

转录因子 AREB 和 MYB 介导的串联染色质修饰赋予杨树持续耐旱性

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摘要: 前期研究表明, *PtrMYB161* 基因在杨树中的过量表达导致了典型的耐旱表型。本研究证实, 该基因通过表观遗传途径调控基因表达, 以响应干旱胁迫。研究发现, *PtrMYB161* 蛋白直接与干旱响应基因 *PtrNAC120* 启动子中的 MYB-core 元件结合, 并招募组蛋白乙酰转移酶 (HAT) 二聚体 (*PtrGCN5-1-PtrADA2b-3*), 形成三元蛋白复合物 (*PtrMYB161-PtrGCN5-1-PtrADA2b-3*)。该复合物增强了核小体组蛋白 H3 第 9、14 和 27 位赖氨酸 (H3K9、H3K14 和 H3K27) 的乙酰化, 提高了 *PtrNAC120* 启动子中 RNA Pol I 的富集, 从而激活 *PtrNAC120* 的表达。*PtrAREB1-2* 是一种重要的干旱诱导型转录因子, 也可以介导 *PtrNAC120* 的转录激活, 从而提高杨树的耐旱性。与 *PtrAREB1-2* 不同, *PtrMYB161* 的表达不受干旱影响。然而, 在干旱条件下, 诱导的 *PtrAREB1-2* 也与 HAT 二聚体形成三元复合物 *PtrAREB1-2-PtrGCN5-1-PtrADA2b-3*, 并结合在 *PtrNAC120* 启动子的 *PtrAREB1* 结合位点 (ABRE) 处, 通过提高 *PtrNAC120* 的组蛋白 H3 乙酰化和 RNA Pol II 水平, 激活 *PtrNAC120* 的转录以响应干旱。在 *PtrAREB1-2* 诱导 *PtrNAC120* 转录激活的过程中, *PtrMYB161-PtrGCN5-1-PtrADA2b-3* 复合物在长期和严重干旱胁迫 (低于 40% 土壤相对含水量) 时被激活。通过过量表达和突变体植株的木质部材料以及原生质体的实验, 进一步说明了在干旱胁迫下, *PtrAREB1-2* 调控系统激活了由 *PtrMYB161* 介导的辅助调控。因此, 研究结果表明, 由 HAT 复合物 *PtrAREB1-2-PtrGCN5-1-PtrADA2b-3* 和 *PtrMYB161-PtrGCN5-1-PtrADA2b-3* 介导的串联表观调控共同维持杨树的耐旱性。

Concatenated Chromatin Modifications Mediated by AREB and MYB Transcription Factors Allows for Sustained Drought Tolerance in *Populus trichocarpa*

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Abstract: Our previous work revealed that transgenic overexpression of *PtrMYB161* in *Populus trichocarpa* resulted in phenotypes typically seen for drought tolerance adaptation. Here, we confirm that such transgenesis instills an epigenetic path to gene transregulation for drought tolerance. We showed *PtrMYB161* protein binds directly to the MYB-core motif in the promoter of *PtrNAC120*, a drought response/tolerance gene, to recruit the histone acetyltransferase (HAT) dimer (*PtrGCN5-1-PtrADA2b-3*), forming the ternary protein complex

(PtrMYB161–PtrGCN5-1–PtrADA2b-3). This ternary system enables enhanced acetylation of nucleosome histone 3 lysine-9, -14, and -27 (H3K9, H3K14, and H3K27) for enriched RNA Pol II occupancy in *PtrNAC120* promoter to elevate its expression for drought tolerance. Unlike PtrAREB1-2, an important drought-inducible transcription factor that can also mediate *PtrNAC120* transactivation for tolerance, *PtrMYB161* expression remains unaffected by drought. However, under drought, the induced-PtrAREB1-2 could also form HAT ternary complexes, PtrAREB1-2–PtrGCN5-1–PtrADA2b-3, and bind to PtrAREB1-binding sites (ABREs) in *PtrNAC120* promoter for *PtrNAC120*'s enhanced H3K acetylation, RNA Pol II occupancy, and transactivation for drought tolerance. While such drought and PtrAREB1-2-induced *PtrNAC120* transactivation proceeded, the PtrMYB161–PtrGCN5-1–PtrADA2b-3 mediated *PtrNAC120* transactivation became active following prolonged and severe drought stress (below 40% Relative Soil Water Content). Further loss- and gain-of-function transgenesis experiments in whole plants and stem differentiating xylem protoplasts suggested that under stress the PtrAREB1-2 regulatory system activates an ancillary regulation mediated by PtrMYB161. Our findings propose concatenated epigenetic regulations mediated by HAT complexes, PtrAREB1-2–PtrGCN5-1–PtrADA2b-3 and PtrMYB161–PtrGCN5-1–PtrADA2b-3, to jointly sustain drought tolerance in *P. trichocarpa*.