International Journal of
Molecular Sciences

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Genetic and Molecular Mechanisms of Hypertrophic Cardiomyopathy

Guest Editor:

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Deadline for manuscript submissions:
closed (31 January 2022)

Message from the Guest Editor

Hypertrophic cardiomyopathy (HCM) is a common inherited genetic disorder affecting, manifest as left ventricular hypertrophy without any underlying cause. The hypertrophy is often asymmetric, most commonly affecting the interventricular septum, and is often associated with left ventricular outflow tract obstruction. Associated findings include myocardial fibrosis, sudden cardiac death, microvascular ischemia, and mitral valve abnormalities. Although the majority of known mutations occur in sarcomeric genes, the pleiotropic manifestations of this disorder that occur in both myocytes and nonmyocyte tissues are not easily explained by these mutations. The majority of patients also do not have identifiable disease-causing mutations, raising questions about other genetic, epigenetic or nongenetic causes. Despite significant advances in understanding how HCM sarcomeric mutations affect sarcomere function and myocardial contractility, the downstream molecular mechanisms that lead to asymmetric hypertrophy, cardiac arrhythmias, mitral valve abnormalities, and microvascular dysfunction are poorly understood. This issue calls for studies that address current gaps in knowledge.

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Message from the Editor-in-Chief

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Journal Rank: JCR - Q1 (Biochemistry and Molecular Biology) / CiteScore - Q1 (Organic Chemistry)

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