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# The Silkworm *og* Gene, Whose Mutation Causes Translucent Larval Skin, Encodes Molybdenum Cofactor Sulfurase

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Uric acid is accumulated in silkworm (*Bombyx mori*) larval epidermis and makes the skin white and opaque. Mutants which are unable to synthesize or accumulate uric acid have translucent larval skin. Such mutations have been mapped on more than 20 loci in various linkage groups, showing that many factors are involved in the synthesis, transportation and accumulation of uric acid. While most of the mutants are deficient in uric acid accumulation, only two of them, *og* and *oq*, have been known to be unable to synthesize uric acid because they lack the activity of xanthine dehydrogenase (XDH), which oxidizes hypoxanthine and xanthine to uric acid in the larval fat body, midgut and Malpighian tubules. Since the *oq* gene has been found to encode XDH protein, the *og* gene seems to be involved in a factor regulating XDH activity expression. In the present study, we found that the *og* mutant lacks aldehyde oxidase (AO) and XDH, both of which require molybdenum cofactor (MoCo). Hypothesizing that the *og* gene is responsible for MoCo synthesis, we isolated a silkworm homolog of the *Drosophila maroon-like* (*mal*) gene encoding MoCo sulfurase, which converts oxo form of MoCo to sulfido form required by XDH and AO. Linkage analysis indicated that the *mal* homolog is on the *og* locus. We also found that two *og* mutants, *ogk* and *ogt*, have a deletion and an insertion, respectively, which cause premature stop codons in the *mal* homolog, while the *og* mutant has several amino acid replacements. The deduced function of the silkworm *mal* homolog, the linkage analysis and the gene structure in the mutants show that the *og* gene is the silkworm *mal* homolog and encodes MoCo sulfurase.