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Mechanisms and regulation of senescence and maturity performance in cotton

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ABSTRACT

Senescence in cotton is a gradual deterioration process that terminates plant growth and development and ultimately leads to plant death. Maturity performance refers to the senescence appearance of cotton during the boll-opening stage, which is the manifestation and result of senescence. Maturity performance categories include premature senescence, late maturity and normal maturity. Premature senescence and late maturity are anomalies of cotton growth caused by genotype and environment interactions. They result from differential expression of a large number of senescence-associated genes during late growth and development stage. In this paper, we review and summarize research progress on the physiology, ecology, and molecular basis of cotton senescence and maturity performance. Strategies for achieving normal maturity performance with maximum cotton yield and quality should include development of optimized varieties, rational application of plant growth regulators, and use of agronomic measures to regulate cotton growth, development and senescence.

Contents

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1. Introduction

The growth and development of organisms typically include immature stages, gradual maturation, natural aging of organs or some activity units, and finally death. In crops, senescence refers to the process leading to the natural termination of plant life or loss of organ function. Cotton is a perennial plant that originated from tropical and subtropical regions, but it has become widely cultivated as an annual crop. Even after long cultivation and domestication, cotton still maintains a thermophilic, heliophilic and indeterminate growth habit (Dong et al., 2005b). Like many other plant species, both the plant parts (tissues and organs) and the whole body (individuals and groups) of cotton go through a maturation process from seedling to maturity and from maturity to the aged and finally death, which is the termination of a life cycle (Kong and Dong, 2011). As a result, there can be three different maturity performance categories including normal maturity, premature senescence and late maturity in cotton.

The performance of cotton in the boll-opening stage is called maturity performance (Kong and Dong, 2011). Normal senescence leads to the formation of normal maturity performance, which can help cotton plants avoid the effects of later adverse weather conditions such as frost, and efficiently utilize limited energy and material resources within the growing season to improve yield and quality. Thus normal maturity performance is of positive significance. Premature senescence and late maturity, however, can decrease fiber yield and quality, with negative significance (Dong et al., 2006). Cotton maturity performance is affected by the sink-source ratio, which is significantly affected by the relationships between vegetative and reproductive growth, root system and canopy, and the carbon and nitrogen metabolism balance (Kong and Dong, 2011). Research on source-sink coordination, the relationships between vegetative and reproductive growth, roots and canopy, and carbon and nitrogen metabolism balance are important to elucidate the mechanism of cotton maturity performance. High cotton yield and fine quality can be achieved by using appropriate cotton varieties and agronomic techniques to regulate plant growth and development and aging and maturity, because efficient and rational regulation of plant senescence by varieties and agronomic techniques will optimize use of light, heat, water and other natural resources during the limited growing season for cotton to develop a normal maturity. In this paper, we review the physiological and molecular mechanisms of cotton senescence and maturity performance and regulation technologies based on existing research combined with our research progress in this field.

2. Mechanisms and factors influencing cotton senescence

2.1. Environmental factors and senescence

2.1.1. External environmental factors

Major external environment factors that affect cotton leaf senescence include light, temperature, moisture, and mineral nutrition. Light not only provides energy for photosynthesis, but it is also important for the formation of auxin. Regulation of plant growth and development by controlling the light cycle plays an important role in regulation of plant senescence (Wu et al., 2012b). Shading can affect the source-sink relationship, often resulting in premature senescence of cotton leaf (Dusserre et al., 2002). Darkness will accelerate leaf senescence. Dark-induced senescent leaves have decreased chlorophyll content so that normal photosynthesis was no longer possible (Guo and Gan, 2011). High plant density may enhance mutual shading of plants, especially after flowering, but recently Echer and Rosolem (2015) indicated that self-shading is not primarily responsible for the decreased photosynthetic rate in older cotton leaves. The result suggested that self-shading will not enhance leaf senescence in cotton grown under high plant density.

Excessively high or low temperatures can damage cotton leaves and accelerate leaf senescence. Under high temperature stress, photosynthesis is affected by an increase in reactive oxygen species in chloroplasts, which leads to chloroplast and thylakoid membrane damage, photosynthesis related protein degradation, and photosynthetic electron transfer changes (Ougham et al., 2008). These changes induce leaf senescence (Sedigheh et al., 2011). Under low temperature stress, membrane damage is related to membrane lipid peroxidation and reduction or loss of protein activity caused by free radicals and reactive oxygen species. This leads to oxidation damage of leaf cells, cytoskeletal damage, and disorder in photosynthesis and related signaling pathways, and finally speeds up the aging process (Sharma et al., 2005). In addition, short-term low temperature stress will reduce nutrient cycling in cotton and the ability to absorb carbon dioxide. Also, cotton plants become susceptible to *Alternaria* infection under low temperature stress, and the eventual result is premature senescence (Zhao et al., 2012a).

Water supply also significantly affects cotton senescence. Drought and water deficiency cause accumulation of ethylene and abscisic acid *in vivo*, promote protein and chlorophyll degradation, reduce photosynthesis, increase respiration, and finally accelerate senescence in plants (Lim et al., 2007). The most active period of vegetative growth in cotton occurs in squaring and early blooming. During this period, drought and water deficiency will significantly reduce the vegetative growth and source-sink ratio, and cause premature senescence. Drought and water shortage during peak boll-setting and the beginning of boll opening will promote leaf senescence and shorten the growth period, leading to reduction in photosynthesis and allocation of assimilates to sink bolls, and will also affect normal maturity and cotton yield (Chastain et al., 2014). Waterlogging may cause anaerobic respiration in cotton and accelerate leaf senescence. Waterlogging during the flowering and boll-setting period may increase soluble sugar, starch and soluble protein content in main-stem functional leaves, increase malondialdehyde (MDA) content, and significantly reduce plant photosynthetic rate (Zhang et al., 2015). These changes show that waterlogging significantly affected the growth and physiology of cotton, led to premature senescence and reduced yield. In general, the longer the waterlogging lasted, the greater the yield loss (Zhang et al., 2015).

2.1.2. Mineral nutrition factors

Inadequate mineral nutrition can cause premature senescence of cotton. Of the mineral elements, most attention has been focused on potassium (K) nutrition. Cotton maturity performance is closely related to available K content in the soil. Potassium depletion increased MDA content, reduced soluble protein and chlorophyll content and photosynthetic rate in functional leaves, decreased root activity during the late growth period, and finally led to premature senescence (Dong et al., 2004). Decreased photosynthetic rate under K deficiency is caused by damaged chloroplast ultrastructure