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Developments of termite neotenics regulated by high Juvenile Hormone titers

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Termites show the highly flexible caste differentiation and each caste is morphologically specialized in their own tasks including reproduction. There are mainly two types of reproductive caste in termites; the primary reproductives which found a new colony, and the neotenic reproductives (neotenics) which take over reproduction when the primary reproductives are lost. Neotenics have highly developed gonads, whereas other imaginal organ formations are strongly arrested. Therefore, organ formation of neotenics must be different from those of primary reproductives. Juvenile hormone (JH) is the multifunctional hormone playing many important roles, including the gonad development and retaining juvenile characteristics. Although JH might be involved in the neotenic differentiation, developmental mechanisms underlying are still unknown. Especially, it is not clear if JH has an effect on their body-parts specific developments. To clarify this issue, using *Reticulitermes speratus*, we (1) quantified the JH titers of neotenics and compared those of other developmental stages, and (2) analyzed gene expression changes of JH signaling molecules [including the homolog of Methoprene-tolerant (Met) which is a JH receptor gene] in their different body parts (head, thorax and abdomen). Consequently, the JH titers of neotenics, quantified by the high performance liquid chromatography-mass spectrometry, were significantly higher than those of nymphs and workers. The JH titers observed in neotenics were clearly higher than those of primary queens shown in the previously published data. These high JH levels might be related to the greater gonad developments and the possession of the higher fertility in neotenics than primary reproductives of this species. Moreover, expression levels of the Met homolog in the abdomens of neotenics were significantly higher than those of any other developmental stages. These results suggest that the developments of neotenics are caused by the large increase of JH titers and body-parts specific activation of JH signaling pathway.