leads to diseases including different forms of anaemias, i.e., myelodysplastic syndrome, atransferrinemia; iron-overload conditions, such as neurodegenerative diseases, or ataxias; and diseases involving dysfunctional Fe/S cluster proteins, such as mitochondrial dysfunction syndromes.

Great efforts have been made to reveale the genetic causes of most of these diseases, as well as their underlying molecular regulatory mechanisms. In the future, we will certainly uncover additional novel genes involved in iron-related diseases and advancing in our knowledge concerning iron regulation in health and disease.

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Message from the Editor-in-Chief

Genes are central to our understanding of biology, and modern advances such as genomics and genome editing have maintained genetics as a vibrant, diverse and fastmoving field. There is a need for good quality, open access journals in this area, and the *Genes* team aims to provide expert manuscript handling, serious peer review, and rapid publication across the whole discipline of genetics. Starting in 2010, the journal is now well established and recognised.

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