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Maternal and placental melatonin: actions and implication for successful pregnancies

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Melatonin is one of the main sources of mitochondrial protection and its protective effects are equal or even better if compared with several consecrated antioxidants. Furthermore, the activation of specific melatonin receptors triggers several cellular pathways that improve the oxidoreduction and inflammatory cellular state. The discovery of the melatoninergic machinery in placental cells was the first step to understand the effects of this indoleamine during pregnancy. In critical points of pregnancy, melatonin has been pointed as a protagonist and its beneficial effects have been shown as essential for the control of trophoblastic function and development. On the contrary of the plasmatic melatonin (produced in pineal gland), placental melatonin does not vary according to the circadian cycle and acts as an autocrine, paracrine, intracrine, and endocrine hormone. The important effects of melatonin in placenta have been demonstrated in the physiopathology of pre-eclampsia with alterations in the levels of melatonin and in the expression of its receptors and synthetizing enzymes. Some authors suggested melatonin as a biomarker of pre-eclampsia and as a pos-

Melatonin (*N*-acetyl-5-methoxytryptamine) is a molecule with wide protective effects that was thought to be exclusively produced in the pineal gland of superior animals.[1] However, it is well known now that its synthesis is virtually present in every cell of every organism, from bacteria throughout the animal and plant kingdoms.[2] This indoleamine is synthetized from the L-tryptophan pathway in a multistep process that has serotonin as an intermediate precursor. The serotonin is acetylated by the enzyme arylalkylamine *N*-acetyltransferase (AANAT) in *N*-acetylserotonin that is finally converted in melatonin by the enzyme serotonin *N*-