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Molecular mechanisms of caste-specific cuticular tanning in termites.

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Termites have three distinctive castes (reproductives, workers, soldiers), which are morphologically specialized for their tasks. Especially, cuticular nature is remarkably different among each caste. To know the formation mechanisms of the caste-specific traits is important for understanding of the termite social evolution. The cuticle of each caste is expected to be the result of specific modifications to the cuticular tanning (sclerotization and pigmentation) process, but the origins of those differences have not been resolved. The tyrosine metabolic pathway plays an important role in cuticular tanning in insects. Thus, we hypothesized that this pathway and related hormone signaling were involved in the caste-specific cuticular tanning process. We performed gene expression and functional analyses of some candidate genes during each molt (worker-worker, worker-presoldier, presoldier-soldier and nymph-alate) in *Zootermopsis nevadensis*. First, gene expression analysis revealed that some tyrosine metabolic genes were highly expressed just before and after molts to soldiers and alates. Moreover, suppressing expression of Laccase2 (Lac2), which is known to act on both sclerotization and pigmentation in some model insects, using RNA interference (RNAi) caused the soldier and alate cuticles with light color and soft nature. Next, expression patterns of the ecdysone and juvenile hormone (JH) signaling genes were likely correlated to those of the tyrosine metabolic genes. RNAi of the ecdysone receptor gene (EcR) also produced a soldier cuticle with similar characteristics to Lac2 RNAi, but did not affect alate cuticular formation. Knockdown of methoplene tolerant (Met), which is a candidate receptor gene of JH, did not change the nature of soldier and alate cuticles, but resulted in shorter mandibles and smaller head capsules of soldiers. These results suggest that the caste-specific cuticular tanning requires expression level changes of the tyrosine metabolic genes, probably regulated by the ecdysone signaling.