The CCAAT-binding complex coordinates the oxidative stress response in eukaryotes.

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Details

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Abstract

The heterotrimeric CCAAT-binding complex is evolutionary conserved in eukaryotic organisms. The corresponding Aspergillus nidulans CCAAT-binding factor (AnCF) consists of the subunits HapB, HapC and HapE. All of the three subunits are necessary for DNA binding. Here, we demonstrate that AnCF senses the redox status of the cell via oxidative modification of thiol groups within the histone fold motif of HapC. Mutational and in vitro interaction analyses revealed that two of these cysteine residues are indispensable for stable HapC/HapE subcomplex formation and high-affinity DNA binding of AnCF. Oxidized HapC is unable to participate in AnCF assembly and localizes in the cytoplasm, but can be recycled by the thioredoxin system in vitro and in vivo. Furthermore, deletion of the hapC gene led to an impaired oxidative stress response. Therefore, the central transcription factor AnCF is regulated at the post-transcriptional level by the redox status of the cell serving for a coordinated activation and deactivation of antioxidative defense mechanisms including the specific transcriptional activator NapA, production of enzymes such as catalase, thioredoxin or peroxiredoxin, and maintenance of a distinct glutathione homeostasis. The

underlying fine-tuned mechanism very likely represents a general feature of the CCAAT-binding complexes in eukaryotes.

Involved units

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