Mechanism Underlying Heat Stress-induced Anther Abortion in Wheat at the Tetrad Stage

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Abstract

Climate change has increased the frequency of extreme high-temperature events during the booting stage of wheat, coinciding with the tetrad stage—a critical window for floret development. Heat stress during this stage disrupts anther tissues, particularly the tapetum, thereby reducing grain number and causing substantial yield losses. In this study, two wheat cultivars differing in thermotolerance, Huaimai 33 (HM33, heat-tolerant) and Fanmai 5 (FM5, heat-sensitive), were subjected to heat stress at the tetrad stage. HM33 exhibited markedly higher anther fertility than FM5, which was attributable to lower accumulation of reactive oxygen species (ROS) and attenuated programmed cell death (PCD) in the anthers. Scanning and transmission electron microscopy, TUNEL assays, and physiological analyses revealed that heat stress damaged chloroplasts and mitochondria in the endothecium and tapetum, elevated Caspase-9 and Caspase-3 activities, and thereby exacerbated PCD, ultimately leading to severe degradation of the tapetum and adjacent tissues. Toluidine blue and periodic acid–Schiff (PAS) staining further demonstrated that structural damage to the endothecium and tapetum blocked carbohydrate transport to microspores, resulting in insufficient polysaccharide accumulation and a pronounced decline in pollen viability. Collectively, heat stress triggers ROS over-accumulation and intensified PCD, disrupts anther architecture and sugar translocation, and consequently increases anther abortion, reduces grain number per spike, and causes significant yield losses. These findings provide a theoretical basis for breeding heat-tolerant wheat cultivars and developing mitigation strategies against heat stress during the booting stage.

Keywords

Tetrad Stage, Heat Stress, Anther Abortion, Reactive Oxygen Species, Programmed Cell Death