Molecular Mechanisms of Freeze Tolerance in Ranid Amphibians: The Role of Cellular Adaptations and Regulatory Control in Hypometabolism

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Abstract

Amphibian freeze tolerance represents one of the most impressive vertebrate physiological specializations, allowing species such as Rana sylvatica to tolerate freezing of up to two-thirds of their body water. At the molecular level, this specialization is based on synergistic action of cryoprotectants, antioxidant enzymes, and membrane stabilizing proteins that ensure cellular integrity during freeze–thawing cycles. Molecular chaperones, anti-apoptotic signalling, and stress-activated transcription factors also maintain protein stability and limit oxidative damage, while ice-binding and nucleating proteins regulate extracellular ice growth. Advances in molecular biology have also highlighted the regulatory role of epigenetic modification, microRNAs, and reversible post-translational modifications in coordinating metabolic rate depression (MRD), which suppresses ATP-prohibitive processes while sustaining minimal cellular activity.

These processes have been explained in North American ranid frogs with some frequency, but their definition remains incomplete in European representatives, including those distributed in the Balkan region. The aim of this study is to investigate freeze tolerance in amphibians by analysing in detail the cellular adaptations and the regulatory mechanisms of stress-protective proteins against oxidative damage. In this context, particular attention is given to iron-related processes and the function of membrane protective transporters, which play a critical role in maintaining cellular integrity under extreme conditions.

References

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