Metamorphosis Under Artificial Light and Added Melatonin

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Melatonin made by insects and plants regulates growth and development. Ecdysteroids like 20-hydroxyecdysone (20E) promote maturation, molting and butterfly emergence through oxidative stress; so as an antioxidant melatonin may delay metamorphosis. Phylogenetic differences in enzymes leave lepidopterans unable to make melatonin in daylight or under short wavelength artificial light at night (ALAN). For example, while white and blue ALAN had no effect on melatonin in mallow plants, exposed painted lady caterpillars and chrysalises contained less melatonin and butterflies emerged faster than unexposed controls. Caterpillars consume plant melatonin, but it is unclear if this compensates for ALAN-impaired endogenous synthesis. Host plants also produce flavonoids like myricetin that impair melatonin synthesis by inhibiting light-sensitive serotonin N-acetyltransferase (SNAT). This raised the question: How much dietary melatonin or SNAT inhibitor is needed to regulate metamorphosis pace relative to ALAN in caterpillars? The hypothesis was: If melatonin delays metamorphosis, then adding it to caterpillar diets will slow butterfly emergence, while inhibiting melatonin synthesis will speed it up dose-dependently. To test this, melatonin was added at 0, 0.5, 1, 5 and 10 mg/g, or myricetin at 0, 0.1, 0.5 and 1 mg/g to caterpillar diets. Butterflies emerged larger and had more 20E with 0.5 mg/g myricetin but developed no faster. Chrysalises didn't form with 5-10 mg/g melatonin diets. Butterflies emerged slower with 1 mg/g melatonin and chrysalises had more melatonin and less 20E in hemolymph than controls. Thus, melatonin could be used to control lepidopteran developmental timing.

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